Third Symposium on Silicosis

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USG 42

pass on through it and are deposited in the more normal lung beyond. When the intrapulmonary reaction extends to the surface of the lung, the pleura becomes inflammed and a local fibrosis ensues. The mechanism of its involvement is not altogether clear as asbestosis bodies are generally scarce in the pleura itself.

Animal experiments have thus far failed to reproduce the widespread fibrosis seen in the human cases that have come to autopsy. Whether this is due to insufficient exposures or whether it is because the fatal human cases have been complicated by an element of healed infection still awaits demonstration. The speaker suspects that the latter may be the case for many infections, including tuberculosis, show a decided tendency to heal with the production of excessive scar formation in animals with asbestosis.

The method by which asbestos exerts its effect has not been clearly defined. There are some who believe that asbestosis is merely a silicosis which manifests itself in a different anatomic form. They hypothecate that the magnesium is leached out of the silicate molecule leaving silica atoms to act upon the tissues. If this were true one would expect asbestos to exert its effect upon connective tissue in any part of the body as happens when free silica is injected, but such is not the case. Sayers and Miller failed to produce fibrosis by intraperitoneal injection of chrysotile asbestos and have classified this substance as "inert". Similar results have been obtained in the Saranac Laboratory both in the peritoneal cavity and other tissues of the guinea pig and in the extra pulmonary organs of other species. In the light of present experience asbestos seems to be a dust capable of producing reaction only in the lungs. Possibly the irritation in this organ is largely mechanical although this remains to be demonstrated.

An unusual feature of this disease is the formation of the asbestosis body. This results from the development of an iron-containing coating upon the surface of inhaled asbestos fibres. It appears after about 2 months contact with the pulmonary tissues. Injection of asbestos into other tissues usually fails to produce such bodies although a very occasional one has been discovered in the peritoneal and subcutaneous tissues. As the coating thickens it fractures into segments whose edges become smooth and rounded. The structures are bright chrome yellow and may be 20, 70 and even 100 or more microns in length, and 2 to 4 microns in width. They resist digestion with trypsin and strong alkalies but not strong sulphuric acid; they apparently persist in the tissues for prolonged periods. Dr. R. G. Mills reports a case in which death occurred 12 years after a short exposure to asbestos dust. Sections of the lungs, which the speaker has studied, still show what appear to be the remnants of largely digested asbestosis bodies in addition to large numbers of naked fibres. This finding in an isolated case suggests that in time the coating on the fibres may be redissolved. Many observers have reached the conclusion that the coating substance is laid down by the body to protect itself against irritation. In support of their view is the fact that numbers of bodies digested out of the tissues with antiformin and injected into animals failed to cause fibrosis or other reaction. But if this explanation is correct one would expect similar deposits on foreign bodies of other types, but such is not the case. It seems much more probable that as Cummings has suggested, the coating material is dissolved out of the fibre and redeposited on its surface. The iron may be derived from the fibre or from the body fluids as proved by injecting iron into asbestos.

The significance of asbestosis bodies in the sputum or in pulmonary tissues depends upon the occurrence of associated fibres. If only a very few are present they may result from accidental inhalation and hence do not mean disease. The speaker has found occasional ones in the lungs of at least a half dozen cases where there was no industrial exposure to asbestos dust.

RELATIONSHIP BETWEEN PNEUMOCONIOSIS AND TUBERCULOSIS

The section on infection relates particularly to tuberculosis for while it is generally granted that all types of pulmonary infection are more common in the silicotic than the normal lung, the evidence in support of this view is not too clearly defined. It is coming to be recognized, for example, that lobar pneumonia is not unusually common or unusually fatal in persons with silicosis. The same may not be true of the bronchopneumonias, which need much more investigation. But there is plenty of evidence to prove a specific etiologic relationship between silicosis and tuberculous infection.

Experimental Methods have substantiated a host of clinical and statistical investigations which prove that two conditions are interrelated. Price of Toronto demonstrated that the addition of

Third Symposium on Silicosis

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Third
Symposium
on
Silicosis

An official transcript of the Third Silicosis Symposium held in connection with the Trudeau School of Tuberculosis at Saranac Lake, N. Y., June 21 to 25, 1937.

Edited by
B. E. KUECHLE, *Vice President and Claims Manager*
EMPLOYERS MUTUAL LIABILITY INSURANCE CO.
SARANAC, WIS.

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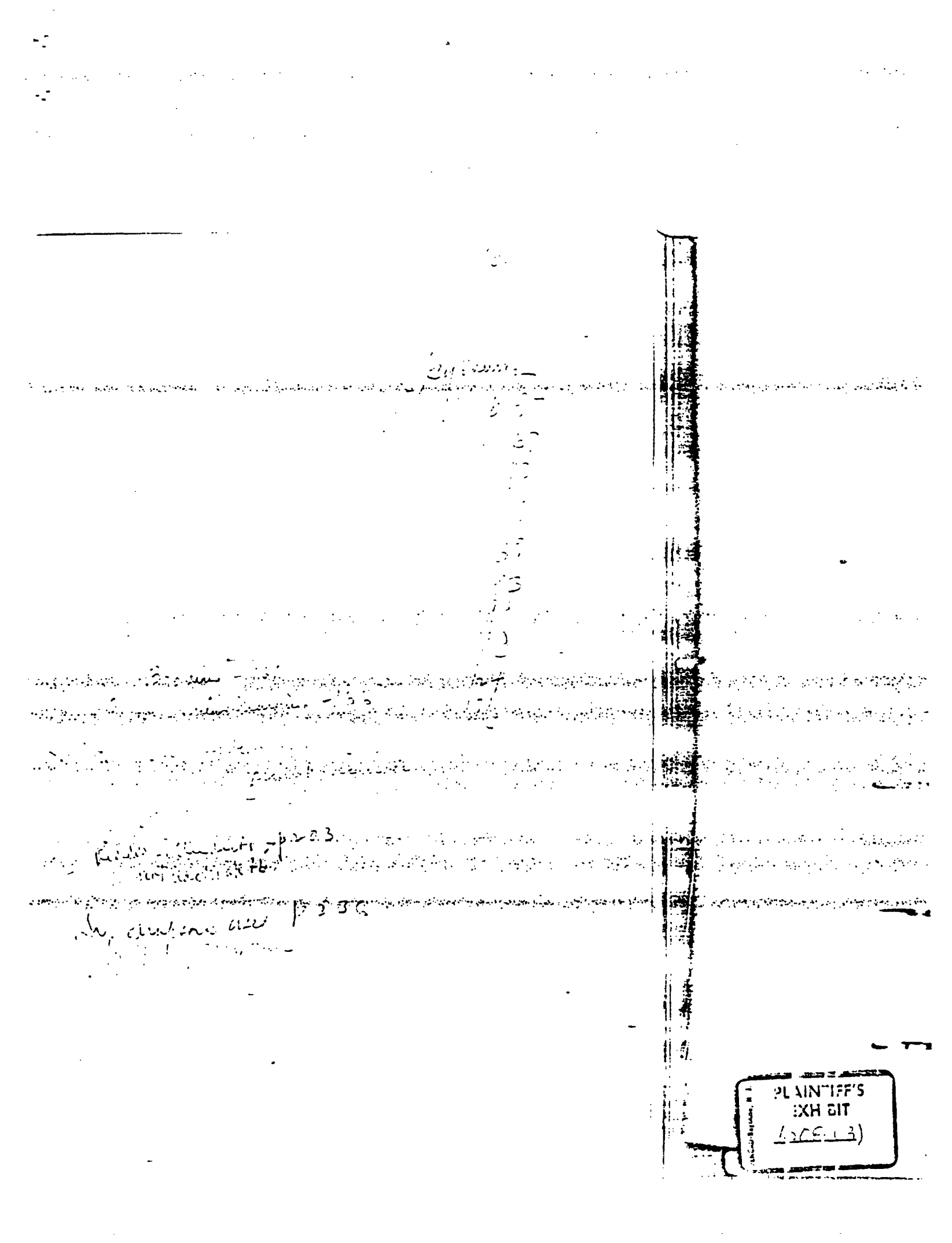
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THE SARANAC LABORATORY FOR THE STUDY OF TUBERCULOSIS

DR. L. U. GARDNER
Director of Saranac Laboratory

It is difficult for most people to understand the geographical and administrative relationships between the Saranac Laboratory, Trudeau Sanatorium and the Trudeau Foundation. The Sanatorium is an institution for the care of tuberculosis patients and is located on the outskirts of the village of Saranac Lake. The Trudeau Foundation is an organization created in memory of the late Dr. E. L. Trudeau to foster research in tuberculosis. The Saranac Laboratory is one of several units for research which are owned and sponsored by the Trudeau Foundation. The latter also maintains a clinical research laboratory inside the sanatorium grounds and fosters clinical and other forms of research.

The group of buildings occupied by the Saranac Laboratory happens to be located in the village and not at the Sanatorium because the first unit, given to Dr. Trudeau as a private research laboratory, was built where it would be convenient to his residence. His original laboratory was merely a room in his home but when he lost everything in a disastrous fire Mr. George C. Cooper, in 1894, gave [illegible] search. On Dr. Trudeau's death the Saranac Laboratory was entrusted to the Foundation, under whose administration it provides diagnostic facilities to the community, carries on experimental research and serves as headquarters for the Trudeau School of Tuberculosis. In the course of years several additional buildings have been erected. Through the generosity of Mr. and Mrs. Frank B. Black of Mansfield, Ohio the John Black Wing provided a library and lecture room as well as offices and additional laboratory rooms. Mr. Ernest V. Smoot of Salt Lake City gave funds for a special animal house in which to conduct dust inhalation experiments, and an anonymous donor provided another building for non-dusted animals.

For years the Saranac Laboratory pursued investigations on the causes of tuberculosis and mechanisms by which it acts within the body in the hope that a cure might be discovered. Many of the

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studied here. Each year since 1916, a [illegible] on all phases of tuberculosis has been offered to physicians and the graduates are now manning many, if not most of our tuberculosis sanatoria.

When the present director came to Saranac Lake in 1918, Dr. E. R. Baldwin, who was then director, discussed with him the high mortality from tuberculosis among the granite cutters in Barre, Vermont, and the subnormal death rate from this cause in marble cutters at Proctor, in the same state. Dr. Baldwin believed that the dusts which these two groups of stone workers inhaled might be responsible for the obvious differences in their response to tuberculosis. A tentative program of experimental research was mapped out and it was hoped that in a short time the effect of these dusts might be evaluated.

It soon appeared that this was a problem of major proportions with many ramifications; years instead of months would be required to obtain results. The action of different kinds of dust in animals and human beings would have to be evaluated to form a background. Only the chronic effects of tuberculosis could be of much significance. Gradually all the facilities of the laboratory were organized to pursue such a study. Funds were obtained through the good offices of the Metropolitan Life Insurance Company, The Milbank Foundation, The Public Health Service, Mr. Frank Black and other friends. The special building in which inhalation experiments are now conducted made it possible to extend the scope of investigation. The results of experiments were published from time to time but they apparently attracted little attention from any one except special students of tuberculosis.

Ten years later, in 1928 Mr. D. L. Cummings joined the staff as chemist. His knowledge proved a most valuable adjunct, for up until that time most of the work had been in the fields of pathology and bacteriology; and his training as a chemical engineer enabled him to grasp the various details of the problem.

It was not until 1932, when depression had made it increasingly difficult to obtain funds that field studies were undertaken. Under Mr. Cummings' direction and in collaboration with Dr. H. L. Sampson, Roentgenologist at Trudeau Sanatorium, a series of studies was undertaken in different industries. These included a physical investigation of the dust or other hazards and an examination of the employees. The program was always one of education and training of the local personnel with the members of the Laboratory Staff as consultants. Trained technicians [illegible] the clinical and roentgenological laboratories in each plant were supplied when necessary. Local physicians have made the examinations except in a few studies in small industries which had no medical service. In such cases Dr. D. M. Brumfield of Saranac Lake has [illegible] to do them himself or made certain that a local doctor designated by the company was familiar with the requirements. Studies have been carried on in the iron ore district about Lake Superior, in a gypsum and cement industries, in a group of fluorspar mines and in a large steel mill. These field studies have proved as productive a form of research as any which could be undertaken for they have yielded the practical information which was needed to evaluate the experimental results observed in animals.

The information obtained from field and experimental investigation has been given to all who could use it. By correspondence, by interview, by lectures, by publications [illegible] the medium of the symposiums, the Laboratory has attempted to place what is known of this disease in the hands of those who are dealing with its practical aspects.

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PROGRAM

Third Silicosis Symposium
Saranac Lake, N.Y.
June 21 -- 25, 1937

Monday, June 21:

9:00 a.m. Introductory Remarks. Dr. L. U. Gardner, Saranac Laboratory.

9:15 The Significance of the Silicotic Problem. Dr. A. J. Lanza, Metropolitan Life Insurance Company.

10:15 Etiology of Silicosis. Dr. R. R. Sayers, U. S. Public Health Service.

2:15 p.m. Dust Concentrations and Their Measurement. Prof. Philip Drinker, Harvard School of Public Health.
Discussion, led by Mr. T. M. Durkan, Saranac Laboratory.

Tuesday, June 22:

9:00 a.m. Pathology of the Pneumoconioses. Dr. L. U. Gardner, Saranac Laboratory.

Discussion.

2:15 p.m. Pathology of the Pneumoconioses. Correlation of Pathologic and Roentgenologic Findings. Dr. L. U. Gardner, Saranac Laboratory.

Discussion.

4:00 Physiology of the Fibrotic Lung. Prof. W. S. McCann, University of Rochester, School of Medicine.

Discussion.

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Wednesday, June 23:

9:00 a.m. Roentgenologic Aspects of the Normal and the Silicotic Lung. Dr. E. P. Pendergrass, University Hospital, University of Pennsylvania.

Discussion.

2:15 p.m. Roentgenologic Diagnosis of Silicosis. Dr. H. L. Sampson, Trudeau Sanatorium.

Discussion.

Thursday, June 24:

9:00 a.m. The Clinical Picture and Diagnosis of Silicosis with Consideration of Disability. Dr. A. R. Riddell, Ontario Department of Health.

Discussion.

12:00 The Occupational Picture. Mr. D. F. Cummings, Saranac Laboratory.

2:15 p.m. Control – Medical Aspects. Dr. A. R. Riddell, Ontario Department of Health.

3:15 Control – Engineering Aspects. Prof. Philip Drinker, Harvard School of Public Health.

Discussion.

Friday, June 25:

9:00 a.m. Administrative Aspects of Silicosis. Mr. D. F. Cummings, Saranac Laboratory.

Discussion.

2:15 p.m. Legislative Control and Compensation. Mr. T. C. Waters, Occupational Disease Commission, Baltimore, Md.

Discussion.

INTRODUCTORY REMARKS

Dr. L. U. Gardner
Saranac Laboratory

It gives me pleasure to welcome you to the Third Silicosis Symposium held by the Saranac Laboratory. Many of you have attended one or both of the previous sessions and it is particularly gratifying that you should care to return again. I trust that the newcomers may find it equally profitable.

As soon as I realized that the group would be composed largely of former members, I was perplexed as to the type of program which should be offered. I therefore sent out a circular letter to all previous registrants for help. Over half of those who expected to return replied that they would like a repetition of the previous program with emphasis on practical aspects. For this reason and since there were also to be a large number of new members, I decided that the best procedure would be to adhere to our original plan. I trust that this may meet with general approval and that those who have been here before will find enough of interest to justify their presence. I imagine that when the discussions have been summarized several points of view will have developed that are different from those reached in previous sessions.

Such success as these meetings have enjoyed has been due to the generosity of guest speakers who have come without compensation to lead the discussions and to the participation of all members of the group. I take this opportunity to thank the speakers who have once again made the tiresome journey to Saranac Lake at considerable inconvenience to themselves. Mr. Waters, whose previous views on Legislative Control and Compensation, is the only new speaker. From his comprehensive knowledge I am certain that we shall all gain information of fundamental importance in dealing with this all-important and timely subject. I deeply regret to inform you that Dr. Lanza, who was to have opened our discussions with a discussion of the significance of the silica problem, is unable to be with us. We shall miss the many contributions which he was able to make from his lifelong experience in the field.

Let me remind you that this symposium cannot accomplish its purpose unless every member contributes to the discussion. Most

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of you are experts in one phase or another [illegible]. We have come together to exchange ideas and to [illegible] each other's help. Only a very few have had no previous contact with silicosis. I shall expect that they will insist that any points which are not clear be amplified.

I have adopted a suggestion made by a former member and requested the different speakers to furnish outlines of their remarks. Most of them have found time to comply and before each talk mimeographed copies will be distributed so that you will be relieved of the burden of taking notes. As in previous years, we will prepare an official transcript which Mr. Kuechle assures me the Employers Mutual Liability Insurance Co. of Wisconsin will again publish and distribute sometime during the early winter. When it becomes available every attendant will be notified.

Members of the Laboratory staff will be pleased to show you the various departments including the dust house where animal experiments are in progress. The medical museum in the basement, at your right, contains a large number of specimens of lungs of persons exposed to many different kinds of dust. You may find it profitable to inspect and study them. For those interested in roentgenology, we have equipped the small room at the head of the stairs with view boxes. Dr. Sampson has set up a demonstration of typical forms of various dust reactions with and without complicating infection. Apparatus for the collection and sampling of atmospheric dust is on display in the chemical laboratory on the floor above this room. Mr. Durkan, Mr. McGrum or Mr. Redlin will demonstrate its use to anyone interested. Please feel free to inspect any or all of these exhibits whenever there is opportunity.

THE SIGNIFICANCE OF THE SILICOTIC PROBLEM

DR. L. U. GARDNER

Dr. Lanza was to have pointed out the significant features of the silicosis problem. As an expert phthisiologist in the Public Health Service he went to Joplin, Mo., to discover the cause of the high incidence of tuberculosis among the lead and zinc miners. He found a dust hazard and a silicosis problem that was apparently the same as that in South Africa. This began an interest that has taken him into almost every mining centre and to most of the silica industries in the country. From personal experience, he knows the problems of the employer and their employees. As an officer in a progressive insurance company, he has done [illegible] the places where hazards exist and to see that [illegible]. As a member of advisory committees, he has had a wide influence on corrective and compensation legislation. He could have set forth the magnitude of our problem in its medical, engineering, economic and social aspects. But he is not with us. I have not attempted to burden any of our other speakers with the task.

I shall merely remind you that the problem is a twofold one. We must discover the individuals who have already developed silicosis because of previous ignorance of dust hazards and see that they are protected from tuberculous infection or are given treatment if they are now infected. But more important still, we must discover and eliminate the dust hazards that may exist in American industry.

There may be 500,000 persons exposed to hazardous concentrations of silica dust in our industries, as Drs. Lanza and Vane have estimated; or there may be 1,200,000 persons exposed to at least some concentration of silica, as Dr. Sayers has estimated. Only study and experience will demonstrate what proportion of the groups exposed will actually develop silicosis. We may estimate from the average experience in the mining industry that approximately one fourth of the 500,000 exposed to dangerous concentrations of silica should contract the disease. But this is only an estimate and it may fall very wide of the mark. We are beginning to suspect that the hazard from silica is not defined only by the number of silica particles of a certain size that may be present in an industrial

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atmosphere. The other components of the [illegible] the inhalation of silica and they may modify [illegible] enough is known about the principles involved to permit even speculation as to the hazard created by mixtures of silica with various dusts. One must still look to the men who have been exposed to discover the damage that has been done and to evaluate the hazard. The only other means at our command is experimentation with animals. Considerable time will be devoted to this subject in the programs that are to follow.

But our problem would be relatively simple if we had to deal only with uncomplicated silicosis, for when it develops in otherwise normal lungs it rarely produces significant disability or interference with working capacity. Silicosis is the menace that it is because it specifically predisposes to tuberculosis. It is the latter which is responsible for disability, lost time and, in many cases, death. And it is really tuberculosis against which we have to fight.

One should differentiate clearly between the prevalence of tuberculosis among silicotic individuals who are at work and the frequence of this complication among the various other conditions which cause their death. When the silicosis problem became acute in this country we had certain concepts about the disease which were largely based upon experience in hard rock mining communities in South Africa and England, and in places like Butte, Picher and Barre at home. In many of these locations the conditions of exposure had been severe and the mortality had been high. Dr. Irvine, the chairman of the Miners' Phthisis Medical Bureau at Johannesburg, had made the following statement which the speaker had erroneously interpreted and has too often misquoted: "A measure of the extent of predisposition to tuberculosis in cases of silicosis is afforded by the fact that of all the deaths which have occurred in the two groups mentioned below" (ante-primary and primary simple silicosis) "some 75 percent have been due directly to active tuberculosis." To have interpreted these words to mean that 75 percent of persons with silicosis will ultimately die of tuberculosis is obviously an error.

Since examining the situation in our own country in more detail, we have learned that the frequency of tuberculosis in different groups of employed silicotic subjects is quite generally higher than that in the rest of the population, that its incidence varies from industry to industry and from place to place in the same industry and

that, of the silicotics who die, tuberculosis is the most common cause of death. We have not yet learned whether the variation in the amount of tuberculosis in different places is due to the [illegible] action of other kinds of dust inhaled at the same time with the [illegible] whether the result merely reflects the opportunities for infection that exist in a particular group. Our own autopsy experience corroborates the findings in the South African gold mines. In a group of silicotic lungs from persons employed in a wide variety of [illegible] located in all parts of the country, the incidence of active [illegible] was 65 percent. In another group of lungs from [illegible] to different types of dust, which produced no specific [illegible] of silicosis, the incidence of tuberculosis was only [illegible] percent. [illegible] there has been considerable misunderstanding as to the actual number of cases of tuberculosis which may develop, figures such as these leave no doubt as to the importance of this complication. The directions which we must follow in our attempt [illegible] that has been done are clearly defined.

To prevent a continuation of this evil we must study the conditions under which men work in our industries, we must examine the applicant for employment to see that he does not already harbor latent tuberculosis and we must examine and re-examine the old employee at regular intervals to learn whether he is being injured by his environment. To accomplish these purposes we must have a trained personnel. Engineers must know how to detect and measure the amount of silica in the atmosphere; they must know how to reduce the concentrations if they are excessive. Physicians with a knowledge of industrial conditions must be able to detect evidence of silicosis and tuberculosis. Management must appreciate its responsibility and be willing to make sufficient expenditure to insure protection. Labor must be taught to realize that it must cooperate by accepting the medical examinations and by using the preventive devices that are offered. But ours is more than an engineering or a medical problem. Since the preemployment examination may exclude men from gainful occupation and since [illegible] involves compensation and in many, silicosis becomes a [illegible] problem as well. The insurance group, the legislator and the compensation official must likewise be informed. To disseminate information to all those concerned is the function of a meeting such as this.

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ETIOLOGY OF SILICOSIS

DR. R. R. SAVERS
U. S. Public Health Service

As the following letter from Dr. Savers indicates, his paper on "Etiology of Silicosis" is a reprint of his paper with the same title read at the second Silicosis Symposium in Saranac Lake in 1935. Its publication is approved by the United States Public Health Service. (Editor)

TREASURY DEPARTMENT
UNITED STATES
PUBLIC HEALTH SERVICE
Washington, D. C.

October 4, 1937

Mr. B. E. Kuechle
Vice-President and Claim Manager
Employers Mutual Liability Insurance Co.
Wausau, Wisconsin

Dear Mr. Kuechle:

I regret very much that I have not replied to your letter requesting a discussion of my paper "Etiology of Silicosis", presented before the group at Saranac this year.

As stated before that group, I am somewhat chagrined at being able to give little, if any, additional data on the etiology of silicosis, for in the time intervening between publication of the previous paper and the present there has been but little new information developed. Some of the things discussed in the former publication (see a discussion of my paper, page 31, in the Second Symposium on Silicosis) probably should be emphasized.

Various factors, including the chemical composition together with the amount of dust, and especially the amount of free silica in the air breathed by the men, is important. However, the methods of determining the amount are only important insofar as they give us an index of exposure which will be comparable with the indices of exposure determined by various observers. In this country we have used particle count correlated with chemical composition chiefly as an index of exposure.

In some countries, or locations, the weight of the dust in a given amount of air when correlated with the chemical composition is used as an index. Each method has its advantages and disadvan-

[illegible] Either can be used satisfactorily as an index of exposure and [illegible] of control equipment.

In the Public Health Service both methods have been used and [illegible] between the two methods have been and [illegible] some cases. It is usually unnecessary to try to use both methods. A simpler method of determination of exposure is still desirable. In the meantime it seems best to continue the count method (in [illegible]) as an index of silicosis hazard.

The relationship of infection, especially tuberculosis to silicosis, may be further emphasized as moderate or marked disability (compensable disability) seldom develops without infection being a factor. Drs. Watkins-Pitchford, Irving, Stewart, of South Africa, noted that tuberculosis was an accelerator of lung fibrosis. Fertell demonstrated that tuberculosis superimposed upon silicosis produces a greater degree of fibroid development than does either disease alone. This can account, at least to some extent, [illegible] disability when infection supervenes. Sight [illegible], however, of the fact that silicosis is a disease entity and [illegible] in character—for a time at least—after exposure to [illegible] has ceased.

No advantage has been demonstrated in discharging [illegible] uncomplicated silicosis (simple silicosis) from their regular jobs, provided that measures to control the dust and medical examination at regular intervals have been instituted.

As stated, there is but a little amount of factual data regarding the progress of silicotic changes. The data in this country [illegible] to show the degree of progress after removal from exposure. It is possible that silicotic nodules of microscopic dimensions may progress to a size sufficient to cause a shadow in a roentgenogram after cessation of exposure. Coalescence of such nodules, in the absence of infection, has not been reported after cessation of exposure. However, infection may supervene, and from data at hand it is the apparent cause of the progression of fibrosis.

The basic data on etiology of silicosis, I believe, was [illegible] covered in the former paper.

With my best personal regards, I am

Cordially yours,

R. R. Savers, Senior Surgeon
Chief, Division of Industrial Hygiene
National Institute of Health

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ETIOLOGY OF SILICOSIS

R. R. SAYERS AND
R. R. JONES

Etiology of silicosis is the study of the causes of silicosis or it is the sum of the knowledge regarding the causes of silicosis. Silicosis has been variously defined. Dorland (Dict. 16th ed. 68) "Pneumonoconiosis is due to inhalation of the dust of stone, sand or flint;" (Webster's New International Dictionary, 1930, pp 1936) "An affection of the lungs occurring in stonecutters, caused by the inhalation of quartz dust".

West Virginia, in the law recently passed, for the purpose of the Act defined silicosis as an insidious fibrotic disease of the lung or lungs, due to prolonged inhalation and accumulation sustained in the course of and resulting from employment, of minute particles of dust containing silicon dioxide (SiO_2) over such a period of time and in such amounts as result in the substitution of fibrous tissue for normal lung tissues; and the term silicosis shall also include silicosis accompanied by tuberculosis of the lungs evidenced by the presence of tubercle bacillus in the sputum.

The Committee on Pneumoconiosis of the Industrial Hygiene Section of [illegible] the American Public Health Association recently defined silicosis as

> a disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis, (some or all of which symptoms may be present), and by characteristic X-ray findings.

While the foregoing definitions were obtained from sources that might be expected to have somewhat different views, as the medical man's, the general public's, the lawyer's, and the preventive and industrial physician's, all have one thing in common: that the cause is silica or quartz.

An accurate knowledge concerning the etiology of any disease is one prerequisite to its effectual prevention, diagnosis and treat-

ment. A certain amount of general information relative to the pathology resulting from the inhalation of dust has been in existence since the time of Hippocrates (400 B. C.). Some of the early writers discussed such diseases of [illegible] the general term Pneumoconiosis. Professor Collis [illegible], in his 191[illegible] [illegible] furnished us with an excellent historical review of the subject of industrial pneumoconiosis. However, it was only late in the 19th and early in the 20th century that clinical and experimental [illegible] were carried out, the results of which have yielded rather definite knowledge concerning the causes of silicosis. Rovida [illegible], in 19[illegible] [illegible] term silicosis in reporting a case of pneumoconiosis, in which [illegible] the condition was due to silica, by chemical [illegible] of the lungs.

Though other dusts, when inhaled in sufficient concentration over a long enough period of time, have been [illegible] producing a definite pulmonary fibrosis, nevertheless [illegible] characterized by nodular fibrosis to date has been [illegible] and experimentally to be associated only with the inhalation of dusts containing silica. Moreover, it has been established that exposure to dusts consisting wholly of free silica (quartz) produces this disease, which has not been shown to be the case for any other specific dust.

SILICA IN NATURE

Silica is the most abundant constituent of the minerals and rocks that make up the crust of the earth. It occurs in two forms, free and combined. The free silica, as a group, are definite compounds in the form of SiO_2. The combined forms are [illegible] silicates. Of free silicas which occur in nature, that [illegible] quartz is by far the most common. Quartz is a hard mineral and chemically resistant to reagents. It is an abundant constituent of granite, schist, and other rocks and the chief component of sandstone and quartzite. Many ores are deposited in veins that consist nearly wholly of quartz. This form of free silica exists in two polymorphous forms, low and high quartz. Low quartz is stable at temperatures below 573 degrees C, and high quartz is stable between 472 and 870 degrees C. At temperatures below 573 degrees C, high quartz spontaneously changes to low quartz, so that practically speaking, distinction between the two classes is of little importance.

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Probably the next most common form in which silica exists in nature is the amorphous hydrated form known as opal ($SiO_2 \cdot H_2O$). Opal is a silica of colloidal origin and occurs almost only in the diatomaceous earths. It is less common in occurrence than quartz. Another type of free silica frequently found in flint, and with flint is found chalcedony, a waxy, translucent form of silica interpreted as consisting of fine fibers of quartz with a small amount of interstitial opal. Other forms of free silica occurring less abundantly in nature are tridymite, cristobalite and siliceous glass or vitreous silica.

OCCUPATIONAL EXPOSURE TO SILICA

Owing to the fact that the earth's crust contains so great an amount of silica, it is obvious that the occupations concerned with the driving of tunnels, development of lodes, ores, and mining, are frequently associated with a silicosis hazard. A second class of occupations exposing the workers to the hazard are those connected with industries that have to do with the processing and industrial use of mineral products, such as, the smelting and refining of ores; the use of sand and gravel for construction purposes; the carving of stone, particularly granite; the manufacture and use of abrasives; and the processing of the various forms of free silica. According to Knopf[9], the most common forms of free silica used industrially are massive crystalline quartz, quartzite, sandstone, flint, tripoli, diatomaceous earths and silica sand. The following table from Ladoo[10] illustrates the great variety of uses to which silica is put in industry, and the kind of silica adapted to each purpose.

TABLE I

OCCUPATIONAL EXPOSURE TO SILICA

Uses of silica	Form of silica used
Abrasive uses	
Scouring soaps and polishing soaps and powders	Quartz, pumice, tripoli, diatomaceous earth, infusorial earth, flint, chert, sandstone, quartzite, feldspar, pumice
Sandpaper	Quartz, pumice
Emery wheel work	Emery, quartz, corundum, feldspar, quartzite, sandstone, flint, chert, pumice
Sand blasting, burnishing, and polishing	Quartz, quartzite, sandstone, flint, chert, pumice
Scouring and polishing powder, soaps, etc.	Diatomaceous earth, infusorial earth, pumice, quartz
Metal polishes, lubricants, and compounds	Quartz, diatomaceous earth, infusorial earth, pumice
Building uses	
Building stone	Granite, sandstone, limestone, quartzite, marble, slate
Silica brick, refractories, cement	Quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth
Lining mill grinding pebbles	Quartz, flint, chert
Insecticide powders and pastes	Various forms of free silica, pumice, diatomaceous earth, infusorial earth
Paint filling and finishing	Various forms of silica, diatomaceous earth, infusorial earth, pumice
Chemical uses	
Glass making: In making silica brick and other refractories	Pure quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth, pumice
Glass	Pure quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth, pumice
Silicon, ferrosilicon, and alloys of other metals, carborundum	Pure quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth, pumice
Other nonsilicate basic ores	Pure quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth, pumice
Lime mold wash	Pure quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth, pumice
Core sand, facing sand	Pure quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth, pumice
Foundry uses	
Furnace for acid covers	Pure quartz, quartzite, sandstone, flint, chert, diatomaceous earth, infusorial earth, pumice
Filtering medium	Pure quartz, diatomaceous earth, infusorial earth, flint, chert, ground glass, pumice, sandstone, quartzite, feldspar, other forms of free silica
In the manufacture of sodium silicate	Pure siliceous sand, quartz, diatomaceous earth, infusorial earth, pumice
In the manufacture of carborundum	Pure quartz sand
Paint: As an inert extender	Ground quartz, diatomaceous earth, infusorial earth, pumice, flint, chert, sandstone, quartzite, feldspar
Mineral fillers: As a wood filler	Pure ground quartz, diatomaceous earth, infusorial earth, pumice, tripoli, flint, chert, sandstone, quartzite, feldspar
In fertilizers	
In insecticides	
In rubber, hard rubber and colored and molded goods, phenol, etc.	Pure ground silica, quartz, diatomaceous earth, infusorial earth, pumice

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TABLE 1 (Continued)

Uses of silica	Forms of silica used
Special asphalt and paint mixtures	
Ceramic uses: In the pottery industry for the manufacture of bodies and glazes	Flint, quartz, and other forms of silica are preferred, although all other forms of silica may be used to a limited extent
In the manufacture of carborundum electric resistance furnaces	Pure quartz is preferred
In the manufacture of fused quartz: chemical apparatus, such as tubes, crucibles, and dishes	Pure quartz is preferred
Decorative materials: In the manufacture of gems, crystal balls, table tops, vases, statues, etc.	Rock crystal, vein quartz, some massive quartz, chalcedony, agate, jasper, flint, and, more rarely, opal, etc.
Explosives: Dust mixtures for paper bodies, fuses, etc.	Powdered crystalline silica, such as quartz
Sand blasting: Sandstone for cleaning castings, etc.	Usually a fairly clean, uniform, fine-grained sandstone containing a high percentage of finely pulverized silica
Optical quartz: For the manufacture of lenses and prisms for optical apparatus	Only the purest, flawless rock crystal or massive crystallized quartz

In a recent survey[5] carried on in a large manufacturing center, it was found that about 9 percent of the industrial workers were employed in occupations where the silica hazard required consideration. According to the census for 1920, there were gainfully employed in the manufacturing and mechanical industries in the country approximately 14 million persons. If the above survey can be accepted as representative of the occupational distribution of these workers, it appears that there are nearly 1,500,000 individuals potentially exposed to a silicosis hazard in the manufacturing and mechanical industries alone. Lanza and Vane[6], in their discussion concerning the prevalence and effect of silicosis, state, "Our very rough, but obviously conservative, estimate of the number of workers exposed to silica dust to a harmful degree in the United States is, therefore, upwards of 500,000."

FACTORS INFLUENCING THE ACTION OF SILICA DUST PARTICLES AS THE EXCITING CAUSE OF SILICOSIS

Although it has been accepted that silica is the exciting cause of silicosis, there are certain factors which must be considered as to their importance in its action.

Early workers were inclined to consider that the injury produced by the dust particle was due to mechanical irritation. Based upon its hard and cutting edges, Gardner[7] has shown experimentally that the inhalation of finely divided carborundum dust, of greater hardness than silica particles, does not produce the pulmonary reaction characteristic of silicosis. Collis[8], one of the early writers to call our attention to the chemical action of dust, [illegible] that silica in solution or powder [illegible] action upon the tissues which [illegible] cells. Lately Miller and Sayers[9] [illegible] experimental studies which illustrate clearly the [illegible] chemical reaction to certain dusts. Only the silica-containing [illegible] Lanza[10] produced a proliferative reaction. Other [illegible] were completely absorbed, leaving no [illegible] and [illegible] in the form in which they were injected. The [illegible] classed as "dust tolerant."

[illegible] dust to cause injury depends upon the [illegible] of the lung, the particle size of the [illegible] absolute relationship to the minimum effective [illegible] be present in the air in particles of [illegible] sizes and of such dimensions that they [illegible] from our knowledge of the [illegible] unable to handle bodies greater than ten microns in [illegible] The normal defenses of the [illegible] my particles larger than ten microns [illegible] other parts of the lung, and the [illegible] bronchial secretions. The solubility [illegible] the production of the disease and the [illegible] also affects the rate of solution, due to the fact that the smaller the particles the greater the total surface area exposed to the [illegible] solvents.

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TABLE 1

SIZE FREQUENCY DISTRIBUTION OF VARIOUS INDUSTRIAL DUSTS AS COMPARED TO OUTDOOR DUST

Kind of dust	Number of samples	Average frequency in percent—size group in microns											
		0-5	5-10	10-15	15-20	20-25	25-30	30-35	35-40	40-45	45-50	50-55	55-60
Silica rock crusher	20	20	20	20	15	10	5	5	3	1	1	0	0
Silica

settle out rapidly while the rate of fall of particles is very slow. For illustrative purposes, the dust may under Imperial Valley at a rate of from 1 to 3 feet per hour, with the specific gravity, while a particle 5 microns in diameter having a specific gravity of 2 falls about 60 feet per hour. Particles of more than 10 microns would settle out in a relatively short time. The fact that the finer particles remain suspended in the atmosphere for long periods greatly increases their chance of being inhaled.

How we may say from the viewpoint of etiology, the harmfulness of a given dust containing free silica is directly influenced by the number of particles of free silica less than 10 microns in diameter that it contains. Probably the greatest harm is produced by particles of from 1 to 3 microns in diameter.

The relationship of dust concentration and duration of exposure are closely associated in their etiological significance. The rate at which silicosis will develop, excluding certain factors considered as predisposing, depends upon the degree of free silica. This degree is obviously dependent upon the amount of silica in the air inhaled, and the duration of exposure.

In 1902, a committee of which Dr. J. S. Haldane was a member (23), reported upon an investigation made to determine the cause of excessive mortality rates from tuberculosis among the Cornish tin miners. This group decided that it was evident that the inhalation of stone dust by these men in the performance of their tasks as miners, was the cause of permanent damage produced upon the lungs. Furthermore, they noted that the condition developed gradually in the case of the ordinary miner, but rapidly in the case of the machine workers who were exposed to greater amounts of dust. Dr. Watkins-Pitchford(19) and Dr. Mavropoulo(19) and other South African workers, in their discussions relating to the etiology of silicosis, emphasize the relationship of the concentration of dust and duration of exposure to the degree of lung changes produced. Mavropoulo has suggested the advisability of intermittent employment as an aid in lessening the amount of changes produced, but aside from the fact that it does delay the process, it has not been shown that it could safely be relied on to prevent silicosis, where exposure is to sufficient concentrations. It is intimated that the rest interval allowed by intermittent employment permits the lung to free itself of some of the particles. The value of this avenue of elimination

[illegible] and the [illegible] for exposure [illegible] and dust are more readily eliminated than those containing a high percentage of free silica. Once the dust is taken up [illegible], the direction of the dust cell in its expulsion from the air passages of person's throat and the lung [illegible] as well as [illegible] the [illegible] of the bronchioles, and some portion will be removed. This rest interval may permit the subsidence of the primary reaction which accompanies the inhalation of foreign particles into the lung passages. Workers in this country have also shown the relationship of the age to the severity of the reaction. Reports of the Picher Group(20), of the Tri-State District of Oklahoma, Kansas and Missouri lead and zinc mines, which is a cooperative undertaking between the operators, the Metropolitan Life Insurance Company, the Bureau of Mines, and the Public Health Service have shown clearly the effects of various periods of exposure to dust with in the mining processes carried on [illegible] upon the health of granite workers [illegible] the duration of exposure necessary to produce [illegible].

Some authors have expressed the opinion that the presence of other inorganic dusts in the silica containing atmosphere may tend to influence the effects of the silica (18,24). The exact role of these accompanying dusts remains to be determined. Hefferan reported the absence of silicosis among a group of [illegible] in a gannister brick manufacturing establishment, where the material used contained more than 80 per cent silica. However, I do not now consider this as evidence that, as he suggests, the presence of other inorganic dusts, along with the silica present, lessens the harmful silica reaction, there are several lines of additional information which must be supplied. First, he did not show that the men were really exposed to harmful concentrations of free silica. In the manufacture of these bricks, the initial step includes the wet mixing of the materials and it is possible that sufficient exhaust ventilation was maintained to prevent the dispersion of free silica into the atmosphere. In fact, Dr. Hefferan states that the exhaust [illegible] the mixing room where this process was carried on [illegible] dust. Later operations were of such a nature that the quantity of free silica which may have been present could become [illegible] modified to such an extent that [illegible] free silica [illegible]. [illegible] to determine the concentration of [illegible] and

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physical properties of the dust in the air [illegible] would aid greatly in scientific interpretation of [illegible].

Some have thought that the relative absence of silicosis in the cement industry was due to the calcium present. However, investigations[29] have led to the belief that the absence of evidence of extensive pulmonary fibrosis among employees in the cement industry is due to the fact that these workers were not exposed to free silica (considering concentration of dust and duration of exposure).

No complete reports of pulmonary fibrosis resulting from the inhalation of organic dusts have appeared in the literature. Always when such fibrosis is associated with the breathing of air containing organic dust, either mineral dust has been shown to be present or at least its absence not definitely proved.

In regard to the presence of asbestos or coal dusts, Chapman[30], Fenkel[31], MacDonald[32], Edgecumbe and others have reported cases of so-called acute silicosis, resulting from the inhalation of air containing high concentrations of silica along with strong alkali in a fine powdered form. Some have suggested that the rapid action of the silica in the production of pulmonary fibrosis is aided by the presence of these alkalies, because silica is more soluble in alkaline solutions. Kettle[33] failed to demonstrate such actions experimentally and states that proof is still to be given that any action of the kind may occur. Pathological reports from the existence of a definite fibrosis, but the entire picture differs greatly from that of the typical silicosis, such as has been furnished by Gardner[20] in his reports upon the examination of tissue from several cases of silicosis as well as his pathological reports made from tissues obtained in his experimental studies.

Sayers, Meriwether and Lanza[34] reported that of the employees in the lead and zinc mines, those who have a history of previous coal mining experience developed asbestosis in a shorter time than employees of the same age without this experience. This cannot be taken to mean, however, that the inhalation of coal dust hastened the action of silica, for it is doubtful if any of these coal miners had worked where they were not exposed to some free silica, so the total dosage of silica was not the same in the two groups.

OCCUPATIONAL HISTORY

An important factor in the [illegible] and exposure to coal dust, exposure of a complex [illegible] history.

TABLE [illegible]
OCCUPATIONAL RECORD

[illegible] August 8, 1931	[illegible]
[illegible] M	[illegible]
[illegible] work: 35	[illegible]

Specific [illegible]

Occupations [illegible]

Silica [illegible]

[illegible]	
1	Coal cutter, miner (chamber)
2	Driver, helper (chamber)
3	Mule driver (dry mines)
4	Loader (dry mines)
5	Shot-firer (dry mines)
6	Farm laborer (Q.v.)

Periods [illegible]

By recording the complete data, the total dust exposure [illegible] determined and compared to other [illegible] of employees.

The following table shows the [illegible] pations of chamber miners and [illegible]. It will be seen [illegible] activity engaged in by these coal miners was studied [illegible] Experience has shown that the various occupations [illegible] process of any dusty occupation are usually associated [illegible] dust exposures.

TABLE [illegible]
DUST EXPOSURE OF COAL MINERS [illegible] OCCUPATIONS [illegible]

[illegible]	Number of [illegible]	[illegible]
Coal [illegible] drilling	1	[illegible]
[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]
Timbering and wiring	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]
Totals	6	[illegible]

[illegible] million particle hours per cu. ft. [illegible] particles per cu. ft. 6 hrs.

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It is apparent from this [illegible] contributes about 73 per cent of the [illegible] miners and laborers, although this occupation [illegible] one-third of the total working hours. Tick hammer drilling, which occupied but 1 hour of the 6½ hour [illegible] day, amounts to 18 per cent of the total exposure, while the practice of entering a room too soon after blasting unduly exposes miners to a large amount of dust, this quantity being about 7 per cent of the total dust exposure. Table 4 shows the method of analysis used to determine an estimate of the individual's total dust exposure.

TABLE 4
ESTIMATION OF THE INDIVIDUAL'S TOTAL DUST EXPOSURE

Occupation	No. of years	Dust concentration in millions of particles per cubic foot (average)	Millions of particle-years per cubic foot
Slate pickers (dry mine)	2	[illegible]	760
Leather (dry mine)	2	71	132
Mole shoes (dry mine)	3	71	213
Miners' laborer (chamber)	3	[illegible]	1110
Contract miner (chamber)	15	[illegible]	7200
Section foreman	5	7	35
Total	30		9290

More detailed application of our knowledge of the true cause of silicosis is illustrated in the following table. In this study of the

TABLE 5
DUST EXPOSURE OF MOLDERS

Activity	Time of exposure in minutes (h)	Average dust exposure in millions of particles per cubic foot (h)	Millions of particle minutes (a × h)
Use of parting compound	[illegible]	[illegible]	1435
Remaining task in molding	412	4.4	1813
Pouring	58	3.1	180
Dumping molds (shake out)	16	[illegible]	620
Total	[illegible]		5958
59.[illegible] million particle minutes / 540	[illegible]	[illegible]	

actual duties required by the job (molder), it is revealed that the greatest hazard is that in connection with the use of a parting com-

pound. It is obvious that the risk [illegible] must do can be materially lessened by either [illegible] pound containing less silica or in properly safeguarding [illegible] by adequate dust control during [illegible] operation. [illegible] pational histories available, and the knowledge of [illegible] total dust exposure revealed by detailed occupational [illegible] is found possible in a recent study to prophesy [illegible] approximately the conditions that would be found [illegible] examination in cases where the total changes were due [illegible] inhalation.

PREDISPOSING CAUSES

Eight countries were represented at the [illegible] held in Johannesburg in [illegible] recent bibliographic [illegible] pneumoconiosis lists references to the literature [illegible]. The disease occurs in the pottery, foundry [illegible] granite, tool and axe grinding, glass, slate, silica grinding [illegible] industries.

Since the literature of practically all the principal [illegible] contains articles on this subject, it is apparent that [illegible] is exempt, and that all races are [illegible] of silicosis. Although the data show [illegible] among the younger miners in districts where the percentage of free silica is high, and among older miners where the percentage of silica is low, age in itself probably is no great factor.

Previous occupation of the men may have a [illegible] in predisposing to silicosis, if they have been exposed to [illegible] other respiratory irritants.

Since respiratory infection has been shown to be [illegible] predisposing and complicating factor in the development [illegible] certainly the history of present and past respiratory infections will have to be given consideration in the statistical analysis [illegible] upon which such conclusions are based.

The factor of individual susceptibility is often [illegible]. Generally speaking, if there be any difference in individual susceptibility, it can usually be considered an acquired [illegible] condition. Ickert[illegible] quotes Bohme and Lucanus and [illegible] as stating that it is essential that the individual possess [illegible] functioning nasal passageways, in order that the self [illegible]

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mechanism may work efficiently. [illegible] fact that Irvine, Simpson, and Strachan report the [illegible] type of silicosis to be more common among the robust type of individuals with less respiratory reserve. Tickert found some slight variation in the susceptibility of persons according to their type of body build. As a whole the group classed as slender individuals developed simple silicosis somewhat slower than the stoutly built persons, but the incidence of advanced silicosis was greater in the former class, being nearly double that developing in the [illegible].

Lehman's[26] experiments to determine the functional efficiency of the upper respiratory tract, in the removal of dust, suggests that abnormalities of the nasal passage [illegible] probably play some part in the rapidity with which silicosis [illegible]. Using dust with a high silica percentage, he found that [illegible] to 73 [illegible] percent of the dust was retained during passage of air through the nose. In carrying out this experiment upon 62 miners, he found that the average retention by the nose in the cases of non-silicotics was about 50 percent while in the case of miners with silicosis, the average retention was only about 22 percent.

Chronic bronchial asthma may be considered a predisposing factor affecting individual susceptibility. The spasmodic attacks, if frequent, lead to a reduction in the individual's vital capacity. Aside from the pulmonary fibrosis, other pathological manifestations of silicosis such as bronchiectasis, emphysema, and right heart hypertrophy and dilatation, may be aggravated by this chronic condition.

Chronic infections of a local or constitutional nature may be shown to influence materially the incidence of silicosis.

As regards infection in general, Lanza[illegible] has stated that in all his experience he can recall seeing but one patient who died of pulmonary fibrosis uncomplicated. The results of the survey in the Tri-State district emphasized the role played by infections in disabling silicosis, and mentioned the frequent occurrence of bronchial spirochetosis. Recently, Proske[illegible] has published reports of additional work along this same line which should do much to stimulate the taking of preventive measures to control such infections in the sinuses and dental regions as may serve as a source of these organisms.

Infections developing along the respiratory tract are of importance. Sinus infections may act by decreasing the efficiency of the upper respiratory tract in the removal of dust from the air passing to the lungs, and also they may be the source of infections that spread to the lower respiratory tract. Acute pneumonic conditions as well as the more chronic lung changes such as chronic bronchitis, bronchiectasis, and bronchiolectasis, emphysema, and pleurisy, all tend to decrease the ability of the lung to rid itself of foreign materials, through lessened lymphatic drainage and decreased power to force the bronchial secretions and foreign matter from the lungs. The dilated bronchi and areas of emphysema [illegible] as a result of abnormal demands made upon [illegible] [illegible] professional singers, trumpeters, and the [illegible] advance to the point where the [illegible] offers [illegible] infection is present.

Christie[72], in a recent review and [illegible] of bronchiectasis emphasized the infective elements [illegible] other pathological changes associated with the condition. The lung changes accompanying silicosis might be expected to lead [illegible] favoring bacterial invasion. [illegible] writer [illegible] with [illegible] believe that the [illegible] of fibrosis seen [illegible] appearing as conglomerate [illegible] dependent upon an infective process, either [illegible] absorption of silica or developing in an area of pulmonary [illegible] due to the action of silica.

TUBERCULOSIS AND SILICOSIS

In 1905, Summons[30] of the Miners' Phthisis Commission of Australia, reported that gold miners there who contract [illegible] of tuberculosis.

The increased incidence of tuberculosis [illegible] groups exposed to silica has been clearly shown in every instance where this hazard exists. Britten[illegible] summarized the report of the Registrar-General of England and Wales for [illegible], and [illegible] the occupational mortality rate for the group of trades [illegible] "Dusty Trades" to be from three to more than ten times [illegible] as the rate for all occupied and retired males.

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[illegible]
PENGAD-Bayonne, N.J.

TABLE 6

STANDARDIZED MORTALITY RATIOS FOR SILICOSIS IN OCCUPATIONS WHERE SILICOSIS IS KNOWN TO BE A HAZARD

MALES AGE 20-65, 1921-25, LIFE INSURANCE EXPERIENCE

Occupation	Mortality ratio (standardized)
All occupations combined	149.6
Iron ore (except taconite) and tungsten mines, underground (silicosis or tuberculosis as contributing cause) (III)	1,238.0
Iron ore (except taconite) and tungsten mines, not reported whether underground or not (III)	1,134.5
Granite workers in the quarry (code 1[illegible])	1,118.5
Coal miners (IV)	846.2
Stone cutters and dressers (III)	712.5
Stone [illegible] workers, quarry (III)	411.4

Lanza and Vane[9] show by one of the most dependable experiences of twelve life insurance companies for the period of 1911 to 1926, that the actual mortality from respiratory tuberculosis among the silica exposed persons was about three times that of a non-silica exposed group.[7] When the comparison is limited to the rates for some of the occupations with a very severe silica exposure, such as metal mining, sandstone and granite quarrying, the excess is still more striking, the rate being about ten times that obtained in the non-silica exposed group.

The initial studies of silicosis by workers in South Africa were started by a demand made upon the health authorities to determine the cause of the excessive mortality from tuberculosis which was increasing at a rapid rate among the gold miners there. The study by Russell and others[10] of the health of the granite workers was prompted in part by the excessive number of deaths reported as due to tuberculosis in the section of Vermont in which the granite industry is located.[7]

Gardner's[12] statement that, "At least 75 percent of those human beings who develop silicosis die of tuberculosis which may make its appearance at any stage of the disease," stresses the importance of the silicosis problem from the viewpoint of anti-tuberculosis activities. Rist and Denbron[13] advance the theory that there is no nodular fibrosis until the element of infection has entered the picture. Such a theory, however, does not agree with experimental work which has shown that the nodules may be produced in the absence of evidence of infection.

Kettle[14] and Price[15] and others have shown that the tubercle bacillus will grow more rapidly upon culture media to which a small amount of silica offers a favorable media for the growth of the organism. Gardner[16] has shown that normal experimental animals inoculated with a strain of tubercle bacilli which are of low virulence will develop systemic tuberculosis and die while control animals not so exposed usually are not seriously affected by injections of such organisms.

PROGRESSIVE TENDENCY OF THE DISEASE

Since infection has been shown to play so important a role in the advanced stages of the disease, the possible relationship of the infective element to the progressive tendency of the disease cannot be overlooked.

Irvine[17] has stated that it is not so much that the condition of the chest is today as what it will become tomorrow. Irvine emphasizes the tendency of the fibrosis to progress even though removed from exposure, and expresses the opinion that it is one of the most serious aspects of the whole silicosis problem. Pneumothorax has been showed to be of value in elimination of [illegible] in tuberculosis although certain improvement in symptoms may be noted after the patient is removed from exposure. According to the observations made by Pohme[18], silicosis progressed, after removal from exposure, in 20 percent of the cases diagnosed as advanced disease, in grade 1, in 30 percent of the cases in grade 2, while practically all cases in grade 3. The course of silicosis among workers no longer exposed to silica dust is adequately stressed in the report of Purcell[19] and the references discussed furnish evidence that even after removal from exposure, silicotics may eventually progress to a [illegible] and even fatal condition. Such reports from workers must [illegible] that with the condition surely serve to emphasize the seriousness of the occupational disease.

In concluding, it may be stated that to understand [illegible] the many etiological factors accounting for the development of [illegible] for instance requires detailed knowledge of the individual's occupational exposure past and present. [illegible] through a complete physical examination, and [illegible] must be given to positive evidence of either recent exposure or infection. Although a slight mention may be made of the predisposing causes, after all, in the great majority of cases, whether individuals will develop silicosis depends nearly entirely upon the concentration of free silica particles under 10 microns in diameter to which they are exposed and the duration of such exposure.

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Discussion of Dr. Sayers' [illegible]

When asked for his opinion of the [illegible] involved in mining coal, Dr. Sayers stated that while from an experimental standpoint both hard and soft coal dust [illegible] as harmless as limestone, gypsum and dolomite, he felt that [illegible] enough knowledge to state definitely how dangerous the [illegible] of coal actually is. In his experience pulmonary changes occurred in miners who had extracted coal from seams situated between layers of sandstone. In other words, he inferred that the silica [illegible] the sandstone produced the disease. With reference to the practice of rock dusting coal mines to prevent explosions, he felt that even a certain amount of combined silica is preferable to the danger from explosion. However, he pointed out that calcium dust [illegible] effective and their use does not create even a theoretical hazard.

Asked for his opinion on the action of sericite and other silicates in the production of silicosis, Dr. Sayers said he was certain from experimental studies and clinical observation that silicosis could be produced only by pure silica. Sericite is never encountered in industrial operations in a pure state but always is accompanied by silica. In 1919 Watkins-Pitchford and Mavrogordato called attention to the fact that sericite particles occurred in the lungs of silicotic individuals but they did not believe that these particles were the cause of the disease. Kettle is of the same opinion. Experimentally, the injection of sericite into the peritoneal cavity of guinea pigs produces a non progressive reaction like that produced by carborundum. Some of the other silicates, notably asbestos, do cause fibrosis; in the lung the reaction is linear in type. He felt that sericite plays a minor part, if any, in the silicosis problem.

Asked if iron ores contain silica, he said that some do and in such case a major hazard exists. Hematite itself, when injected into animals, causes an "inert reaction."

The question of urinary excretion of silica by silicotic subjects was brought up. It was asked whether excretion ever ceases in advanced silicotics who have left their dusty occupations. Dr. Sayers mentioned a man still excreting silica 7 years after his retirement. He thought that some silica might be excreted in the feces, but had made no determination of the amounts. The significance of silica excretion is unknown at the present time; it possibly indicates only an exposure to silica.

Dr. Sayers had not observed silicotic nodules in the [illegible]. Dr. Gardner cited one human case with microscopic nodules [illegible] in diameter, and mentioned that in other [illegible] microscopic foci.

Dr. Sayers could not state whether there was any relation between the amount of emphysema and the number and [illegible] of silicotic nodules in the lungs.

He believed that the "miner's asthma" of the coal miner was anthracosilicosis, i. e., silicosis modified by an excessive amount of carbon. It is a local term peculiar to the coal industry like "potters rot", "miners phthisis", "grinder rot" etc., in other trades.

In his study of the anthracite coal industry, 2% of miners exposed to dust concentrations of 300 million particles per cubic foot of air containing only 5% of silica developed silicosis. No disability was encountered where the concentration of the dust was only 50 million.

He was asked whether disability from silicosis could be measured physiologically before an x-ray diagnosis had been made and replied in the negative. The diagnosis, in his mind, depends upon the degree of disability, upon the [illegible] and upon all other physical findings. Taken alone, no one of these constitutes an adequate basis for a diagnosis.

It was asked whether disease of the nose and throat favored the development of silicosis. Dr. Sayers thought that it had little influence altho he had no actual proof. He quoted the work of Lehman, published in the J. of Ind. Hyg., to the effect that only 8% of inhaled silica is retained in the noses of persons having silicosis while 50% of the dust is retained by non-silicotic [illegible].

Prof. Drinker was requested to express his opinion of the significance of Lehman's work. He conferred his inability to evaluate it and stated that in correspondence the author had taken the same position. He had merely reported his findings and left it for others to draw conclusions. His method is open to criticism from a technical standpoint; in its favor is the large number of subjects used. (It should be borne in mind that the prolonged inhalation of silica might have as much effect upon the nasal mucosa as upon the lungs, and that the nasal mucosa of the chronic silicotic might therefore be a less effective filter for the retention of silica particles than that of a normal person. *Leroy U. Gardner, M. D.*)



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Dr. Savers was asked what per[illegible] of silicosis showed evidence of cardiac embarrassment [illegible] was subsequently raised many times during the course of the symposium, the various references will be considered in one place under the clinical aspects of the disease. Dr. Savers answered the question here by stating that in 15 years he had [illegible] only one case of silicosis not complicated by infection with cardiac embarrassment.

With reference to the prevailing dust storms, he said that he did not feel qualified to state how great a hazard was created by these storms. As far as he could determine, there was no danger from silicosis. The dust particles are often too large to be significant and many of them are classified as [illegible]. The influence upon acute respiratory disease is not so easy to define. There are no scientific data to prove that pneumonia has become any more frequent in areas where dust storms are prevalent. It was his opinion that dust storms would not prove to be an important factor in the production of chronic respiratory disease and probably not of acute ones.

Dr. Gardner mentioned his experience with experimental animals which developed acute pneumonia a day or two after being placed in dusting chambers where they were subjected to massive exposures. These animals presumably carried infections that were lighted up or caused to develop by the dust exposure; in its absence the infection might never have caused disease.

Mr. Cummings referred to the work of Brown and Sampson (Correlation of Clinical and Roentgenological Observations in Pulmonary Tuberculosis. Radiology, 22:1-14, 1934) indicating that in non-silicotic individuals the adult (apical) forms of tuberculosis practically never make their first appearance after the age of 25. If disease develops, some evidence of it is almost always present before this age. He then asked whether Dr. Savers believed that the same was true of the silicotic subject—whether this condition altered the "natural" limitation of age upon susceptibility. He also asked whether the tuberculosis first appearing in the silicotic after the age of 35 to 45 years was endogenous or exogenous in origin.

Dr. Savers replied that the question could not be answered decisively. He agreed that persons under 30 years of age should not be exposed to hazardous concentrations of silica for if they are infected with tubercle bacilli their infection might readily become

progressive. He inferred that while these endogenous infections occur, he leaned toward exogenous infection in the patient under 30 as the source of his disease. He cited the regulation of [illegible] under 30 from employment in the tuberculosis [illegible] of the U. S. Government Hospitals because they had in the [illegible] to much more tuberculosis than women over [illegible]. He [illegible] assumed that their infections were exogenous and derived from the patients for whom they cared. He said that the same [illegible] true in the presence of a silica hazard, which would still further increase the natural susceptibility of the early age period to exogenous infection.

When asked whether it would not be good policy to [illegible] employment under the age of 30, he replied that it would be much faster to clean up the plants and eliminate the hazard rather than discriminate against young men.

The question of variation in individual [illegible] was raised. Dr. Savers said that he could [illegible] 10 men of the same age the amount of disease that would [illegible] to an exposure to a given amount of dust.

He was asked whether a man who had developed a definite second stage silicosis and then left his dusty occupation would ultimately be disabled as a result of further progression of his disease. He replied in the negative citing the example of a man who had been away from his dusty trade for 10 years with no knowledge that his x-ray film showed extensive nodulation. The disease unquestionably progresses but without infection it does not [illegible] to symptoms. In his experience second stage, uncomplicated by infection, was not disabling.

Someone inquired whether the reaction to silica in the [illegible] continued to progress with a constant death of cells and replacement by new phagocytes and if, as a consequence, increased susceptibility to tuberculosis always remained at an exaggerated level. Dr. Savers replied that no one knows how long increased susceptibility to tuberculosis persists. He felt that since [illegible] of silica may be excreted "for 10 or 12 years" after leaving a dusty occupation, some cellular activity probably still occurs in the lungs. Dr. Gardner suggested that in the absence of a necrotizing [illegible] process most of the dust presumably is walled off inside of [illegible] fibrous nodules after a man has been away from his dusty occupation

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for a period of years. Therefore [illegible] dust particles would be expected. [illegible] are frequently carried into the substance of the silicotic nodule where they grow without restraint. In his opinion [illegible] susceptibility to tuberculosis would be present as long as [illegible] silica and the necrosis which it produces in the body. If all of the necrotic areas are calcified, as sometimes happens, probably this susceptibility diminishes or disappears. The seriousness of the progressive feature of silicosis depends upon the number of nodules present. If 200 pulmonary nodules should double in size during a 10 year period there would be little effect, but if 200,000,000 nodules did the same thing they might reduce the functional reserve [illegible].

When asked whether silica produces any type of malignancy, Dr. Sayers stated that he had no knowledge of it. Among 27,000 Picher cases not one showed evidence of malignant growth in the lung. Mr. Cummings mentioned two cases of carcinoma involving the lungs of silicotic iron miners, one of which was primary and the other metastatic. Apparently neither was caused by the silicosis. (In the 1932 report of the South African Miners Phthisis Medical Bureau 0.37% of 1083 silicotic white miners showed pulmonary cancer; among 1109 non-silicotic white miners, the percentage was 0.54 and among 1023 whites not working underground there were 0.58%. *Leroy U. Gardner, M. D.*) If the silica were responsible more cases should be discovered in the thousands of men that are being examined in silicotic surveys.

In reply to the question "where are the most potent hazards from dust?" Dr. Sayers replied "Where the concentration of pure silica is highest." In this country probably the Tri-State District (lead and zinc mining) is the worst. Crusher plants, where there is no protection, are notoriously bad. In these cases, he felt that the incidence of disease depends largely upon the concentration and size of dust particles in the atmosphere, and that other factors that might be present underground (mine gases, etc.) play little part.

Dr. Russell of Timmins, Ontario, was asked whether crusher house conditions in the Porcupine area are comparable to those underground and whether the rate of development of silicosis in the two locations is the same. He replied that it took 3 or 4 times [illegible] develop disease in the surface operation as underground. He admitted that extensive precautions had now been taken to

improve conditions in the crusher [illegible] so that [illegible] were now greatly reduced.

A question on the fluoride theory of [illegible] the action of silica seems not to have been answered. [illegible] the Saranac Laboratory have thus far failed to confirm the hypothesis. Animals injected with a mixture of calcium fluoride and approximately 10% of silica particles have failed to show [illegible] of progressive lesions after about 3 months observation. Intraperitoneal injection of the same fluorspar-silica mixture has produced no fibrosis in 5 months. Animals fed daily with [illegible] doses of calcium fluoride develop silicosis no more rapidly [illegible] than normal animals in the same room [illegible] *U. Gardner, M. D.*)

Someone asked whether there was any relationship [illegible] silicosis and generalized arteriosclerosis. Dr. [illegible] that blood pressures had been taken upon all [illegible] the U. S. P. H. S. and that neither his service nor [illegible] which he was aware had been able to demonstrate a relationship between the two conditions. With reference to certain reports of an [illegible] particularly in coal miners, he felt that [illegible] others had been too small to warrant conclusions.

In reply to a question as to the influence of [illegible] development of silicosis he stated that it had none. [illegible] polishing and in packing soap powders and other dusty [illegible] products develop silicosis. The diagnosis has also been [illegible] women residing in mining towns where their [illegible] between great piles of mine waste. Female animals [illegible] as rapidly as males.

Solubility Hypothesis.—The chairman called attention to the fact that Dr. Sayers had indicated that there [illegible] as to the validity of the solubility hypothesis. He asked Dr. Sayers whether he believed that the hypothesis had been proved and the latter replied that in his opinion it had neither been proved or disproved but it was a useful basis for explaining the evolution of silicosis.

Dr. Gardner stated that he himself had found [illegible] in reconciling many observations with the hypothesis. He mentioned the fact that cellular reaction developed so rapidly after the injection of suspensions of silica particles that it was hard to believe that

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much chromium had occurred and he also [illegible] recorded to show experimental evidence the most [illegible] to prove that the more soluble forms of silica [illegible] were much less active in the tissues than the less soluble crystalline quartz. He cited observations by Kratt and King which had been substantiated by Mr. Dworski in the Saranac Laboratory, which showed that the concentration of silica in the blood varied [illegible] in different individuals, indicating that if it were [illegible] circulated through the body it was probably eliminated [illegible]. In bone excised, silica exerted no demonstrable [illegible] Tuberculosis of rather [illegible] or experimental [illegible].

He cited further unpublished [illegible] by Dworski which made him hesitate to believe that [illegible] excreted in the urine was derived from inhaled crystalline [illegible] deposited in the lungs. Dworski had made repeated tests of the amount of silica in the urine of 28 normal individuals and had shown that the daily excretion varied widely. (The figures varied from 5.2 to 25.3 mgs. in 24 hours. Expressed as mgs per 100 cc of urine the variation was between 0.57 and 2.64). Investigation of the diet had indicated that this was the probable source of the silica in the urine. Vegetables and beer were particularly high in silica. Analysis of 2 popular brands of beer showed 8.87 mgs. per 100 cc in one and 14.74 in another. One subject, whose average excretion was 12.67 mgs. in 24 hours, eliminated an average of 28.9 mgs of silica after drinking one bottle of beer the preceding evening. Another, excreting 10 to 15 mgs., increased to 40 mgs after drinking beer. Silicotic subjects likewise showed great variation in the amount of silica eliminated in the urine and this variation was influenced by the amount ingested. The net result of his observations to date indicated that the average difference between the silica excreted by normal persons and that in the urine of silicotic subjects was not as great as the fluctuations due to variations in diet.

Prof. Drinker would not express a definite opinion on the subject but mentioned the fact that Titus in his laboratory had failed to demonstrate an appreciable amount of solution of silica in blood serum whereas Emmons and Wilcox had recently reported a very considerable degree of solution in the same medium.

The chairman recalled that the Harvard observers had attempted to separate particulate silica from the serum by centrifugation at 3500 r. p. m. for 15 minutes, a procedure which is notoriously diffi-

cult in the case of particles from 1 to 10 microns in diameter. Dworski had corrected Titus' work and shown that his low figures for dissolved silica were due to the exclusion of most of it by filtration through celluloid.)

Mr. Cummings concurred in the conclusion of Dr. Lehmann that the physical state of the silica was apparently the factor which determined its capacity to provoke reaction in the tissues. Silica combined with water to produce a variety of forms which were not always distinguishable by the physical chemist. Experiments carried on to show that silica in the proper [illegible] was [illegible] with increasing degrees of hydration. Anhydrous crystalline silica, which was the least soluble form, was the most active in causing tissue changes. In his opinion the answers of chemical reaction on the surface of the silica molecule. Dr. Gye's demonstration that the surface of quartz had the peculiar property of absorbing unimolecular films of [illegible] substances offered an explanation of the [illegible] by which silica particles affect the tissues. Silica surfaces might also provide [illegible] and lower resistance to tuberculous infection. In municipal [illegible] might be absorbed and accentuate the harmful action of silica. (It was known that drillers were usually more prone to develop tuberculosis than other underground workers and it was also known that chronic pneumonia could be produced by the aspiration of mineral oil.)

The chairman then mentioned that the inhalation of fine sprays of mineral oil administered for 1½ hour daily over a period of 2 to 2½ years had failed to cause chronic pneumonia in guinea pigs. Experiments in which animals inhaled a combination of silica and oil were contemplated but not yet begun. He called attention, however, to the fact that it was perfectly possible to coat silica with quite silica without any oil, both under industrial conditions [illegible] experimentally. (It was not mentioned by the time but [illegible] phospholipids, to be discussed below, might be absorbed on the surface of silica particles.)

He was asked whether there was any evidence to substantiate H. Freeman's theory, that freshly fractured quartz is more active than the same material after it has remained in contact with air for [illegible] lapsed periods.

Mr. Cummings recalled that in the laboratory calculations [illegible] had been performed with finely ground silica particles [illegible]

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large quantities. Two tons of this material [illegible] for a period of several years and it had [illegible] different experiments. There was no indication that the silica lost any of its potency with age.

Dr. McKenty reported that he had just come from the Banting Institute where he had seen a new type of [illegible] apparatus which produced uncontaminated, freshly fractured quartz dust by grinding lump quartz in a quartz-lined mill. Concentrations of 4,000 to 8,000 per cc. or 112 to 224 million per cubic foot of silica particles, less than 5 microns in diameter, were maintained. Rabbits exposed in this apparatus developed silicosis in [illegible] months. Dr. Irwin proceeded on Heffernan's hypothesis that freshly fractured silica existed in the form of SiO_2 and not SiO and that unsatisfied oxygen atoms either interacted with the tissues to produce fibrosis or took up water to produce a poisonous gel on the surface of the silica particle. On the theory that the unsatisfied oxygen atoms might be made to combine with something other than tissue radicals, Denny, Robson and Irwin introduced [illegible] of metallic aluminum into their quartz dust and prevented the development of silicosis in rabbits (see Canadian Med. Assn. Jour. July [illegible]). Dr. McKenty proposed that the same theory might explain the [illegible] of silicosis in foundry operatives, who work with [illegible], and its common occurrence in drill runners in mines.

Dr. Enzer commented upon the [illegible] of the silica in the blood and suggested that possibly the silicotic lesions then elsewhere might reabsorb soluble silica so that it failed to circulate in appreciable concentration and produce injury in remote organs. To support this view he stated that he had failed to discover silicotic nodules either in the spleen or the bone marrow of 15 human cases which he had studied very carefully. Dr. Gardner confirmed this observation from his own experience but pointed out that nodular fibrosis only occurred where particulate silica was concentrated in sufficient amounts. He mentioned that he had seen one or two human cases in which fibrous nodules had developed in the spleen and liver. Such cases constituted rare exceptions and were probably a result of associated infection. In guinea pigs, on the other hand, lesions regularly developed in these viscera, probably because of anatomic peculiarities.

Discussion of the Nicol-McNally theory of the action of fluorides in accelerating solution of silica was proposed.

Dr. Gardner replied that he had had a group of [illegible] which [illegible] living in a quartz dusting room fed every day [illegible] of calcium fluoride until the theoretical lethal [illegible] during the last two weeks before they were killed. [illegible] 11½ months they developed appreciable [illegible] controls not so fed. The experiment was [illegible] peated but was now only in the 5th month, too soon for [illegible] action to have developed in the controls. He stated that [illegible] sions were reserved but that at the present time he knew of [illegible] dence which proved the Nicol-McNally hypothesis.

The chairman was asked to comment on a paper by [illegible] of the Banting Institute who suggested that the similarity [illegible] tubercles and silicotic nodules might be explained on the [illegible] in such disease a non-specific irritant [illegible] liberated that [illegible] nodule formation. Dr. Gardner recalled that he had published [illegible] paper in the American Journal of Pathology [illegible] pointing out the similarities of the two types of [illegible] by proliferation of the same type of cell, [illegible] degeneration with or without resultant calcification [illegible] with considerable fibrosis. Fallon had postulated [illegible] silica dust and the tubercle bacillus [illegible] causing the death of phagocytes and [illegible] tant might have developed. Chemical examination of the [illegible] dead areas showed an excess of phospholipids. [illegible] had demonstrated that such substances extracted from dead tubercle bacilli would provoke tubercle formation. Fallon then showed that phospholipids from cells killed by silica would likewise cause [illegible] formation when injected into animals. Fallon's observations [illegible] attractive theory deserve the attention of experimenters in this field.

Protective action of other substances. The chairman stated that he proposed to discuss the protective action of other [illegible] on the following day but since Dr. Sayers would not be present he would appreciate an expression of the speaker's view at the present time. He asked Dr. Sayers whether he believed that the [illegible] of silicosis, which he had observed in anthracite coal miners, [illegible] proportional to the concentration of silica in the [illegible] they inhaled and whether the coal that was present with the [illegible] in any way modified its action.

Dr. Sayers replied that he did not know. He had not [illegible]

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recorded sufficiently accurate descriptions of [illegible] formed dusts. He was satisfied that most of the [illegible] investigations of anthracite mine dust which [illegible] less than 5 million particles per cubic foot did not develop [illegible] silicosis in a workable time. He was undecided as to whether the coal had in any way influenced the picture. He felt that aluminum dust probably belonged in the same category. He doubted whether freshly fractured surfaces of silica were of primary importance in accounting for an insoluble atom. If organisms were involved, it could hardly persist in such state for long. Experience had demonstrated that silica that had been expected to do its work for prolonged periods was still biologically active. He advised other workers, working on the less developed silicosis more rapidly than others elsewhere in the country, to be emphasized that concentrations of rock dust were much higher at the face. He was willing to accept the theoretical possibility of modifying action but he [illegible] cautioned to remember that it was the silica which produces silicosis and that in the various clinical and engineering studies reported by the Public Health Service a correlation between the degree of dust and the development of silicosis had been established in a considerable number of observations.

The chairman commented that Dr. Sweet's opinion reflected one which was prevalent among most observers who worked in the clinical field. They evaluated end results and could only take into account changes which were sufficiently well marked to be obvious on physical or roentgenologic examination. The experimentalist, on the other hand saw a disease in its diffuse but it was of necessity a type that was forced to develop in months rather than in years. His methods might demonstrate the mechanism by which chronic changes were produced in human lungs [illegible] used for much longer periods of time. He might be able to interpret the principles underlying the evolution of tissue reactions. But he should constantly check his experimental observations against the engineering, clinical and pathological observations made in the field. An apparent lack of correlation might mean a new discovery or it might only indicate the failure of the experimentalist to reproduce conditions with sufficient accuracy for his purposes.

The experimentalist today was concerned that with pure silica he could produce a specific reaction in many species of animals. But when he worked with mixtures of silica and other substances his re-

sults were surprisingly inconsistent with [illegible] pattern of human subjects. Years ago Dr. [illegible] and Dr. Hibbs had reported that coal dust would prevent the development of experimental silicosis and the degree of the reaction was [illegible] which removed the silica [illegible]. Yet coal miners still continued to develop [illegible] powder experiments tend to produce [illegible] of high concentrations of different [illegible] Dr. [illegible] to create disease in man. The experiments [illegible] appearance of the non-specific type [illegible] although many of them had tried to [illegible] under the skin. Then came the discovery of the [illegible] in [illegible] Today the experimentalist had [illegible] interpretation. He was coming to realize that other [illegible] tissue tangle might affect its [illegible] cells. Perhaps they only modified the [illegible] perhaps they retarded the inhalation of silica perhaps they [illegible] or prevented its action on the tissues. It seemed probable that [illegible] substances might have similar effects on the production of other [illegible] diseases. It was quite possible that [illegible] of the [illegible] complete protection and that only the [illegible] silica was responsible for the disease which [illegible] into lungs. It was also possible that [illegible] the [illegible] factor which was given free play in the development of [illegible] of human silicosis but which had been [illegible] mineral world. Continued experimentation would probably [illegible] the significance of all these observations.

The audience had already heard some discussion of the protective action of aluminum, which had been [illegible] by [illegible] Operation. In the clay dusts [illegible] the [illegible] objectives of similar protection by the action of [illegible] fumes of [illegible]. (The subject was actually presented by the [illegible] but in reporting it is more convenient to consider it with the experimental pathology of the disease.)

Individual susceptibility.—Dr. Sweet was questioned as to [illegible] of individual exposed to silica for a sufficient period of time would develop silicosis. He was then asked if he could [illegible] the characteristics of the individuals who [illegible]. He replied that he could not although there were undoubtedly [illegible]

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apparent resistance. He [illegible] was probably an inadequate [illegible] merely failed to detect in his analysis. He [illegible] that those who remained well might have had a more [illegible] respiratory filter than those who contracted silicosis. [illegible] he knew of [illegible] to discover such individuals in [illegible] examinations. He also admitted the possibility that [illegible] such as the negro, who is susceptible to keloid formation, [illegible] have a greater capacity to form scar tissue in response [illegible] but this had never been proved. He concluded by stating [illegible] he believed that some individuals might be unusually [illegible] to inhaled dust, this theory still awaited demonstration and [illegible] no way to prove it at the present time.

The chairman remarked that he formerly believed that all the animals of a group exposed to a given concentration of silica dust would develop silicosis within about the same period of time. More recently he had had an experience which had led him to question this viewpoint. He stated that all of the animal species with which he had worked, including various [illegible], dogs, fowl, amphibia and even fish, would react specifically to silica but he now had evidence that certain breeds, and perhaps certain individuals might fail to develop silicosis when exposed to the inhalation of silica. He mentioned recent experiments in which large groups of blue Belgian chinchilla rabbits had been exposed to high concentrations of both quartz and chalcedony dusts for [illegible] and even 3 years without developing silicotic nodulation. Careful check had demonstrated that the air in their cages contained concentrations of 1 to 3 micron silica particles which were far in excess of the amount necessary to produce reaction. Guinea pigs and white rats exposed at the same time in the same room had developed typical reactions in about a year. In previous experiments rabbits exposed for only 6 months had developed nodular fibrosis. On reviewing the records it was discovered that the animals used formerly were of the strain known as Australian, a smaller brown or white animal with a short neck and thick skin. Study of the condition indicated that the Belgian rabbits did not inhale enough of the available silica to cause fibrosis. That such animals were not [illegible] to the action of silica was demonstrated by a large number of [illegible] experiments. When a sufficient quantity of silica was introduced by vein they developed silicotic fibrosis in their livers, spleen and bone marrow and to a

[illegible] degree in the lungs. It appeared that the [illegible] undiscovered anatomic [illegible] their inhaling effective amounts [illegible].

In the case of white rats [illegible] remained free from accidental [illegible] various kinds of dust for [illegible] developed advanced nodular [illegible] duration. Rats with [illegible] had so much thick [illegible] their lungs. They failed [illegible] 18 months.

If 100 or more guinea pigs [illegible] most of them [illegible] but there were always a certain number, perhaps 8 [illegible] which outlived the other members of the group and [illegible] previously.

Thus it appeared that certain [illegible] more resistant to inhaled [illegible] considerable speculation as to [illegible] beings but only a few of [illegible] systematically. (The rejection of [illegible] gold mines whose roentgenograms showed [illegible] in this direction but [illegible] section.) Lehmann's study [illegible] tract as a filter was [illegible] factor of resistance. The chairman [illegible] done some work in this [illegible].

Prof. Drinker stated that [illegible] study of nasal filtration in normal [illegible] than it had previously [illegible] frequently failed to [illegible] breather and whether [illegible] Lehmann claimed that the [illegible] filters than those of [illegible] adequate exposure.

Prof. Drinker stated that he had [illegible] possible to test nasal resistance [illegible] study of the Barre granite [illegible] service was reexamining.

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Early Conceptions.—In reply to a question [illegible] Dr. Cummings said he had been engaged in connection with a theory developed [illegible] Dr. Graham Anderson, Dr. Stevenson stated that he believed the early difference was that there was a constant interval. The Chairman drew attention to accelerate the development and change the nature of the [illegible] compounds. He had recommended the Dorchester Laboratory [illegible] on Pulsed had said [illegible] rumours had indicated that [illegible] phthisis [illegible] might have influence silicosis. He was [illegible] however, that the point had not been verified.

Mr. Cummings reported that [illegible] separate miners [illegible] had discovered 90 men with confirmed radiological evidence of phthisis. A thorough analysis of the [illegible] in comparison with the non-phthisics had convinced [illegible] selection tended to influence upon the development of [illegible] considerable [illegible]

Asthma.—Dr. Sayers was asked [illegible] there was any evidence that asthmatics, particularly young persons with chronic infections, were more liable to develop silicosis. He concluded by [illegible] replied that he did not know but there was theoretical ground for believing that they might. He quoted Dr. Wainwright's belief that accommodation tended to accentuate the severity of the "asthma" from which coal miners suffer.

Rock dusting.—Dr. Sayers was asked [illegible] the silicosis hazard was [illegible] in rock dusting coal mines to be carried out further.

He replied that if the recommendations of the U.S. Bureau of Mines were followed there would be no hazard. Only non-siliceous dusts like limestone, gypsum or [illegible] had been recommended. In certain mines, notably in Scotland, shale was employed as the dust material. Since this rock [illegible] contain rather more than 5 per cent. of free silica there was a theoretical hazard. In Wales the miners themselves had bitterly opposed the use on account of the possible hazard. In other mines talc had been employed because of certain desirable physico-chemical properties. Dr. Sayers believed, however, that the non-siliceous material that had been recommended were just as effective and that they should be substituted to avoid even a theoretical hazard.

Sericite.—The chairman asked [illegible] the present status of the sericite question and replied that [illegible] years time after intravenous injection into rabbits no evidence of progressive fibrosis had

been discovered. He had treated three [illegible] chemically and petrographically [illegible] all the recent silicates. He had [illegible] effect of sericite in combination with quartz because [illegible] had no effect it would be one of [illegible] tion.

Dr. Sayers stated that sericite [illegible] separated by colloidal silica and Dr. Stevenson commented [illegible] very difficult to obtain sericite in pure [illegible]

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DUST CONCENTRATIONS AND THEIR MEASUREMENT

Percy Pierce Drinker
Harvard School of Public Health

The physician's concern with dust exposures is usually limited to appraising the hazards from inhalation of dust in terms of some arbitrary standard of tolerance.

The engineer usually works in close association with the physician, especially if there is a known or suspected health hazard. In addition to concern with the effects of dust on the health of those working in the dusty atmosphere, he is concerned with the economic loss which the mineral dust causes or creates and how best to control it.

Clearly, both groups must express by appropriate means. Very often both wish to know the precise composition of the dust, together with its chemical and physical properties. It usually falls to the lot of the engineer to devise means to develop suitable techniques for answering such questions.

Concentrations found in practice. Dust concentrations are recorded in weights of dust per unit volume of air, as grains per cubic foot or milligrams per cubic meter or as numbers, as hundreds per cubic centimeter, or millions per cubic foot.

Substances which can be determined in small amounts, like chemical poisons, as lead or manganese, are usually reported in milligrams per cubic meter. Substances which are difficult to determine chemically are usually recorded by count. If particle size is such that happens to be fairly uniform, it does not make any difference which method of recording is used as it is possible to convert one set of units into the other without complicated mathematics. But if the dust is made up of mixed sizes, as it apt to be the case in laboratory problems, the accuracy of such conversions may be in doubt unless somewhat involved formulæ are used.

For the past twenty five years the Branch have been issuing several reports on air pollution in Greater Boston. Concentrations are usually of the order of 0.25 to 1 milligram per cubic meter. In Boston, with the Owens' counter, we have found outdoor dustiness for almost two years and found concentrations varying from about 300 particles per cubic centimeter on a very clear day to 2500 or more

on a dull, hazy day. By the impinger method, [illegible] daily concentrations are of the order of 50 to 250 particles per cubic centimeter. In general, it can be said that concentrations of 5 milligrams per cubic meter or less indicate air which is acceptable from a dust standpoint, reasonably well with air which is free from any dust pollution of any sort.

In the mining, drilling, or handling of coal, concentrations in excess of 100 milligrams per cubic meter, which is 3 per cubic foot will generally be found unsafe, while 10 milligrams per cubic meter will cause [illegible]. I have found occasional concentrations in modern coal cutting mills of well managed [illegible] coming close to 1 gram per cubic meter – the dust was coal almost entirely free from [illegible]. Concentrations were exceptional. With water sprays and [illegible], such concentrations can be reduced to well below 100 milligrams per cubic foot or its gravimetric equivalent.

In the handling of inflammable dusts, [illegible] or pulverized soft coal, plant or mining practice [illegible] a long momentary concentration which are usually more than 1 gram per cubic meter.

The engineer, therefore, will be called upon to control dusts varying from 1 milligram to 1 [illegible] a range of 1,000,000 fold. All of these ranges are actually found in practice, and the technics for their measurement have been developed.

Instruments and Methods in Use Today. [illegible] will be served by reviewing the [illegible] in use today for dust determination. The [illegible] is probably a matter of personal preference [illegible] that it cannot be acquired [illegible]. Those [illegible] necessary for common laboratory procedures should not be discarded or revised.

The simplest possible method [illegible] of sedimentation. This procedure is of two types. The first consists of a [illegible] upon which dust is allowed to fall for a given period. An [illegible] of this method is that the [illegible] collected may be [illegible]. The second [illegible] a known volume of air [illegible] which fall upon the [illegible] after a [illegible] period is proposed as a standard [illegible] for this and is perhaps the simplest [illegible]

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respected. Apparently, it is not used by the United States Bureau of Mines.

Sample taken from rafters have been used with some success, but no evidence to exactly the same concentrations as samples collected on surface weights or pans. They provide qualitative proof that dust is present but no indication as to concentration and they must be used with caution.

Konimeter Grab. One of the best-known grab samplers is the konimeter of Beattie, which was reported in the U.S. Bureau of Mines Technical Paper No. 30 in 1912. Various modifications of Beattie's konimeter have been used by many investigators in the United States and Europe. In its simplest form the apparatus consists of a glass tube with a rubber bulb at one end. The tube is partially filled with water and the bulb squeezed to expel the air. When the bulb is released it expands and the water rises in the tube, drawing in a sample of the air to be tested. The tube is then stoppered and the insoluble dust deposited on the walls of the tube is counted microscopically. In some of these samples the workers themselves carried the suction pumps. In others, the operated pumps were attached to the side of the pump house supposed to simulate human breathing zone. A cubic foot per minute was selected and that rate per minute for the dust, although there was no valid reason for it.

This report by Higgins, *et al* was rather extensive on dust concentrations produced in mining about coal, with and without precautionary measures. A large number of samples were taken, petrographic analyses were made, dimensions of the dust taken and the petrography studied. All of this information was correlated with examinations of the workmen. Granting our modern improvements in all of the techniques involved one notices the great similarity of the conclusions of Higgins' report to those from our more recent and much more complicated documents.

In Britain they propose using filters with salicylic acid instead of sugar at air flows of 200-300 litres per minute. The salicylic acid is dissolved out in alcohol and the dust weighed or analyzed as desired. This method is to be used for collecting samples for special analyses and is not intended for use in the general estimation of dust concentrations.

We have often taken samples for similar purposes with a household vacuum cleaner discharging into a clean paper filter bag. The paper lets through some dust at the start but rapidly plugs and very soon operates at an efficiency which is higher than that of the usual impinger apparatus. We have run such samples for hours and even days.

Electrostatic Precipitation. Small precipitators operating at low voltages are used in dust problems in the laboratory and field. The present technique of the Bureau of Mines in the application of electrostatic precipitators involves the use of a precipitator tube. The Western Precipitation Company has developed a small precipitator which seems very well adapted to field sampling where the collection of relatively large amounts of dust to weigh is needed.

On several occasions we have used a thermal precipitator. Such samples are much smaller than those obtained by other methods, but we feel that the method is unusually accurate. These precipitators are not adaptable to field use.

Konimeter-Smith Impinger. The konimeter-impinger combination used in mine have been described in the literature previously and will not be repeated here.

It was suggested recently by workers in the field that a precipitator could be used in conjunction with the konimeter. Such samples at a rate of 1 cubic foot per minute. We have been using such micro impingers and have found that they operate at five liters per minute and built according to Figure 2, gives results which are entirely comparable to those obtained by the konimeter. The present cost is about $2.50 each and they operate on a small compressed ejector or from a water operated ejector, as was modified used for suction in the chemical laboratory.

The technique of counting the captured dust particles is the same whether the sample was taken by *konimeter, precipitator* or impinger. The impinger liquid, usually *redistilled water*, is diluted serially and the particles counted in a special counting cell. The optical set-up is substantially that developed years ago for counting the relatively large microorganisms (15-50 microns) found in drinking water. A 10x objective, a 7.5 to 10 x eyepiece, an Ocular length of 160 mm. As some of the first work on counting dust particles was done by the pioneers in water microscopy, it is natural that they were used to those made dust estimates by a technique with which they were not familiar. However, that is no reason why we should reject a technique which is optically equivalent to our present practice.

It is often said that a count made by two analysts or by the same analyst on different days will rarely check. Even though the novice is intelligent and [illegible]

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methods or even with microscopy. I have [illegible] figures obtained by inexperienced counters and [illegible] are out of training. Such results are due to the fact that the optics of the present light-field methods are unsound. One cannot count accurately particles comparable to the ordinary bacteria in size with a set-up which is intended for counting algae. Naturally, one misses a great many of the tiny particles and the less experienced the observer, and the worse his vision, the more particles he is apt to miss.

Echuck of the Metropolitan Life Insurance Company, and Cummings of the Saranac Laboratories showed that the present light-field counting technic failed to disclose small particles which have been shown definitely to be silicosis producing. They suggested instead the application of a simple dark field set-up to which Hatch and Pool have made some useful modifications (7).

Hatch and Pool showed that the ratio between light and dark field counts gave very useful information as to the dispersion and particle size. For instance, in a granite-cutting shed, the dark-field count will be about six times the light field and may be 100 times the light field in a brass foundry where the smoke from zinc-oxide particles is excessive. Again, if one samples from a cloud of flint dust from time to time, as the dust settles out, the ratio of dark-field to light-field counts increases rapidly as the size of the particles in the suspension decreases. That is, the combined dark field, light-field method offers a practical substitute for particle-size measurement which, at present, is a wearisome job no matter how done.

Bausch & Lomb Counter.—Some years ago (2), Owens in London developed a dust counter in which 50 cc. of humidified air is driven at high velocity through an orifice with a trumpet-shaped approach. The orifice was 1 centimeter long and 0.1 millimeter wide. On the downstream side was a cover slip on which the dust particles were deposited. The density of the dust deposit was controlled in some measure by the amount of air driven through the orifice. The particles were then counted under oil immersion at about 1500 x magnification. Owens in England and many others throughout the world have used this instrument with great success for sampling air out of doors. In dusty factories or mines, however, the dust deposit is usually so heavy that it becomes impossible to count the particles with any real accuracy.

In South Africa, the konimeter was developed for sampling

air in the gold mines. It operates on somewhat the same principle as Owens' but is perhaps less accurate.

Within the last year, the Bausch & Lomb Company* have put out a dust counter which is essentially a modification of Owens' original instrument with many practical modifications suggested by the late David S. Beyer and his associates in the Liberty Mutual Insurance Company. The microscope is mounted on the instrument and samples are counted by dark field. Twelve samples can be taken on a glass disc which is rotated beneath the orifice slot through which the dust particles are driven. At present, the instruments are equipped with a 0.1 millimeter orifice, but experiments with a 0.4 millimeter orifice indicate that it has many advantages over the narrower slot.

The Liberty Mutual Insurance Company has investigated the characteristics of the Bausch & Lomb counter and has kindly allowed me to quote from their report, the essentials of which will be published presently in the Journal of Industrial [illegible]

"The variable ratio $\frac{\text{B. \& L. Count}}{\text{Impinger Count}}$ depends primarily on the size range of the dust being sampled. The finer the sizes, the greater the value of the ratio. No correlation fine [illegible] are possible for use in changing Bausch & Lomb counts to [illegible] impinger [illegible] under the varying conditions of concentrations, size range and composition found in industry. The variations in ratio $\frac{\text{B. \& L. Count}}{\text{Impinger Count}}$ using the 0.4 millimeter slot are smaller in all cases than the variations in this ratio using the 0.1 millimeter slot."

Like the Owens' counter, the Bausch & Lomb instrument features tiny particles around 0.6 microns to which the impinger technic (with light field) is relatively insensitive. For example, in a brass foundry with zinc-oxide smoke, the Bausch & Lomb dust [illegible] were too heavy to estimate, while the impinger technic showed counts below five million per cubic foot. Obviously, the impinger counts were greatly in error.

In criticism of this type of instrument, one usually hears the argument that it takes only grab samples instead of 15, 20, and 30 minute samples, which are claimed to be a better average. The most

*Made by Willson Products, Inc., Reading, Pennsylvania, and Macalister Bicknell Company, Cambridge, Massachusetts.

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elementary reasoning shows that the average [illegible] samples every more information than one [illegible] in equivalent period. Furthermore, it need hardly be mentioned that dust sampling has been done for years in South Africa with the Konimeter on a scale which has not even been approached in the United States and the results have been as fruitful as any we in the United States are likely to attain with the impinger or any other instrument.

Thermal Precipitator.—In connection with field studies of silicosis, the British have described a so-called thermal precipitator (Fig. 13). Dusty air is passed through a slot bounded by two cover glasses between which is a platinum wire heated to 300° C. Dust is driven from the hot wire and deposited at high efficiency upon the cover glasses which are then removed and the particle count studied under oil immersion at 1500 x magnification. It has been found possible to mount such slides and thus eliminate the carbon and treat them with acid to remove the acid-insoluble matter.

The apparatus is light, easy to set up, and easy to operate. Changing cover slips after each run is, at present, a fussy business in the field where one's hands are apt to be dirty and where the air of the work place is always more or less dusty.

It is interesting that with this instrument, which is much more accurate than the impinger, and with the theoretically correct optical set up, the results found in practical field problems could have been predicted from those reported by Ehrenet *et al*, in 1917, using their rather crude technic in their survey of the Joplin Mines.

Solubility of Dusts.—It is recommended in dust sampling that some of the dust may be sufficiently soluble to preclude use of water as the collecting fluid. However, it happens to be a suitable medium in which to see quartz particles under the microscope. It is, of course, reprehensible that the impinger technic does not permit the elimination of the combustible and the acid soluble particles, but that drawback can be avoided in practice by a chemical analysis of a fair-sized sample of the dust taken by other means.

Composition and Analysis of Dusts.—In mining work, the local geologist and mining engineer usually have accurate information as to the ore body and the common gangue minerals. Frequently, the geologist can furnish thin sections which can be viewed under the petrographic microscope and the probable nature of the dust from drilling, blasting, and handling such rock can be estimated. Generally, the air floated dust has a [illegible] of the parent rock whose differences will depend upon the [illegible] and friability of the rock ingredients.

In mixed dusts, as from [illegible] one has [illegible] the percentage of soft clay substances and carbonaceous matter exceed quartz in the air floated specimens. In the presence of [illegible] may be just the reverse.

The mineralogical analysis [illegible] below two microns is difficult and seldom important. In any event, one estimates chemically the combined water, the loss on ignition, the acid soluble material, and the residue. The chemical estimation of free silica, as the [illegible] is considered [illegible] inaccurate in the very range in which we are interested and is probably not worth the trouble which the method requires. On the other hand, if one knows the mineralogical composition of the original coarse material and the chemical analysis of the dust, one can usually reconstruct the mineralogical composition. It is generally admitted that the mineralogical [illegible] which we are most concerned is between [illegible].

In this brief paper, I have not [illegible] upon all of the procedures in [illegible] subject is one which is changing very rapidly [illegible] application of control measures alter the whole picture [illegible] month to month. Methods which were [illegible] today. For this reason, I do not [illegible] to represent standard sampling methods should be written into laws or codes as they are certain to prove a handicap [illegible] to future progress. Let the law state merely that the method will be one which has the approval of the [illegible] and let the authority then make changes from time to time [illegible] appropriate.

1. Dunnfeld, J. J. A. DeHaVille, J. C. [illegible] and Associates, U. S. Pub. Health Service, Bull. No. [illegible]
2. Dreessler, W. S. Hoch, T. J. Industrial Dust, McGraw-Hill Book Co., New York, 1925.
3. Watson, H. H. A system for [illegible] and [illegible] chemical, and [illegible] examination [illegible] use of thermal precipitator. Journal of Hygiene, [illegible] 1926. At the September 19th meeting

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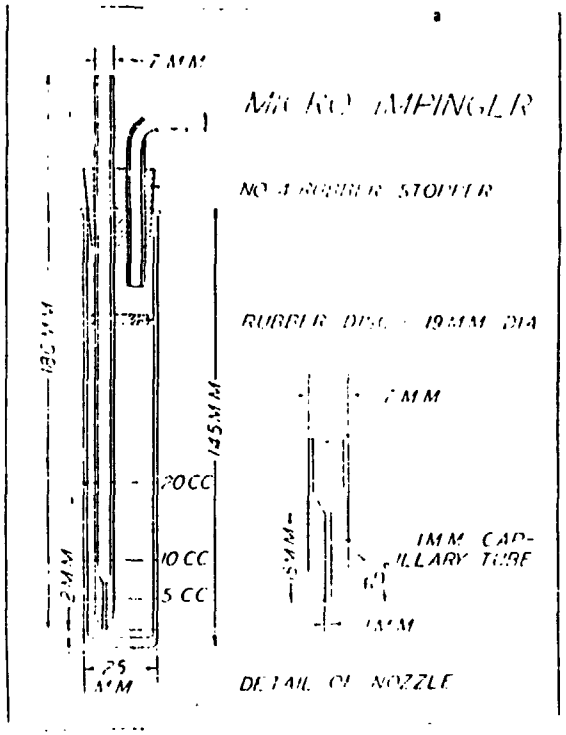

Figure 1. The micro-impinger for sampling at a rate of 3 liters per minute (Courtesy Wilson Products, Inc., Reading, Pennsylvania)

Discussion of Prof. Drinker's Paper

Led By Mr. [illegible]

Prof. Drinker was asked to discuss the effect of [illegible] on the collection rates of the Owen's Konimeter and the Greenburg-Smith Impinger. His question was prompted by the [illegible] separation from the paper would not affect the [illegible] as successive samples were taken.

Prof. Drinker replied that he had used no blotting paper on the tubes when sampling in humid atmospheres. He had [illegible] and found that [illegible] factory tests had been [illegible].

He was asked about the liability of dust particles to [illegible] on the blotting paper lining the sampling tube. It was [illegible] whether this contingency was [illegible] would be caught in comparison to the same [illegible] in the inhaled air.

Mr. Stratton commented upon the liability of particles to [illegible] when they were impinged upon [illegible]. He asked whether the 0.6 micron average [illegible] of the Bausch & Lomb appearatus as contrasted with the 0.5 micron [illegible] the Greenburg-Smith Impinger might be explained on the basis of [illegible] under fluid and in air respectively.

Prof. Drinker replied that the possible error from [illegible] was considered by Greenburg-Drinker-Dautrebande and found not verified the results reported by Greenburg-Smith. He also read the report by Dallen and Goodhue in Science (Oct. 18, 1935) which seemed to indicate a coagulation effect but he did not consider the results significant.

Mr. Stratton called to mind that an apparatus used to [illegible] particles of pigment operated upon the impingement principle. He inquired why the same principle did not [illegible] of particles in these two dust collecting devices. He then [illegible] experimental tests to prove his contention. A dust chamber was closed and dusty air to the point where counts of [illegible] were obtained. Into the chamber was blown a quantity of [illegible] dust particles ranging from 5 to 10 microns in diameter. After a proper interval counts were taken with an impinger apparatus to

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determine the size frequency of the dust in the sample. The median size of the particles did not fall within the 2 to 10 micron range but was about 0.7 microns. He had been forced to conclude that since there had been very few fine particles left in the atmosphere before precipitating the larger ones, the latter must have fractured during the process of collection.

Prof. Drinker was asked whether there was a significant variation in the volume of air drawn into the sampling tube of the B and D apparatus at different plunger speed. He replied that the manufacturers had determined the optimum rate and within reason it did not matter whether the plunger of the pump was pulled gently or with a violent jerk.

He was also asked whether the orifice of the apparatus would ever become plugged and replied that this was the chief fault which he found with it. Fibres of blotting paper and other large particles from the atmosphere would obstruct the slit.

Someone commented that difficulty had been encountered in counting high concentrations of dust owing to imperfect dispersion of the particles impinged upon the counting slide.

Dr. Gardner mentioned that he had just received a letter from the manufacturers stating that they would shortly modify the apparatus by increasing the width of the slit apperture from the existing 0.1 to 0.4 mm. and thereby secure greater dispersion of particles on the slide. (Since the meeting the new type of apparatus has been tested and it has been found possible to count particles up to concentrations of 50 or 60 million as measured by the instrument itself. It should be noted, as Prof. Drinker mentioned in his talk, that the concentrations of 50 to 60 millions as recorded on this apparatus bear no relationship to a similar concentration that might be obtained with the standard impinger method).

Mr. Cummings brought up a question relating to dispersion of particles in the collecting fluid rather than fracture by impingement. He reported that C. E. Brown of the Bureau of Mines, after many tests, had convinced himself that alcohol was a better collecting fluid than water for use in the impinger apparatus. He made counts in the Michigan iron mines upon samples collected in alcohol. They were, as anticipated, much higher than those from duplicate samples collected in water. Brown therefore concluded that he had demonstrated the greater efficiency of alcohol. But Cummings disagreed since he believed that iron ore dust existed as aggregates and that

[illegible] of the particles of dust in the atmosphere he was more likely to succeed in [illegible] of alcohol. This result, he interpreted [illegible] fracture but he thought it was due to [illegible] of surface tension.

Mr. Cummings then asked whether an engineer [illegible] and when he measured its concentration, and thereby [illegible] Dr. Drinker replied that [illegible] necessary.

Prof. Drinker recalled that [illegible] engineers to [illegible] stations. The tool [illegible] concentrations which could [illegible] after precipitation [illegible] He agreed that the engineer [illegible] did not measure the hazard [illegible]

Mr. Dean thought he had evidence [illegible] the variation in dust counts [illegible] about apparatus in a glass plant. He reported that [illegible] moderate velocity of [illegible] was free from [illegible] dust concentrations were extremely low, but that in other parts of the plant, where no dust was being created, the dust carried [illegible] convection currents produced high concentrations. In this case the engineer was able to explain or discover a hazard. He then felt it should be emphasized that an adequate dust survey should [illegible] samples from all parts of the plant regardless of their [illegible] dustiness.

Dr. Gardner remarked that the obvious measure of a hazard was what happened to the employee exposed to the conditions which the engineer defined. Dust counts might be high or they might be low but they were only hazardous if they caused deleterious reactions in the persons exposed to them.

Prof. Drinker was asked to comment upon the [illegible] appearance of particles under one micron in diameter. He said that he did not know, probably because there was no known and standardized exposure to silica where the particles were uniformly of [illegible] dimension. He thought the answer would involve prolonged [illegible] observation. He had recently experimented with [illegible]

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crons and under, produced by burning [illegible] had obtained negative results from injections.

Dr. Gardner then recalled Saranac Laboratory experiments (mentioned in the session on pathology) in which it had been shown that the rate of tissue reaction was inversely proportional to size. Reports of the effects of particles 10 to 12 microns and 1 to 3 microns had already been published. Unreported experiments showed that injection of quartz particles one micron and under (minimum size not determined) into the veins of rabbits caused an [illegible] generation of the liver and death within 3 or 4 months. Still more recently a large sample of vitreous silica with particle size of 20 Angstrom units (0.002 microns) had been obtained. Injection of one gram of this material into the [illegible] of rabbits had caused no immediate effects nor had there been any progressive fibrosis in animals killed after an interval of 6 months. He was of course greatly surprised at the lack of toxic symptoms. The absence of fibrosis might be accounted for by the lack of retention in sufficient concentration. The particles were so extremely small that they would tend to pass through the capillaries of the various organs and as a matter of fact examination of the urine had shown a large excess of soluble silica. Inhalation experiments had been tried with this dust but they had been a failure due to the development of endemic pneumonia in the exposed animals. No conclusions were being drawn at the present time but the experiments were being continued.

Prof. Drinker was asked his opinion of the accuracy of x-ray diffraction in determining the composition of atmospheric dust. He said that a geological confrere had advised him to stick to the standard petrographic and chemical methods.

Dr. Gardner said that in the Saranac Laboratory they had been attempting to correlate the reaction produced in the lungs of animals and men by mixtures of silica with other substances. As an introduction to the pathological findings which he intended to report on the following day he asked Mr. T. M. Durkan of the Laboratory staff to report some interesting observations on the behavior of silica in association with other substances in the atmosphere.

Mr. Durkan presented data on four examples of exposure to mixed dusts studied by the Saranac Laboratory. The first sample dealt with a natural and the second an artificial mixture of mineral dusts used in the experimental dusting rooms of the Saranac Lab-

oratory. The third and fourth examples referred [illegible] exposure in industrial plants which had been correlated with clinical findings. He stated that ferruginous chert, a natural mixture of hematite and a form of free silica known as chert, was being used in an animal dusting experiment begun four years ago. Failure of the animals to develop fibrosis after months of exposure, even though the dust concentration was in the hundreds of millions and the percentage of silica in rafter samples was high, led to the [illegible] that the dust suspended in the air contained only one fifth as much silica as did the original material (see table). Ferruginous chert high in silica was then reground in a colloid mill to the finest size [illegible] commercially and used in the dusting machine; [illegible] suspended dust contained much less silica than did the parent material, although the difference was not so great. Similar data were also presented for artificial mixtures of quartz and calcined gypsum employed in another animal dusting experiment [illegible] that while the percentage of silica might [illegible] for rafter samples than for the parent material, [illegible] the cases cited was much the lowest for air-suspended dust.

COMPOSITION OF AIR-SUSPENDED SHELTERS AND [illegible] DUST IN EXPERIMENTAL [illegible]

	Natural mixture of dust (ferruginous chert)	
	Ground finely fine	Ground finely fine [illegible]
Parent material (in dusting machine)	[illegible] SiO2	[illegible]
Rafter dust (settled on top of cages)	[illegible]	[illegible]
Air-suspended dust (collected with impinger)	[illegible]	[illegible]
	Artificial mixture of dust (quartz and gypsum)	
	1 part quartz, 1 part gypsum	1 part [illegible]
Parent material	[illegible] SiO2	[illegible]
Rafter dust	[illegible]	[illegible]
[illegible]		[illegible]

Reference was next made to the mixing and [illegible] plaster, an operation that offered a potential hazard. The material was a mixture of one part gypsum and two parts sand, the sand being composed of quartz and calcite particles. Dust concentration near

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the mixing and packing machines were high, [illegible] contained a moderate amount of quartz, yet none of the men working under these supposedly unfavorable conditions had silicosis even after about 30 years exposure. The absence of silicosis was explained by the small amount of silica in the air-suspended dust, as shown in the table.

COMPOSITION OF AIR SUSPENDED, SETTLED AND PARENT DUST NEAR MACHINES TO CLOSE CARTON PLANTS

	Plant A	Plant B
Parent material	[illegible] quartz	40% quartz
Settled dust (settled on floors)	[illegible] quartz	
Air-suspended dust	4% quartz	17%
(Settled dust less)		

To analyse the condition further, the air-suspended dust collected at plant A was treated with acid and the insoluble portion (quartz) separated into fractions by water sedimentation. It was found that nearly all the quartz particles were larger than 50 microns in size, and that the fraction smaller than 10 microns was only [illegible] of the total sample. Two factors were interested to account for this condition: (1) washed sand containing only large particles was used and (2) gypsum, owing to its hygroscopic nature, gathered the quartz into clumps which settled out of the atmosphere rapidly.

The last example of mixed dust exposure was taken from a mill where limestone and clay accompanied by some quartz were mixed and ground. Dust counts were in the hundreds of millions.

COMPOSITION OF AIR SUSPENDED, SETTLED AND PARENT DUST IN MILL FOR PRODUCING LIMESTONE AND CLAY

Parent material	[illegible] quartz		
Settled dust (settled at breathing level)	12		
Air-suspended dust (collected at breathing level)	6	[illegible] 10 microns	9%
		[illegible] 10 microns	1%

There was an appreciable quantity of quartz in the atmosphere, as shown in the table, but not a single man in the mill had silicosis and some had worked there more than 30 years. This seemed to indicate that there was an inhibitory or protective action, probably by the clay, which prevented the quartz from exerting its harmful effects. However, an explanation also offered was that the absence of silicosis may have been due merely to an insufficient number of

quartz particles in the air. Although [illegible] particles smaller than 10 microns made up 3 per cent of the weight of air-suspended dust, the number of such particles might be a thousand times greater than 1 per cent of the dust concentration. Lack of satisfactory evidence for inert, dust-forming particles of the order of one micron or less made it difficult or impossible to tell how much more quartz was there than clay.

From the data presented several conclusions were drawn: (1) air-suspended dust may be considerably different in composition from the original or parent material, or from that which has settled out of the air; (2) minerals accompanying quartz in pulverized form may modify or inhibit the usual potency of the latter to cause silicosis; and (3) the [illegible] hazard associated with mixed dusts, as found in the cases cited, is much less than would be indicated by the percentage of quartz in the parent material.

PLAINTIFF'S EXHIBIT [illegible]

PATHOLOGY OF THE PNEUMOCONIOSES

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In order to understand the pathology of silicosis it is necessary to appreciate the fact that the body attempts to dispose of inhaled silica dust in the same manner as it does all other inorganic particulate material. But silica particles, being peculiarly toxic, injure the cells of the various mechanisms involved so that the protective process fail and more serious results ensue. It is essential to recognize the difference between the reaction to silica and that to other types of dust so that the two effects may be differentiated in roentgenograms.

METHODS OF INVESTIGATION

Various methods are open to the investigator of this disease. He gains much information from serial roentgenograms of the living subject if he can follow them for a period of years. The causation of the disease is defined by studies of the dust and its concentrations in the industrial environment and by surveys of the employees. Autopsy material as it becomes available is a valuable check upon the accuracy of the roentgenographic interpretation and for that reason it means most to the pathologist who is familiar with all phases of the particular dust hazard concerned. The tissues are examined by the usual gross and microscopic methods but in addition probably one of the most valuable adjuncts are large, thick sections from known locations in the lungs. Such sections, four inches or more in diameter, mounted between Lucite slides, give a better idea of the topography of the changes responsible for the shadows seen in a roentgenogram than the small thin sections with which the pathologist ordinarily works. Valuable supplementary information may be obtained by incinerating thin unstained sections to reveal the mineral particles present in the lesions. The identification of such particles requires familiarity with petrographic techniques. Valuable information may be gained from chemical analysis of dried lung tissue. Such analyses must be made on generous samples taken from representative parts of the lungs. It is well to analyse the tracheobronchial lymph nodes separately since they

accumulate dust particles from all parts of the lung. One must know the amount of inorganic ash for a given weight of dry lung tissue and the percentage of various elements including silicon in the ash. In the absence of typical tissue changes or other evidence of fibrosis, the presence of an increased amount of silica in the ash in silicosis are more than the discovery of a tubercle bacillus in a surgical lung tissue would mean tuberculosis. In the hands of the experienced observer, the gross and microscopic examination of the tissues is the most reliable index of the presence of silicosis.

To study the evolution of disease due to dust and to determine the conditions under which it develops experimental models are both valuable and reliable. The natural model [illegible] of dust [illegible] is, at the same time, expensive and exceptional. It constitutes the final check on the hazard [illegible] and [illegible] or mixture of dusts. It is the only way in which the effects of synergistic phenomena in cases of mixed dusts can be [illegible].

Injection methods are useful both because [illegible] and less expensive and because they can be used to [illegible] investigation of the dusts which are biologically most [illegible] exception of a heavy, which is unusual in its action and [illegible] particulate dust has been shown to produce [illegible] action when inhaled into the lungs from the [illegible] into the tissues.

In the Saranac Laboratory the method of repeated intravenous injection of small doses of standard sized dust particles into the ear vein of rabbits has proved most satisfactory. Ordinarily 1 c.c. of a suspension of dust particles, 1 to 3 microns in diameter, suspended in physiologic salt solution (1% by weight) are injected on 20 consecutive days, each dose containing a period of about 2 months. Most of the particles of this size are retained in the liver, spleen and bone marrow, only a few are caught in the capillaries of the lung. Silica dust produces typical progressive nodular fibrosis wherever the particles [illegible]. Other dusts are phagocytosed and remain in the tissues without producing more than a very slight chronic inflammation which has shown no change over a period of 2 years after completing the injections. This method of injection does not traumatize the tissue where the dust is going to localize; it does not produce non-specific reactions from pressure of a large volume of particles and [illegible] introduced directly between the cells of an organ. Because of the divided dose administered the dust particles are gradually collected

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by the action of phagocytes in the organ [illegible] is to be studied. The results are much more [illegible] than those produced by other methods.

The intraperitoneal method, by which 0.2cc of a 10% suspension of dust particles is injected into guinea pigs in one dose, has the advantage of enabling one to estimate the potentialities of a dust to produce reaction in the living body with considerable rapidity. Sayers and Miller, who devised the test, have reported results which conform to clinical experience and have been confirmed in the Saranac Laboratory.

A variety of other injection methods have been employed but they are all open to the criticism that the non-specific effects of pressure make interpretation difficult.

The results of many years experience with various methods of experimentation in the Saranac Laboratory may be catalogued as follows:

1. All species of mammals, birds, amphibians and fish that have thus far been tested react to silica in a specific manner.
2. White rats, if they are resistant to pulmonary infection, develop the most typical fibrous nodulation in the shortest period of time.
3. Guinea pig silicotic nodules do not resemble those in man so closely as the rat lesion; but these animals are more useful for the study of associated tuberculosis than the rat.
4. Only certain strains of rabbits seem to develop silicosis by inhalation during a period of 2 to 3 years. The refractory strains are susceptible to silica injected into the veins.
5. Monkeys, cats and fowl develop silicotic reaction on inhaling silica dust.
6. White mice react to silica injected into the subcutaneous and other tissues; inhalation experiments have repeatedly failed probably because of the susceptibility of the strains employed to chronic pulmonary infection.
7. Tadpoles and gold fish have reacted specifically to injected silica.
8. Most of these species have been similarly treated with other

dusts, including a variety of silicates and have [illegible] failed to develop progressive fibrosis.

9. Inhalation of asbestos dust by guinea pigs has [illegible] the development of a peculiar type of pulmonary fibrosis. Rabbits exposed at the same time have not been [illegible]. Injection of asbestos dust has thus far failed to [illegible] in organs other than the lung.

(These results were illustrated by a series of 15 [illegible])

THE GENERAL TISSUE REACTION TO INJECTED DUST SUSPENSIONS

By whatever method dust particles are introduced into the body their primary treatment in all of the organs [illegible] Their ultimate disposition and [illegible] their chemical composition, size, shape and [illegible] response to particulate matter [illegible] 3 microns in diameter consists of:

1. Phagocytosis by dust cells in these [illegible] where the particles come to rest.
2. The enlargement of such cells as more and more particles are ingested and in many instances [illegible] the nuclei to form giant phagocytes. Frequently the large cells contain so many dust particles that their structure is completely obscured.
3. The paralysis of the overloaded cells so that they [illegible] their native capacity for free and independent migration.
4. Cells which take up fewer particles tend to migrate away from the functional cells of an organ and accumulate in the connective tissues or they enter the lymphatics and are carried to a lymph node which receives the drainage from the organ.
5. A very slight amount of chronic inflammation may develop about clumps of dust-filled phagocytes but unless the dust is pure silica such reaction is not progressive [illegible] evidence of progression has not been manifested with [illegible] period

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of 2 years. (These responses were illustrated by lantern slides. Iron, aluminium oxide and the silicate garnet were selected for the purpose but any other non-siliceous dust could have been used.)

THE SPECIFIC REACTIONS TO INJECTIONS OF SILICA PARTICLES

The primary response to silica particles is the same as that to dust of other type, namely phagocytosis. But the cell which myocytes too many silica particles is killed by the toxic material. It disintegrates and frees them again. More phagocytes are formed and enter the area. They in turn pick up the liberated particles and in time there are enough cells so that each contains only a sublethal dose of irritating silica. They are activated only enough to stimulate reparation. They carry the silica to the nearest lymph nodule where the accumulated irritant causes the connective tissues to multiply. If the local dosage is too great it can overwhelm all of the newly formed tissue. The result is a progressively enlarging nodule of connective tissue, perhaps with a degenerate hyaline core of silica particles.

Sometimes the dead tissue at the centre attracts lime salts from the blood and undergoes calcification but more often it is absorbed and replaced by fibrous tissue. Fibrosis is the characteristic feature of the reaction to silica but it is fibrosis of an unusual type. The collagen or gelatinlike material of which it is largely composed, is laid down in excessive quantities by living cells but they are soon compressed to so small a compass that they are barely recognizable. The collagen fibres become very thick and heavy and assume a glasslike or hyaline appearance; they react in an unusual manner with the dyes used to stain microscope sections. As the number of collagen fibres in the limited area of reaction increases, they are forced, apparently by pressure, into rings of concentric lamellae. The mature silicotic nodule consists of a spherical onionlike mass of pale, thick collagen fibres having a whorled arrangement. The borders of the nodule are sharply defined and separated from the surrounding tissue. The mass comes in view from a structure only visible under a microscope to one 2 to 3 mm. in diameter. In its centre is a variable number of silica particles which are visible in polarized light. To see them most readily the tissue should be destroyed by burning the section in a muffle furnace (Lantern slides).

The liver cell is replete with specialized areas to the presence of which silica produces its peculiar effects in the tissues. Probably the most generally accepted hypothesis is that which assumes that solution takes place in the alkaline fluid of the body. The enzyme action (which in turn poisons the cell). But direct proof of this action in the tissues is wanting and not all of the observed facts are in accord with this hypothesis. The specific cell poisons are independent in the matter and will concern us all here and also the other [illegible]

OBSERVATIONS CONSISTENT WITH THE SOLUBILITY HYPOTHESIS

1. Gye and Purdy's demonstration that [illegible] tissue, poison and Gye and Purdy's [illegible] sions produced by colloidal silica heal by fibrosis.

2. Lettle demonstrated that the silica particles in solid nodules lose their characteristics. The tissue responds because they become coated with [illegible]

3. Cummings, Redfern and Gardner demonstrated [illegible] of fraction to silica particles is inversely proportional to their size. This might also be applied in other ways.

4. Fung observed that the urine of rheumatic patients [illegible] soluble silica than that of normal persons. (Evidence in Dr. Sayers' paper on etiology for refutation of this statement. Dorsett shows that the urinary silica is [illegible] of alimentary origin.)

5. Frank demonstrated the presence of silica in the [illegible] its concentration is essentially the same in the tissues of normal subjects.

6. Emmons and Wilcox approximately demonstrated an appreciable amount of solution of free silica in [illegible] discussion on etiology where their method and [illegible] criticized). Their observations are at one with those of Titus who employed a more appropriate technique.

PLAINTIFF'S EXHIBIT [illegible]

OBSERVATIONS AT VARIANCE WITH THE SOLUBILITY HYPOTHESIS

1. Experiments recently completed in the Saranac Laboratory demonstrate that all forms of free silica except precipitated amorphous silica will produce progressive fibrosis. The severity of the reaction is not proportional to the degree of solubility, however.
   The biologic activity of the crystalline silicas is of the following order: tridymite, fused quartz, crystobalite, and vitreous silica.
   Cryptocrystalline silicas as chalcedony, chert and tripoli are all very active.
   Amorphous silica as *opal* is only moderately active but will produce some nonnodular progressive fibrosis in a year's time.
   Amorphous silica as diatomite, on the other hand, is extremely active and produces as much nodulation as quartz.
   Colloidal silica in dispersed state has an acute poison which causes almost instantaneous death upon intravenous injection.
   Precipitated colloidal silica has failed to cause reaction. (These effects were illustrated by lantern slides.)

2. Representative silicates[1] in various states of hydration have without exception failed to produce any fibrosis within a period of 1 or 2 years. Since many of these silicates are quite soluble they should have dissolved and caused reaction, if solution were the only explanation. (Lantern Slides).

3. Studies in the Banting Institute failed to demonstrate the development of fibrosis when soluble silica was permitted to diffuse through the wall of collodion capsules implanted in the peritoneal cavity of guinea pigs.

4. The tissues react too promptly after injecting particulate

[1] In the category are included the many silicate minerals known collectively as asbestos. Chrysotile, for an explained reason, has been very difficult to study even when ground to fairly fine particles. One ram in diameter. Such cells cultures injected intravenously either immediately or within 1 or 2 days and there are no apparent lesions. Serpentine, a pure chrysotile mineral of the same chemical composition as chrysotile has no such effect. It is assumed that a residue of the free fibers of the chrysotile in serpentine may still possess fully the wounds from mechanical effects upon the circulation.

silica for much solution to have occurred. Acute pulmonary edema becomes manifest within 15 to 30 minutes. (Lantern slides showing intracutaneous reactions to quartz and to carborundum.)

The absence of tissue reaction or protective phenomena at points remote from deposits of silica or the presence of fibrosis that does not appear to be in direct relationship with the cells in the immediate vicinity. There is no evidence to support the statement of Gye and Purdy that all of the tissues in the body of a chronic rabbit are poisoned by circulating toxic silica.

It seems clear to the speaker that an adequate explanation of the action of silica upon the living cell is still lacking. Neither hypothesis cannot comprehend all of the observed facts. Only the "physical injury" obviously does not explain why the smooth "blunt, sharp" particles of diamond, alundum, and of the carbide cause no fibrosis (Slides). It may be that we are dealing with a combination of mechanical and chemical factors each producing an effect not attained by either one alone. In the case of quartz it is possible that amorphous silica as opal is not quite the same substance in the tests of short duration as are the other forms of silica investigated. Play in the connection, a result of the solubility of each one of the silicas to be emphasized. The suggestion of this observation primarily, Mr. E. W. Thompson has suggested that diatomite might be unusually active because of the very large surface exposed. The diatomes are pierced with an extensive number of very fine holes, each one of which is lined by a layer of amorphous silica.) Perhaps most promising of all the present hypotheses are those unknown and ill-defined properties of the surface of the particles. Mr. Cummings, in his discussion of the physical chemistry of the absorption phenomena which Langmuir demonstrated Hoffman has speculated about unsatisfied free valences on the surface of freshly fractured silica. Obviously it is still necessary for someone to make the crucial experiment which will reveal the mechanisms by which silica exerts its influence on the tissues.

PULMONARY PNEUMOCONIOSES

Pulmonary Anatomy and Physiology. A series of lantern slides was shown to illustrate the normal anatomic features of the lung

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and their functions were [illegible] perhaps not more than 50% of the dust in the [illegible] anything zone enters the lung because of protective mechanisms. These consist of the coarse hairs, which guard the nostrils; the sticky mucus secreted by the cells lining the nose, throat, trachea and bronchi; the cilia, microscopic hairs covering the surface of the cells in these locations, which by waving create currents that sweep foreign bodies toward the throat; the cough reflex, which expels mucus with entrapped dust; mobile phagocytes or dust cells, which ingest and transport out of the air spaces the dust particles that pass the upper barriers; and finally a lymphatic system, one of whose major functions is to remove foreign bodies from the lungs to reservoir-like lymph nodes.

General Response to Inhaled Foreign Particles. The protective mechanisms of the upper respiratory tract effectively exclude a large part of the fine dust in the atmosphere. Perhaps some kinds are handled better than others but this subject has not received much attention. Particles that pass these barriers enter the lung with the inspired air and drift toward the subpleural air spaces with surprising rapidity. Here they attract free-moving phagocytes which ingest and carry them into the connective tissue framework of the lung where the lymphatic system is located. When excessive quantities of dust must be dealt with the same phagocyte often continues to pick up particles until it is overloaded and apparently paralysed so that it does not move very far. The cell may remain inside the air space or it may enter the connective tissue and become immobilized. With lesser amounts of ingested material the dust cells enter the lymphatic vessels a few at a time and are gradually carried by the lymph to lymphoid nodules located either inside the lung or in larger masses at its root where the stem bronchi come off from the trachea. The latter are known as the tracheobronchial lymph nodes. If the inorganic dust is inert and [illegible] as known today, of anything but free silica or fibres of asbestos, its only effect is a variable amount of pigmentation and a slight chronic inflammation about the deposits. With large accumulations such as occur in the lungs of soft coal miners the pigmentation is sufficiently marked to be visible to the naked eye. Linear deposits occur along the lymphatics in the pleura and walls of the blood vessels, and focal collections are found in lymph nodules throughout the lungs. Such deposits are so soft that they cannot be felt and they have no effect

[illegible] the normal function of the [illegible] which [illegible] [illegible] the blood [illegible]

Response to Inhaled Pure Silica. The preliminary [illegible] the same as that to other particulate foreign bodies but the [illegible] and injures the protective mechanisms in the [illegible] in other parts of the [illegible] The [illegible] accumulations of [illegible] The stimulated dust [illegible] of irritating silica in the [illegible] lymph nodules. The irritant [illegible] multiply and a nodule is formed. The expanding nodule [illegible] lymphatic vessels and [illegible] the free [illegible] of lymph and [illegible] increasingly difficult. As more dust [illegible] dust cells do not carry it into the lymphatics [illegible] now tend to move into the [illegible] Multiplication of the local [illegible] formation in this location. [illegible] and destroy a certain number of the capillary [illegible] these delicate membranes. The interchange of oxygen [illegible] dioxide is no longer possible [illegible] particular location. However, the margin of safety is a wide [illegible] with only one-fourth of his normal lung capacity. [illegible] hundreds of fibrous nodules scattered through his lungs [illegible] intervening air-space walls appear quite normal and [illegible] continue to function so that he seems to breathe normally [illegible] conscious of the condition in his lungs. He has [illegible] reserve capacity and only manifests shortness of breath [illegible] den violent exercise creates a sudden demand for large [illegible] oxygen.

Progressiveness of Silicosis. [illegible] shown that an experimental animal or a human [illegible] his dusty exposure with no [illegible] after an interval of some years subsequent [illegible] marked nodulation. This simply means that he [illegible] silica in his lungs when the exposure ceased to cause the formation of the fibrous nodules. If his lungs had not contained enough [illegible] such nodules could never have formed and developed to a [illegible] ontrable in a roentgenogram or to the naked eye. [illegible] continues and the nodules increase in size until the [illegible]

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room diameter of 3 or 4 mm. Then an equal [illegible] recently established and no further progression occurs [illegible] do not develop and the lungs never become completely replaced by uninterrupted development of the nodules already present.

Atypical Forms of Silicosis.—It is produced by a variety of causes. If a portion of the lung has been exposed by infection [illegible] occurred previously to or during the course of [illegible] exposure occurs in an abnormal manner and the lung tissue does not function effectively to remove dust from the part in question. The result will probably be one of the [illegible] of massive fibrosis to be discussed later.

If very excessive quantities of pure silica are inhaled the protective mechanisms are inadequate to deal with the particles penetrating into the lung and silica begins to accumulate in the walls of the air spaces almost from the start. There is [illegible] a diffuse fibrosis involving all of the connective tissues of the lungs. Nodules form but they are often of microscopic dimensions and are obscured in the generalized process. Such reactions are occasionally reported after as little as 18 months of intensive exposure. This so-called "acute" or "rapid" silicosis is a rare condition and is only known to develop where the silica is almost pure, the particles extremely fine and the ventilation so bad that the dust concentration becomes almost unbelievable.

Other forms of silicosis may occur which have not yet been identified. Animal injection experiments would indicate that conversion forms of quartz, known as tridymite and cristobalite, produce a progressive fibrosis which is diffuse rather than nodular in type. No inhalation experiments have been attempted yet nor are there any clinical or pathological data on human beings exposed to these dusts. Inhalation might still result in a nodular form of disease.

Response to Mixtures of Silica and Other Dusts.—This is an extremely important subject, for the majority of industrial dusts are of mixed type and only in the occasional industry do men inhale pure silica. The character of the reaction in the lungs depends upon the relative amounts of silica and the adulterant substances in the atmosphere. Small amounts of silica (perhaps 5% or less) have little influence upon the picture; the pulmonary changes are the same as if there were no silica in the dust. One sees linear deposits of dust in and about the lymphatic vessels with focal collections in the lym-

phoid nodules. The dust provokes a fibrosis characteristic [illegible] to silicosis.

If more silica is added to the mixture the [illegible] become more pronounced and as they [illegible] a limited amount of fibrosis develops. It is generally linear in its distribution [illegible] nodules may be discovered in the lymphoid tissues [illegible] may be a few nodules large enough to detect with the naked eye on the cut surface. (They form first in the [illegible] because there are [illegible] ducts in the subpleural [illegible] and because there are [illegible] deposits where the deep and superficial set of lymphatics [illegible] the pleura).

[illegible] the proportion of silica in the dust may be [illegible] such a mixture is exposed to the naked eye [illegible] develops [illegible] after prolonged exposures of [illegible] years. With [illegible] forms of adulterant dusts they [illegible] from [illegible] on this point is still lacking. [illegible] different appearance [illegible] Instead of a sharply defined [illegible] fibrous tissue, the mixed dust [illegible] a variety of appearances. It may consist of a localized [illegible] in a mass of loose connective tissue [illegible] dust. Its borders are ill defined and [illegible] fingers of pigmented connective tissue radiate into the walls of adjacent [illegible] There are considerable accumulations of pigment [illegible] around the lymphatic vessels.

If the inhaled dust contains [illegible] [illegible] to be gathered at the center of the area of fibrosis. [illegible] the nodules the characteristic [illegible] of [illegible] tissue which surrounds the periphery is the relatively [illegible] and very irregular connective tissue which is [illegible] by the non-siliceous dust.

The picture produced by a mixture of silica and other [illegible] is characterized by its variable nature. Dependence [illegible] of silica which enters the lung, one may find [illegible] from [illegible] pure linear or peribronchial type of reaction [illegible] altered by non-siliceous dusts to nodular lesions with or without [illegible] typical zone of pure typical silicosis. Such [illegible] encountered in the anthracosilicosis [illegible] or the [illegible] silicosis of hematite miners.

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Protective Action of Other Substances. As noted [illegible] of Dr. Sayers' paper on etiology, a [illegible] may do more than merely modify the form of the anatomic reaction to silica. Some of the non-siliceous dusts may have only such an effect, other [illegible] retard or [illegible] shape prevent the interaction between the siliceous component and the tissues, and still others may exert their effect upon the silica in the atmosphere so that agglutination occurs and neither the silica nor the adulterant substances are inhaled in proportion to their concentration in the atmosphere.

There are a number of observations in the clinical, engineering and experimental fields which point to the validity of such a belief but they have not yet been sufficiently studied to comprehend their significance.

For example, it is well recognized that silicosis only develops in granite cutters after very long exposures. The Public Health Service study at Barre, Vt., indicated that there was an undue proportion of the cases with linear rather than nodular shadows in the roentgenogram. Autopsies showed nodulation but most of the cases studied by this method were complicated by tuberculosis. Only a few lungs of men dying of other causes, after prolonged exposures to granite, could be examined. These generally showed only the non-siliceous linear type of reaction. In one case with a localized quiescent tuberculous infection there were silicotic nodules in the vicinity of the tuberculosis and in the rest of the lung a linear change. This led the speaker to adopt the old hypothesis that with granite cutters, at least, infection was essential to the development of nodulation. Subsequently one granite cutter has been autopsied whose lungs showed advanced discrete nodulation without detectable evidence of infection. Grossly, there are areas in the lungs and lymph nodes which suggest tuberculosis but this impression could not be confirmed by microscopic examination.

The experimental evidence also indicates that there is something peculiar about granite silicosis. Guinea pigs and rabbits exposed for apparently adequate periods to inhalation of fine granite dust failed to develop nodular fibrosis. If the animals were infected with tubercle bacilli the lesions tended to approximate the silicotic type but they were far from typical.

More recently, granite dust was tested by the intravenous rabbit method already mentioned. The usual procedure failed to produce anything more than an inert silicate type of reaction. The [illegible] of 1 gm. of 1 to 3 micron particles had been injected. Chemical and petrographic analysis had demonstrated that the material contained 0.33 gm. of quartz with 0.66 gm. of feldspar and mica. Enough quartz was present for a little as 0.2[illegible] gm. of quartz alone will produce silicotic cirrhosis of the liver and spleen. To make certain that enough quartz was present, a second series of rabbits was injected, this time with a total dose of 3.3 gm. of granite containing 1 gm. of quartz particles 1 to 3 microns in diameter. As [illegible] there was no evidence of silicosis in the organs after one year had elapsed. Fortunately two animals survived to be killed after 2 years. In these there was a suggestion that progressive reaction might be beginning. The picture was similar to that observed in rabbits killed immediately after completing the series of 29 injections with normal quartz alone.

It would now seem that the silicates in the [illegible] granite held the quartz in check so that it reacted [illegible] the tissues. To determine whether infection would [illegible] oretical intuition, another series of rabbits infected with tubercle bacilli in various ways, was injected with the 1 gram [illegible] of granite dust. They have been followed for only [illegible] months but [illegible] is not [illegible] to suggest that the infection has [illegible] the quartz to [illegible]. The remaining animals are to be killed during the next two [illegible] to check the hypothesis.

Field studies of the composition of industrial dusts frequently show that a theoretical dust hazard does not necessarily produce silicosis in the workmen exposed to it. Thus [illegible] from [illegible], the rafter samples frequently [illegible] the dust collected from the atmosphere. The quartz particles [illegible] but were not fractured as finely as the other components and [illegible] comparatively heavy they settled out quite quickly. The [illegible] and liver non-siliceous particles remained longer in suspension. Mr. Dutlan mentioned the same finding in reporting his studies in [illegible] and gypsum plants.

In the gypsum industry a limited number of men are employed in the manufacture of sanded plaster. They are exposed to high concentrations of dust composed of a mixture of calcined gypsum and sand. [illegible] analysis of the crude atmospheric dust revealed [illegible] considered as dangerous concentrations of silica dust but [illegible]

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logic examination of 17 men employed for [illegible] years in such exposures failed to disclose a [illegible]. On investigating the dust in these departments it was found that washed sand [illegible] ing used and that most of the silica particles were too large to be inhaled. The fine quartz in the atmosphere was apparently agglutinated into clumps by the gypsum and fell out of the atmosphere quite rapidly.

In the same industry there is a certain amount of bulb stone grinding and the stones, when the first inspection was made, were being trimmed in the same room with the grinding. In spite of the theoretical hazard, no silicosis had developed in the operatives. Investigation of the atmospheric dust demonstrated that it existed as large aggregates which could be seen with the naked eye as they settled on microscopic slides. It was postulated that the opposite electric charges of the silica and the gypsum particles were responsible for the agglutination which protected the men from inhaling effective quantities of silica.

An extended series of animal inhalation experiments has been performed in which guinea pigs and white rats were exposed to artificial mixtures of calcined gypsum and pure quartz particles, all less than 3 microns in diameter. In the first experiment, equal volumes of the two substances were placed in the dust hopper. Concentrations averaging approximately 250,000,000 particles per cubic foot were maintained for 8 hours a day, 6 days a week. Analysis of impinger samples of air borne dust inside the animals' cages showed only 31.0% silica, although the original dust contained [illegible] of silica. Obviously there had been an appreciable reduction in the *amount of silica from the hopper to the breathing zone.* The exposures were continued for a period of 25 months, during which time 20 non-tuberculous animals were killed and studied. After 18 months considerable diffuse dust reaction had developed but there were no silicotic nodules. During the next 7 months the amount of reaction increased but in no instance was there evidence of nodulation. The 15 surviving animals were removed to a normal atmosphere and killed at intervals during the following 6 months. In 6 of the 15 a small number of nodules developed but in only 2 of them were such lesions of mature type. In the lymph nodes draining the lung the reaction was slightly more advanced. Mature hyaline nodules were first noted after 23 months exposure but they

appeared in only 8 of the 15 animals killed after stopping the exposure.

This experiment seemed to offer confirmation of the theory [illegible] from the study of the men in the plant. Controlled observation had demonstrated that the percentage of silica in the original mixture had been reduced from 50 to 31% as the dust floated into the animals' cages. With a total concentration of 250 million particles this should give 77.5 million quartz particles at the breathing zone. Experience had indicated that after 2 years exposure this concentration of the same quartz, without the added gypsum, would produce extensive nodular fibrosis in practically all the animals exposed. When an equal amount of gypsum was added, only about half the animals surviving the exposure period showed any nodules and in them the lesions were few in number and mature in only [illegible] cases. Obviously not much of the silica in the atmosphere of the cages was inhaled. The gypsum was protect[illegible]

The experiment was repeated using one [illegible] of the same quartz dust and three parts of gypsum in the hopper. The concentrations were kept higher, with an average light field count of 642,000,000 per cubic foot and dark field 1,[illegible]. The reduction of the silica from the hopper to the breathing zone was [illegible], a concentration of 85.6 million light field and [illegible] million dark field per cubic foot being available inside the cages. Both guinea pigs and white rats have now been exposed under these conditions for a period of 16 months without developing mature silicotic fibrosis of the lungs. In occasional animals killed after 14 months or later, there have been foci of hyaline fibrosis in the [illegible] bronchial lymph but this has not been a constant finding. The character of the reaction has not progressed regularly throughout the series of animals killed at successive intervals.

The exposures to this mixture will be continued as long as animals survive in the hope of demonstrating decisively whether any mature silicosis will develop.

To make certain that this apparent protective action occurred in the atmosphere and not inside the lungs of the animals, a group of [illegible] guinea pigs was exposed on alternate weeks to pure quartz and to pure gypsum. The dust concentrations in the gypsum [illegible] 413 million per cubic foot for [illegible] 1,160,000,000. In a year's time the animals [illegible]

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[illegible]

showing typical nodular fibrosis and by [illegible] had fully matured. There was no evidence of protection by gypsum within the lungs.

Since calcined gypsum alone causes hardly any reaction when inhaled into the lungs or upon injection in various tissues it seems safe to conclude that the inhibition of reaction to high concentrations of quartz mixed with the gypsum was a result of agglutination of the two kinds of particles. This occurred, in part, in the atmosphere through the formation of heavy clumps of quartz and gypsum which settled and thus reduced the silica content of the dust before it reached the breathing zone inside the animals' cages. Presumably a further reduction in the silica content of the dust took place in the tortuous passages of the upper respiratory tract. There the clumps [illegible] were readily retained than individual, small, discrete particles of silica. If this is the only factor needed a time will probably come when enough silica has accumulated in the lungs to cause reaction. Whether this will be attained during the lifetime of an experimental animal remains to be demonstrated. Apparently it does not occur in men exposed under industrial conditions.

A Saranac Laboratory study of 6800 hematite miners in the Lake Superior district reveals a gross incidence of nodular silicosis of 20%, which approximates the average figure of 25% found in [illegible] many hard rock mines the world over. But the nodulation which many of these men show is of a fine type and the disabling massive complicative forms of the disease are comparatively rare. The gross incidence of tuberculosis, most of which are presumably tuberculous, is only about twice that of the adult male population of the same area. Among the deaths recorded since the study was begun 4 years ago, 54% occurred in men previously diagnosed as silicotic. In this silicotic group pulmonary tuberculosis was certified as the cause of death in 23 or 43.4%. The accuracy of the figures is no greater than in any mortality statistics as only part of the cases came to autopsy, but the apparent incidence of fatal tuberculosis is low in comparison with the standards obtained elsewhere. Among the English autopsy cases with silicosis, the incidence of tuberculosis was 59.1%, and in the Saranac Laboratory cases it was 65%. Both of these groups were made up of persons with miscellaneous exposures to silica in various industries. The autopsies that have been obtained on iron miners have revealed varying stages of siderosilicosis of the types described above.

A long series of experimental studies have been made with dusts of the hematite ore and the ferruginous chert from these mines, which have shown some important results. They are illustrative of the modifying action of other substances upon silica which [illegible] miners are encountering today. The "hematite" dust consists of a mixture of hematite, ferruginous chert, normal quartz and a small amount of jasper. Chemical analysis reveals 69% Fe_2O_3, 16% FeO, 4% CaO and smaller amounts of MgO and other substances. The [illegible] is ground so that 65% of the particles are 5 microns or less in diameter. It was used in an inhalation experiment involving 109 guinea pigs. Analysis of the air flow [illegible] inside the animal cages revealed 79% Fe_2O_3 and 7.03% SiO_2. Both elements being visible, more silica than iron in the dust placed in the hopper. The average dust concentration maintained for 3 hours daily was 9.0 mppcf by count by light field, and 6.625 mppcf by dark field. From 5 to 6 on the motor agitating the dust in the hopper was turned off so [illegible] animals still remained in the same room and [illegible] dust settled on their bedding. The average counts between hours 6 to 24 varied between 3 and 7 million per cubic foot by light field and 50 to 200 million by dark field. The concentration of respirable quartz in the atmosphere when the [illegible] was 3.06 million per cubic foot by light field and [illegible] million by dark field count, a very appreciable concentration in addition to the lesser amounts during the remaining 16 hours of each day. The maximum period of exposure was 3½ years; at the end of this time a number of surviving animals were removed to a normal atmosphere and killed at intervals during a subsequent period of 8 months.

At the end of the exposure the pleural surfaces of animals killed showed heavy streaks and spots of blackened iron dust and their tracheobronchial lymph nodes were likewise deeply pigmented. But on macroscopic examination there was no evidence of fibrosis. There were large accumulations of giant phagocytes packed with particles of iron in the subpleural and some of the deeper air spaces. There were masses of such cells in the connective tissue along the lymph planes and in the lymphoid tissues but this dust had caused only a minimal amount of chronic inflammation and nothing that could be called fibrosis. There were no silicotic nodules. Microscopic sections illustrated these changes.

Chemical analysis of some of these lungs yielded 0.[illegible]% of

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ash, about 3 times that in a normal guinea pig [illegible] 6.1% of Fe_2O_3 and 1.8% of SiO_2.

Here were animals exposed for 8 hours a day for a period of 3½ years to 540 million particles per cubic foot of a fine iron ore dust containing 40 million particles per cubic foot of the same sized quartz. Some of the silica entered their lungs but not enough to produce any suggestion of a silicotic type of reaction.

Ferruginous chert—like the hematite consists petrographically of a mixture of hematite, ferruginous chert, normal quartz and rutile, but on chemical analysis it is shown to contain 46.8% of SiO_2. Experiments have been made with two different lots of this material. The first was ground at the mine but only about [illegible]% of the particles were of hygienic significance (3 microns and under). Much of the silica being harder and more resistant to grinding was of large size. Nevertheless an experiment was attempted hoping that by using excessive concentrations enough silica would be present at the breathing zone to produce a sidero-silicosis.

When the behavior of this dust was investigated in the dusting room it was found that there had been a marked reduction of its silica content between the hopper and the breathing zone in the animals' cages. The following analyses are illustrative:

	Original Dust	Rafter Sample	Impinger Sample Particles 4 microns and under
SiO_2	46.8%	38.4%	10.47%
Fe_2O_3	33.4	55.3%	80.93

The average dust concentrations were as follows:

For 8 hours daily:

Light field counts 1,095,000,000 per cubic foot.
Dark field counts 4,025,000,000 per cubic foot.

For 16 hours daily:

Light field counts 700,000 to 4,400,000 per cubic foot.
Dark field counts 33,800,000 to 56,000,000 per cubic foot.

The inhalable silica concentration for the 8 hour daily period was 114,000,000 per cubic foot light field and 121,400,000 dark field.

Eighty guinea pigs were exposed under these conditions for a maximum period of 1 10/12 years. Twenty of them survived and were removed to a normal atmosphere; they were killed at intervals during the following 2 1/12 years.

Sample animals autopsied at the end of the 1 10/12 year exposure were essentially the same as the animals already described under the hematite experiment although there was possibly a little more evidence of chronic inflammation about the dust deposits. Chemical analysis of their lungs showed only 10.25% of ash of which 5.8% was SiO_2 and 59.1% Fe_2O_3. (The smaller percentage of ash is undoubtedly due to the shorter period of exposure.)

As the survivors were killed during the following 2 years there was no appreciable change in the gross or microscopic appearance of the dust reaction in the lungs. There was, however, a decrease in the amount of silica discovered by chemical analysis. It dropped from 5.8% of the ash at the end of the exposure to 3.18% [illegible] year later. The iron showed a corresponding decrease from [illegible] to [illegible]. It was assumed that this decrease was due to lymphatic elimination to the tracheobronchial lymph nodes since microscopic sections of these structures showed an appreciable [illegible] in the amount of dust present. This assumption [illegible] chemical examination because in each case all of the [illegible] were reserved for microscopic preparations and were not subjected to chemical analysis.

In this experiment animals had been exposed to nearly three times the amount of silica and nearly twice the amount of iron but the duration of the exposure period was only 2/3 as long as with the hematite. The maximum total period of life, including dusting and subsequent "elimination" was shorter by 5 months. Again no suggestion of silicosis had appeared. The same concentration of quartz without the addition of the iron has produced typical [illegible] in about 1 year's time and the lesions progress when animals are removed to a normal atmosphere.

To satisfy ourselves whether sidero-silicosis could be produced in experimental animals a new lot of ferruginous chert was obtained from the mine and reground so that all of the particles, quartz, iron and other elements were of a size significant for the purpose in hand.

The following table shows the variation in the chemical composition of samples of the reground chert after it was placed in the dusting room:

	Original Dust	Rafter Dust Sample 1	Rafter Dust Sample 2	Impinger Sample Sample 1	Impinger Sample Sample 2
SiO_2	67.58%	60.36%	61.71%	40.21%	[illegible]
Fe_2O_3	28.12	30.60	30.10	40.80	[illegible]

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The particle sizes of the atmospheric dust were as follows: less than one micron, 85%; 1 to 2 microns, 13%; 2 microns and over, 2%. Note that even with the fine dust there is still a very appreciable reduction in the percentage of silica from the hopper dust to that inside the animals' cages. With the coarser silica in the first chert experiment the reduction was 2½ times greater.

The total dust concentrations maintained for 8 hours daily were as follows:

Light field 552,000,000 per cubic foot.
Dark field 1,983,000,000 per cubic foot.

The silica component is estimated at 230,000,000 light field and 793,000,000 per cubic foot by dark field count. The amount of dust present during the remaining 16 hours of the day was not determined.

In the atmosphere of chert dust thus defined have been placed 100 guinea pigs, 25 white rats and 15 rabbits. The course of the reaction has been followed for 2 years and thus far there has been as little or even less pulmonary change than was observed in the first chert experiment. The lungs showed pneumoconiosis due to deposits of dust in focal areas in the air spaces and in the connective tissues. There was no fibrosis and no suggestion of nodule formation. The walls of air spaces containing collections of dust cells were slightly thickened by infiltration with chronic inflammatory cells but the included capillaries were still visible. Most of the collections of dust cells a little emphysema had developed.

A chemical analysis after the maximum exposure showed 15.5% of ash of which only 1.6% was SiO2 and 49.4% was Fe2O3. Obviously the amount of silica which reached the lungs after 2 years exposure to high concentrations of quartz known to be of inhalable dimensions was very small.

The white rats in the same experiment have been followed for a maximum period of 18 months. Their lungs show only early silicotic lesions, a change that has been observed after only 6 or 8 months exposure to quartz alone. In their lymph nodes more mature fibrosis developed after 18 months. The experience with these animals is confirmatory but rather surprising for the white rat is very sensitive to silica and its tissues react to very small amounts of it.

The rabbits were all of the blue Chinchilla variety. Each is to be peculiarly protected against inhalation of dust. After 6 months there was little dust in their lungs and no evidence of a silicotic type of reaction either here or in the associated lymph nodes.

To prove whether the chert was capable of causing a reaction if a sufficient quantity established contact with the tissues, a group of rabbits were tested by the intravenous injection method. The first received a total dose of 2 grams of 1 to 5 micron particles containing approximately 1 gram of free silica; the second received a gram half the dose or 0.5 gm. of silica per rabbit. With each dose the animals were killed and studied at 1 year after the injection. The old rabbits in the 2 gram series were killed at 11 years.

When injected in this manner the chert produced a progressive fibrosis of the liver, spleen, lymph nodes and lungs. The fibrosis did not develop as rapidly as the experimental dust silicosis above. With the smaller dose of one gram, after 11 years, there were only bands of fibrous tissue containing nodules here and there in the liver and spleen. With the double dose most of the liver had been destroyed and replaced in one year's time. A dose of 0.1 gm. of pure silica has the latter effect in one year.

This experience, like that with gypsum, coal dust and other siliceous dust mixed with silica would protect against the action of the latter. The most obvious explanation was that the chert in the atmosphere with the formation of clumps which were too large to remain in suspension *in vivo* to pass the upper respiratory passages. Even with it was *recognized that the amorphous* form of the silica in chert was somewhat modified by the presence of iron, the findings did not seem to be of such great significance as the difference in reaction which was obvious even in animals injected with many times the quantity known to be necessary to produce fibrosis.

To make certain whether the heavier might also affect the response to silica after both substances were already in the body, an additional experiment was done in which guinea pigs were exposed on alternate weeks to pure quartz and then to hematite alone. The quartz dust concentrations were high, 800,000,000 per cubic foot (light field) for 8 hours daily. The hematite exposures were made at the same time as the other experiment and were of concentrations of 580,000,000 by light field. The experiment was con-

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[illegible] for a total period of 2[illegible] months and the [illegible] a normal atmosphere for 6 more months. A [illegible] was sufficient to produce active connective tissue proliferation about accumulations of dust cells in the lymphoid tissues and in the pulmonary framework. Such reactions increased in amount during the next 10 months of exposure and still more in the following 6 months out of the dust. Local areas of actively growing cellular connective tissues were formed. They had ill defined borders which radiated into the normal connective tissues of the surrounding lung. However, there was no necrosis nor hyaline formation which characterizes the mature silicotic nodule of the guinea pig. In the tracheobronchial lymph nodes where more silica is concentrated, hyaline nodules did develop after only 6 months of exposure and their number increased in most of the animals killed subsequently.

This experience indicated that if the silica got into the lungs it would produce reaction but that reaction was actually modified and retarded by the iron dust in the immediate vicinity. The mature type of reaction characteristic of pure silica in the guinea pig never developed in the lungs but there were lesions which closely simulated those regularly found in human beings exposed to the same kind of dust. In the lymph nodes mature silicotic foci were found 5 months earlier than they usually occur in guinea pigs exposed to quartz alone. This finding would suggest that the silica was being eliminated from the lungs to the lymph nodes at an accelerated rate. Years ago Haldane and Mavrogordato had a similar hypothesis with respect to the action of coal dust. But speculation is out of place here. An unexplained but well verified observation has been made, iron oxide retards as well as modifies cellular reaction to silica. Whether the effect is permanent or whether in time the silica already in the lung will produce characteristic reaction, only further observation can establish. Certain men in the iron mines from which this dust came develop more typical silicotic nodulation. But time that is the factor which has been minimized in these experiments? Or did the men who have manifested the mature type of lesion develop such changes during exposures to relatively pure silica dust encountered in rock development work? No answers are possible at the present.

The analyses of industrial atmospheres and those in the experimental dust rooms have indicated the extent to which admixture with various substances may reduce the atmospheric concentration

of silica between the point of dust generation and the [illegible] zone.

The experimental data on the inhalation of granite, of iron ore in natural combination with varying quantities of silica and of [illegible] artificially mixed with different amounts of finely ground quartz all point to a protective action by these [illegible] substances. Such protection may manifest itself in the atmosphere and indicates the desirability of a detailed study of the properties of air-borne mixtures of silica and other types of dust. In addition the injection experiments and the alternate inhalation experiments both indicate that iron ore and the silicates associated with quartz in granite dust may inhibit the action of silica on the tissues.

RESPONSE TO INHALED ASBESTOS FIBRES

The reaction of the lungs to inhaled [illegible] different from that to any of the particulate dusts [illegible] finding suggest that the body has not developed an effective defense against fibrous foreign bodies. Asbestos fibres are not removed from the functional portions of the lungs and deposited in the tracheobronchial nodes; they tend to remain [illegible] to rest.

During the lung they are caught on their regular walls of the terminal respiratory bronchioles. There they are engulfed by phagocytes which often enlarge and become multinucleated giant cells. Such cells do not move very far; they either adhere to the wall of the air passage or they enter its substance. Stimulation by the fibres results in the development of a mild degree of fibrosis which develops as a cuff about the bronchiole. In animals the reaction is not a progressive one which continues after cessation of exposure as in the case with free silica. Any fibrosis that is present merely becomes more dense due to organization and contraction. A hypothesis [illegible] held by the speaker, that constriction of the bronchial [illegible] would result in collapse of its peripheral air spaces with resultant fibrosis from the collapse itself has not been verified by further study. It would seem now that the fibrosis in the peripheral portions of the respiratory units is produced by deposition and reaction to new increments of dust in the terminal air spaces. As the wall of the respiratory bronchiole undergoes fibrosis its inner surface becomes smoother and the tube loses its elasticity. The inhaled fibres [illegible]

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pass on through it and are deposited in the lung beyond. When the intrapulmonary lymphatics reach the surface of the lung, the pleura becomes inflammed and a local fibrosis ensues. The mechanism of its involvement is not altogether clear as asbestosis bodies are generally scarce in the pleura itself.

Animal experiments have thus far failed to reproduce the widespread fibrosis seen in the human cases that have come to autopsy. Whether this is due to insufficient exposures or whether it is because the fatal human cases have been complicated by an element of healed infection still awaits demonstration. The speaker suspects that the latter may be the case for many infections, including tuberculosis, show a decided tendency to deal with the production of excessive scar formation in animals with asbestosis.

The method by which asbestos exerts its effect has not been clearly defined. There are some who believe that asbestosis is merely a silicosis which manifests itself in a different anatomic form. They hypothecate that the magnesium is leached out of the silicate molecule leaving silica atoms to act upon the tissues. If this were true one would expect asbestos to exert its effect upon connective tissue in any part of the body as happens when free silica is injected, but such is not the case. Sayers and Miller failed to produce fibrosis by intraperitoneal injection of chrysotile asbestos and have classified this substance as "inert". Similar results have been obtained in the Saranac Laboratory both in the peritoneal cavity and other tissues of the guinea pig and in the extra pulmonary organs of other species. In the light of present experience asbestos seems to be a dust capable of producing reaction only in the lungs. Possibly the irritation in this organ is largely mechanical although this remains to be demonstrated.

An unusual feature of this disease is the formation of the asbestosis body. This results from the development of an iron-containing coating upon the surface of inhaled asbestos fibres. It appears after about 2 months contact with the pulmonary tissues. Injection of asbestos into other tissues usually fails to produce such bodies although a very occasional one has been discovered in the peritoneal and subcutaneous tissues. As the coating thickens it fractures into segments whose edges become smooth and rounded. The structures are bright chrome yellow and may be 90, 70 and even 100 or more microns in length, and 2 to 4 microns in width. They resist diges-

tion with trypsin and strong alkali. Experiments [illegible] that [illegible] results partly in the production of [illegible]. Dr. Mills reports a case in which death occurred 2 years after a short exposure to asbestos dust. Sections of the lung, which the speaker has studied, still show what appear to be the remnants of heavily digested asbestosis bodies in addition to large numbers of uncoated fibres. This finding in an isolated case suggests that some of the coating on the fibres may be reabsorbed. Many observers have reached the conclusion that the coating substance is laid down by the body to protect itself against irritation. In support of this view is the fact that numbers of bodies digested out of the tissues with antiformin and injected into animals failed to produce fibrosis or other reactions. But if this explanation is correct we should expect similar deposits on foreign bodies of other types but this is not the case. It seems much more probable that a common [illegible] is secreted, the coating material is deposited [illegible] and then is [illegible] on its surface. The iron may be [illegible] or from the body fluids as proved by experiments [illegible].

The significance of asbestosis bodies in the [illegible] tissues depends upon the occurrence of a [illegible]. If only a very few are present they [illegible] of isolation and hence do not mean [illegible]. The speaker has found occasional ones in the lungs of at least a half dozen cases where there was no industrial exposure to asbestos dust.

RELATIONSHIP BETWEEN PNEUMOCONIOSES AND TUBERCULOSIS

This section on infection relates particularly to tuberculosis for while it is generally granted that all types of pneumoconiosis predispose to infection are more common in the silicoses than the normal lung the evidence in support of this view is not too clearly defined. It is becoming to be recognized, for example, that lobar pneumonia is not unusually common or unusually fatal in persons with silicosis. The same may not be true of the bronchopneumonias, which need [illegible] more investigation. But there is plenty of evidence to prove a [illegible] etiologic relationship between silicosis and tuberculosis [illegible].

Experimental Methods—have yielded a wealth of clinical and statistical investigations which prove that [illegible] are interrelated. Price of Toronto demonstrated that the white mice

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soluble sodium silicate to artificial culture [illegible] found the growth of tubercle bacilli. The observation was confirmed by Kettle and by Cummings in the laboratory both for the silicate and for fine crystalline silica. In using quartz, however, evidence of stimulated growth was not obtained and Kettle and both Cummings and Kettle concluded that the silica, perhaps acting as a buffer, merely accelerated the growth at the start. More recently Vorwald, Dworski and Steendon in the Saranac Laboratory have renewed their attack upon this problem and have demonstrated that the amount of growth obtained from certain synthetic media to which quartz has been added actually weighs four times as much as that from control media without quartz. They now seem to be fairly on their way to define the conditions under which such stimulation occurs.

In the animal body Kettle, the Saranac Laboratory group, and others have demonstrated that tubercle bacilli begin to grow quite rapidly in tissues that are reacting even mildly to injected or inhaled silica. When the silicotic lesions are extensive and a true inflammatory response have subsided, the stimulating effects are not so obvious, in fact it even appears that the bacilli barely remain alive in old silicotic fibroses. Perhaps this is due to other inhibitory factors such as decreased blood supply and lowered oxygen tension.

A variety of methods is available to the experimentor. The one most effectively applied in the Saranac Laboratory has been that of inhalation infection of guinea pigs with the attenuated strain, R_1. In normal control animals such infection will produce a self limited tuberculous infection which in the great majority of cases heals and disappears within two or more years. In animals inhaling silica the infection almost invariably progresses, resulting in disease which is either acute or chronic as the time of the infection relates to the coexistant fibrosis. In guinea pigs exposed to other types of dust, R_1 inhalation infection typically heals but the character of the healing process varies with the type of dust involved—sometimes the tubercles calcify and become fibrous, at others they spread for a time and then heal with excessive scar formation. Since in general the behavior of the R_1 inhalation infection has tended to parallel what is known about tuberculosis in human lungs exposed to various types of industrial dusts the scientist has been inclined to accept the experimental evidence as indicative of what may happen clinically in the case of dusts whose action is unknown.

[illegible] the [illegible] the [illegible] in the adult human [illegible] primary origin such as the one we have studied. [illegible] criticism we ourselves are the first to recognize, in fact from the very outset we have attempted to verify conclusions as well as to prove new ones. However, the technical obstacles are great. [illegible] to the lung always stimulates the primary tuberculous [illegible] regardless of how or where it is administered [illegible] the introduction of endogenous [illegible] before or after the [illegible] can be given. While the same is not true of [illegible] desirable to keep conditions constant for purposes of [illegible]

At the present time the primary R inhalation [illegible] provides a [illegible] different kinds of dust [illegible] reliable method at our command.

Other techniques are confirmatory or may be used [illegible] trends which must later be verified by the [illegible] regularly which they suggest stimulate [illegible] is that of intravenous injection of [illegible] into rabbits. Human organisms are ordinarily of low [illegible] and small doses do not usually cause progressive tuberculosis [illegible] normal rabbits; in silicotic ones they do.

Kettle devised his white mouse [illegible] which [illegible] reported that tubercle bacilli would multiply rapidly in [illegible] mixed with silica particles. But the procedure [illegible] that it depends upon chance contact between the [illegible] and the dust particles. Furthermore, when a variety [illegible] dusts, including many of the silicates, were [illegible] studied (Vorwald and Lindau), [illegible] were as active and even more active than silica in [illegible] multiplication of tubercle bacilli. Since there is no [illegible] to support these findings, they are at the present [illegible] suspicion. Before accepting them they must be [illegible] data either clinical or experimental. The Kettle technique consists in the injection of a small quantity of dust particles, suspended in salt solution, into the subcutaneous tissue of the thigh of a mouse. After allowing 4 or 5 days for reaction between the injected particles and the tissues, a suspension of living tubercle bacilli is injected into the animal's tail vein. Bacilli which [illegible] the blood stream and lodge in a focus of silica nodules [illegible] those lodging in a focus of non-[illegible]

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theoretically multiply very slowly. As used in the laboratory, every animal was injected with silica in one flank and with one or more other types of dust in other parts of the subcutaneous tissue. The blood did not always carry bacilli even into the silicotic foci so that negative results could not be evaluated. Positive results were only considered suggestive when a majority of animals so injected showed evidence of bacterial multiplication.

Time permits only the briefest summaries of the results which have been obtained by the use of these different experimental methods. (A series of lantern slides was used to demonstrate the following.)

The self-limited multiple primary complex produced by the inhalation of attenuated *R*, bacilli in normal guinea pigs was shown first, pointing out that the subpleural tubercles in the lungs attain the stage of ulceration but do not spread and that these foci and associated cases in the tracheobronchial lymph nodes heal and almost completely disappear within a period of 12 months to 2 years.

When such infection is given to guinea pigs inhaling silica the outcome depends upon the stage of the reaction to silica.

If infection is administered at the time of starting the dust inhalation a chronic progressive tuberculosis begins to develop after 5 or 6 months which kills the animals in about a year's time.

If infection is given after the inhalation of dust, the tuberculosis tends to become increasingly acute as the silicotic reaction approaches maturity.

1. After 3 months exposure to excessive concentrations of silica dust there is no specific tissue reaction but inhalation infection produces many more tubercles than in normal control animals. The effect is also observed with other types of dust and is therefore not specific. In the silicotic pigs, however, most of the tubercles in some of the animals tend to spread locally; in other forms of pneumoconiosis they heal.

2. After from 4 to 6 months exposure to dust there are early silicotic reactions which cause some progression in a majority of the tubercles.

3. After 10 months dust exposure, immature non-fibrous silicotic nodules have developed which, combined with superimposed infection, result in a chronic ulcerative tuberculosis.

4. After 12 months or more of exposure to the dust, silicotic nodules become mature and hyalinized. Infection now causes an acute tuberculosis which in every case terminates fatally within 3 or 4 months.

If infection is administered first and the tubercles allowed to heal partially before the dust exposure is begun, there are the tubercles which still harbor living bacilli to be reactivated by silicosis. The progressive, destructive effect of silica on the tubercles and phagocytes must cause the bacilli to spread from the caseous centres. Tubercles, which do not happen to have inhaled appreciable amounts of silica, heal by scarring and are cured of the disease. The intimate contact between [illegible] a direct or an indirect chemical [illegible] although other explanations are possible.

The inhaling silica and infected guinea pigs [illegible] type tubercle bacilli [illegible] however, more frequently [illegible] of progression. Excessive formation of scar tissue [illegible] cavity formation and other characteristics [illegible]

The inhalation infections in guinea pigs exposed to various types of dust have produced the following results:

1. *Mixtures of quartz and gypsum dust* (50% SiO_2)
Temporary progression of the tuberculous infection followed by fibrous healing of [illegible]

2. *Hematite* (containing 6% SiO_2)
More extensive progression of the infection [illegible] fibrous healing, often with loss of specific [illegible] tissue changes. Cases worse than [illegible] gypsum-quartz dust.

3. *Ferruginous chert* (approximately 30% SiO_2)
Still more progression of the infection but [illegible] same tendency to fibrous healing may occur [illegible] arate animals. This result is not [illegible] from [illegible] the

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le matite group, some animal dusts, coal mines, silicotic tuberculosis with cavity formation

4. *Granite*

This dust, a natural mixture of [illegible], quartz and [illegible] silicates, influences tuberculosis in a manner similar to that of clay. It delays healing, promotes fibrosis and in some instances causes chronic silicotuberculosis. Its behavior is somewhat erratic and the same results are not always found in animals when the experiment is apparently identical.

5. *Asbestos*

The infection progresses peripherally in most animals; it subsequently tends to recall both the formation of much more scar tissue than in the animals inhaling asbestos dust alone.

6. *Calcium Dusts, marble and lime dust*

There is practically no spread of the infection although rare exceptions occur. The tubercles heal but do not always resolve. Caseation is definitely more frequent than with other types of dust.

7. *Bituminous coal*

There is no spread of the infection produced by R, type of bacilli. Some tubercles fail to resolve and become fibrous. In animals infected with fully virulent tubercle bacilli there is no evidence that inhaled coal dust protects against such infection.

8. *Silicon carbide*

For unexplained reasons this substance has behaved like free silica in influencing the course of R, tuberculosis. In 3 different experiments, two of them inhalation infections and one subcutaneous infection, the inhalation of this dust has produced chronic progressive tuberculosis. This is the only experimental result thus far obtained which is at variance with clinical observation. Dr. Cook and others who have studied it have exposed to the carbide of silicon have failed to show any excess of tuberculosis. The reasons for these experimental findings are still

unexplained. The dust also causes moderate chronic inflammatory changes with nodule formation but contains practically no free silica.

These experiments demonstrate that inhaled particles of quartz dust almost always transform a primary [illegible] tuberculous infection into progressive disease. The chronicity of the disease will depend upon the amount of silica present. If the dust does not enter the picture until the tubercle is acute and generally fatal in a short time. If the tubercle bacilli have been kept alive and protected from [illegible] for [illegible] weeks [illegible] without the complicating dust [illegible] results on the interval before progressive [illegible] occurs depends on how much infection is building up a resistance [illegible] Experimentally the spreading disease evokes [illegible] productive reaction. [illegible] experimentally healed R infection when [illegible] of silicosis. Here again the [illegible] chronic in its course.

[illegible] artificial mixture [illegible] R infection [illegible] the dust. But there is [illegible] to progression in the animals exposed to [illegible] dominates the picture and [illegible] characteristics of tuberculosis are [illegible] all have difficulty in recognizing [illegible] encountered in uncomplicated [illegible] found in the lungs of [illegible] have materially facilitated the interpretation of [illegible] human lungs.

[illegible] nonsilicious dusts, R, tuberculosis [illegible] healing by resolution may be expected [illegible] of frequent occurrence. [illegible] and returned in the sterile caseous [illegible] of the tubercles. No evidence has been produced [illegible] a tuberculous infection [illegible] years ago.

The silicates, asbestos, frequently [illegible] R infections but subsequent [illegible]

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...is the rule. Many human cases have been reported ... instances. However, none of these that have come to our ... of chronic progressive tuberculosis. Post-mortem material is by no means adequate, however, to obtain information as to the frequency of healed and active tuberculosis in any group of persons with pneumoconiosis; the roentgenogram is much more informative. The effect of the other dusts studied has thus far been studied only by injection methods. The results cannot be considered trustworthy until verified by the inhalation technique and clinical observation.

In ... of the studies of pneumoconiosis and associated infection it has become increasingly obvious that the scar of a healed infection may accumulate excessive quantities of dust inhaled subsequent to its development. This point probably has considerable significance in the interpretation of some of the massive conglomerate areas of fibrosis found in human lungs.

Another point of considerable practical significance is the variability of the reaction in animals infected with tubercle bacilli and compelled to inhale mixtures of silica and other substances. Although the treatment of all members of any group was as nearly identical as possible, the individuals tend to react differently. This is not nearly so frequent where infection is associated with the inhalation of pure silica. The modifying action of the non-siliceous components of the dust are frequently able to cause an unfavorable outcome but in dealing with living variables like the tubercle bacillus and the animal body unpredictable influences will always appear to tip the balance. The same is of course true in any study of human cases.

PATHOLOGY OF THE PNEUMOCONIOSES

CORRELATION OF PATHOLOGIC AND ROENTGENOLOGIC FINDINGS

Dr. L. U. Gardner
Saranac Laboratory

The various manifestations of reaction to inhaled dust were illustrated by a series of lantern slides. They were classified under the following categories:

Non-specific dust reactions.—As far as is known today, all inorganic dusts other than free silica and the silicates produce but one general type of reaction. It is possible that the fibrous development is subject to modification in the case of dusts the lungs of which have been infected by tubercle bacilli, but ... of the exceptions is not well docketed.

The characteristic features of the reaction to inhaled inorganic dusts are:

1. Deposition of dust in the walls and ... of the lymphatic vessels ... vessels and bronchi and in the interlobular ... focal deposits in the lymphoid tissues ... and at their roots.
2. The development of chronic inflammatory ... deposits, which is so slight that it requires a ... to detect it.
3. After prolonged exposure to excessive concentrations of dusts, collections of over-engorged dust cells ... within groups of subpleural air spaces. The ... such spots produce slight chronic inflammatory ... thickening but there is ... on the surface ... on gross examination of the lungs.

Roentgenograms of persons with such non-specific dust ... show an exaggeration of the linear shadows normally ... the branches of the pulmonary arteries and veins. This is ... by the fact that the lymphatics coursing through the walls of these vessels are ... most exposed and when they are surrounded by dust ...

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and associated chronic inflammation they appear [illegible] the wall of the blood vessel.

Similar thickenings of the linear shadows in the lungs may be caused by other conditions and hence the appearance in no sense is specific for pneumoconiosis; chronic pulmonary infections, disease of the heart and arteries and some other conditions may also thicken the blood vessels and hence they cast a similar shadow. In the early stages of the reaction to silica dust, before nodulation develops in the lungs, there is perilymphatic reaction which causes the same effect. For these reasons it is difficult to evaluate the significance of exaggerated linear markings in a pulmonary roentgenogram. A history of exposure to dust does not facilitate diagnosis for the individual so exposed may have also experienced other causes of the same condition.

Fortunately the matter is of no great moment for such reactions in no way interfere with pulmonary function and they are not a cause of disability. The change probably does not influence susceptibility to tuberculosis and certainly not unless it is produced by relatively high concentrations of free silica. The difficulty here is to be certain that the silica to which the individual had been exposed was the cause of the exaggeration. Among our autopsy specimens of persons exposed to different types of dust, the incidence of active tuberculosis was [illegible] in lungs with the linear perilymphatic type of reaction in contrast to [illegible] in those with silicotic nodulation.

Pure Silica Reactions are manifested in several forms and these again differ as they occur in either a normal lung or one with infection. The various forms are necessarily described individually.

A. SILICOSIS IN AN OTHERWISE NORMAL LUNG

1. *Preliminary stage of linear perilymphatic reaction.* Such reaction can only be differentiated from the non-specific type described above by microscopic examination. By such inspection one may be able to detect the silicotic type by the presence of associated minute, fibrous nodules in the lymphoid tissues. Gross examination fails to reveal such nodules in the early case and the roentgenogram of course cannot reflect their shadows.

Probably persons with such reactions, like the guinea pigs previously described, are more susceptible to tuberculosis than normal subjects. Statistical evidence of such increased susceptibility may

[illegible] exhibit excess of infection in an [illegible] from one industry but it is very difficult to apply this [illegible] the cause of the infection in an individual. A definite proportion of the population have tuberculous infections [illegible] their occupations and the linear markings on the film are not [illegible] of silicosis. In South Africa they have found [illegible] to difficult from a compensation aspect that they pay [illegible] tuberculosis in miners whether there are demonstrable evidence of [illegible] or not. Ontario has gone to the other extreme and [illegible] the silicotic with tuberculosis by [illegible] when infection ensued on the ground that a proportion of all [illegible] regardless of occupation, would develop tuberculosis and [illegible] to determine in which case the silica [illegible] infection.

2. *Stage of Discrete Nodulation.* Gross examination of the lungs shows sharply defined, hard fibrous nodules [illegible] and usually 2 or 3 mm. in diameter, uniformly [illegible] out both lungs. The number and size of the nodules [illegible] with the intensity and duration of exposure. The outer portions of the lower lobes of the distended lung are frequently free from nodules but in their stead exhibit a well marked [illegible] is not obvious in the collapsed lung. The explanation is probably that the emphysema developed after the nodules and that as these fibrous structures contracted they drew up the lung [illegible] then became over-distended to fill the available space. In the early phases of nodular silicosis the tracheobronchial lymph nodes may enlarge from the reaction to the silica that they contain. As fibrosis develops in them the contracting scar tissue again decreases the size of the organs.

A roentgenogram shows the shadows of these clear-cut nodules uniformly distributed throughout both lungs with or without associated clear zones of emphysema in the outer and lower portions. The shadow of the mediastinum is wide if the contained tracheobronchial lymph nodes are enlarged. It is [illegible] to classify cases of silicosis according to the number and size of the nodular shadows present and hence the division into stages 1, 2 and 3. This classification is based upon the assumption that a regular progression from the first stage occurs. There is some [illegible] as to the value of this procedure; it was decided to omit [illegible] the Johannesburg Silicosis Conference.

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B. SILICOSIS IN A LUNG ALREADY PREVIOUSLY DISEASED

Many cases of silicosis, in addition to the above described discrete nodulation show massive areas of confluent fibrosis in one or more parts of the lung. Usually such lesions occur upon a background of generalized nodulation but in some instances the massive areas are the only manifestations. Associated with such changes there is a chronic fibrous pleurisy and a coarse emphysema in which the margins of the lungs are blown out into large bleb.

Such reactions have been ascribed to many causes and perhaps with considerable justification for obviously one not yet arrived at to the exclusion of others is possible. It is commonly believed that these massive lesions resulted from confluence of nodules which were once discrete. A committee of which the speaker was a member described them in this manner in its report published by the Public Health Service in 1935. Strachan and Irons believe that they develop in areas of atelectasis or collapse of the lung produced by pressure of a silicotic nodule on a bronchus supplying the area involved. Gulland, Armstrong and Fagan, the speaker, think that most of the reactions of this type are due to the excessive accumulations of dust in portions of the lungs damaged by infection. Perhaps other causes are the result of the action of mixtures of silica and other substances for they are always heavily pronounced by non-siliceous dusts and they tend to be most frequent in certain industries where mixed dusts prevail. Perhaps both healed infection and the non-siliceous admixture play a part for such is the implication from experimental observation. One speaker has yet to discover a case where atelectasis could be incriminated. A simple conglomeration of previously discrete nodules is inadequate to explain the pathological course.

If one examines such areas carefully it is noted that the mass of scar tissue, which is dense and tough as shoe leather and usually black or slate grey in color is made up of spherical nodules embedded in dark, distinguishable from a more diffuse matrix of the same composition. It is not composed of nodules closely packed together. The whole mass has an ill defined outline; there are long strung like processes of the fibrous tissue radiating from its borders into the surrounding lung. The masses may be single but often they are multiple and occur in both lungs. Sometimes they occur in the

upper lobes where tuberculosis is commonly found. [illegible] the nodules are also present the [illegible] and decrease in size as they [illegible]

On the basis of damage from [illegible] the disturbed anatomic structure, including the [illegible] tissue system, tends to retain more [illegible] than the other parts of the lung. This silica, acting upon [illegible] causes this to undergo a sclerosis or scar formation [illegible] the diffuse matrix. The silica concentrated by the [illegible] be absorbed more in the mass producing the nodules [illegible]

If [illegible] infection is present [illegible] the confluence [illegible] of the confluence [illegible] and the confluence [illegible] is frequently made in animals. If one generalized [illegible] has already developed when a [illegible] infection [illegible] the lungs, then any more [illegible] the infected area. The result would be [illegible] in balance with a localized area of massive fibrosis.

In the course the infection plays [illegible] of the lesion but it then heal and [illegible] of infection. There is no active [illegible] of the bacteria that caused the condition [illegible]

The roentgenogram reveals the [illegible] of fibrosis with or without the background [illegible] nodulation. It shows the chronic adhesive fibrous [illegible] the more discrete areas of emphysema at the borders [illegible] large nodules. The fluoroscope [illegible] the lungs which are firmly bound to the chest wall [illegible]

Clinically the subject is short of breath and [illegible] severe physical exertion. He has no symptoms of the [illegible] who has accompanies an active infection. His condition [illegible] change much in repeated examinations. It is the [illegible] to differentiate this condition from some of those [illegible] which will be discussed later.

Whether the person with massive conglomerate fibrosis [illegible] susceptible to tuberculosis than others is not known. He [illegible] rarely breaks down with such infection it is [illegible] that the conglomeration was due to a latent tuberculosis and that the [illegible] case of his massive fibrosis.

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C. SILICOSIS AND TUBERCULOSIS

Silicosis and tuberculosis may coexist in a variety of forms with different clinical manifestations.

1. *The primary complex* of childhood infection usually heals and leaves small localized fibrous or calcified scars that persist throughout the remainder of life. There may be one or more such foci in the pleura in some part of the lung other than the apex and an associated involvement in the tracheobronchial lymph nodes. Since such lesions are usually acquired before the age of 20 and since they tend to heal in most persons, they are apt to be sterile and free from living tubercle bacilli by the time chronic silicosis has developed enough dust to produce visible signs of silicosis. The scar may attract and retain considerable amounts of dust for this is the usual extent of their participation. In the rare cases, particularly where they are acquired later in life and heal only partially, inhaled silica may be transported into their interior and establish contact with a few living tubercle bacilli. Under such circumstances a reactivation may occur after a variable period of time, resulting in a gradual spread of the infection and the production of a focus of chronic ulcerotuberculosis (see 3 below). Not many cases of such reactivation have been followed in roentgenograms and it is generally assumed that they rarely occur because the calcified scars of healed lesions which have not progressed are common in cases of silicosis. The prevalence is sufficiently imminent in the minds of some to warrant exclusion of young persons with such lesions from employment in a silica industry. Men over 40 or 40 years of age are presumably safe risks.

2. *The typical form of tuberculosis* on a background of silicotic nodulation is relatively uncommon. It is manifested by localized areas of chronic ulcerative tuberculosis located either in the apical or subapical portions of one or both lungs or, less often, in the lower lung. The latter distribution is more common in silicosis than in otherwise normal lungs. Such tuberculous progresses typically by direct extension and by aspiration through the bronchi to other parts of the lung. The latter results in disseminated patches of tuberculous or chronic tuberculous pneumonia. Cavities develop either in the old lesion or in the new areas of infection quite early in the course of the disease. There is almost always an associated pleurisy. The background of nodulated silicotic nodulation is usually of slight degree

[illegible] seems to have little influence upon the [illegible]

The roentgenogram shows local [illegible] tuberculous lesions in any individual usually [illegible] localized [illegible] of tuberculous pneumonia [illegible] nodulated background barely defined nodular [illegible]

There are characteristic symptoms [illegible] pulmonary tuberculosis [illegible] Such cases offer little difficulty in diagnosis.

3. *The atypical form of chronic ulcerotuberculosis* is the most common manifestation of tuberculosis in the silicotic lung. [illegible] complicated, are chronic, which do not respond to treatment and [illegible] infection. The problem is one [illegible] tuberculosis is [illegible] or inactive and whether it is [illegible] progressive [illegible] is an active disease.

Chronic tuberculosis is a very chronic [illegible] the caseous action of silica and a [illegible] the same area as the lung. [illegible] of very dense, hearth pigmented scar tissue which [illegible] portion of the lung. It slowly [illegible] expectoration of infectious material the [illegible] except as a late and terminal manifestation. [illegible] cases formation. Chronic pleurisy and [illegible] are almost always present.

Gross inspection of such a lesion may [illegible] of recent infection and the appearance [illegible] of massive fibrous development as a [illegible] infection. Microscopic examination [illegible] dense of very chronic tuberculoid [illegible] by any methods for guinea pig inoculation [illegible] On the other hand there may be definite [illegible] caseation of tubercle size. [illegible] the disease continues to develop, the infection spreads [illegible] parts of the lung by way of the bronchi and there [illegible] crops of chronic ulcerative tuberculous [illegible] The background of [illegible] well marked, [illegible] apparently [illegible] becomes [illegible]

The roentgenogram shows the localized [illegible]



tuberculous process upon a background of generalized nodulation. The localized areas simulate those caused by the healed infections but they increase very slowly in extent as serial roentgenograms are taken year after year. Cavities may be hard to detect in the dense shadow about them.

Clinically the symptoms of silico-tuberculosis are not those of an infection until the tuberculous element gains the upper hand and begins to spread into more normal lung tissue. Prior to that event the subject is short of breath and unable to perform hard physical labor. He has no fever, rapid pulse, loss of weight or appetite, etc. Tubercle bacilli are not found in the sputum except in rare instances and then it is necessary to use guinea pig inoculation or culture to detect them. In a long series of such examinations an occasional positive finding is followed by more negative ones.

These differences between the manifestations of silico-tuberculosis and of ordinary tuberculosis have led many phthisiologists like Ornstein to conclude that there is no element of tuberculosis involved. However, they are at a great disadvantage in not being able to follow men at work with such conditions over periods of years, observe them begin to manifest symptoms of infection and finally die of what anyone would regard as tuberculosis.

4. *Atypical form, acute perinodular tuberculosis* is fortunately a rare manifestation of tuberculosis, just as rare in fact as the so-called galloping consumption or phthisis florida in non-silicotic subjects. It develops, most often in comparatively young individuals with well-advanced generalized silicotic nodulation. It terminates fatally within a period of 8 to 12 months after its discovery. Its evolution has been observed in serial roentgenograms of a few human subjects but autopsy material is scarce and of course confined to its terminal stages. Experimental animals have furnished the only anatomic specimens of the earlier phases.

The disease is manifested by small localized areas of pneumonia associated with which there are thin zones of inflammatory reaction in the air spaces about each silicotic nodule in the lungs. As the disease advances the perinodular inflammation spreads and coalesces with that about adjacent nodules until large patches of caseous tuberculous pneumonia are produced. Cavities form either in the original sites of infection or in the fresher pneumonia patches.

The roentgenogram shows the ill-defined shadow of the patch of

pneumonic tuberculosis and the shadow of the [illegible] nodules whose borders have lost their definition. The [illegible] the uncomplicated nodules might be compared to those [illegible] of raw pop corn; the shadow of the perinodular [illegible] with those of kernels that had popped.

This type of tuberculosis is manifested by symptoms of a [illegible] intoxication with high fever, rapid loss of weight, etc. and [illegible] or later by the appearance of bacilli in the sputum.

One may speculate as to why the manifestations of tuberculosis are so variable in the silicotic individual. For some of them [illegible] are [illegible] but for others the reasons are far [illegible] clear. Acute perinodular tuberculosis is probably the result [illegible] first infection with the tubercle bacillus in a lung with fully developed silicosis. The native resistance to this infection has been [illegible] by the presence of the silica and there has been [illegible] ity from a previously healed infection. The [illegible] comparable to animals receiving primary inhalation [illegible] their silicosis has developed. It will be recalled that [illegible] fection ran a course which was increasingly acute as the [illegible] came more mature.

The chronic forms of tuberculosis are more difficult [illegible]. Both the typical form and the atypical silico-tuberculosis [illegible] manifestations of reinfection in a resistant and partially [illegible] munized host, in other words they are reinfection either from [illegible] genous sources or from spreads from preestablished foci of infection. The typical form usually develops in persons [illegible] is minimal in amount. Perhaps the nodulation would [illegible] visible at all had it not been for the coexistant infection which [illegible] erated its development. Not enough is known about the relationships between the time of development of the silicosis and the appearance of the infection to evaluate this factor. In some of them it appears as if an adult type of reinfection tuberculosis had developed in an individual relatively immune to tuberculosis, [illegible] had already developed an early silicosis.

The atypical silico-tuberculosis seems also to arise [illegible] a background in certain individuals, although perhaps they may have more silicosis at the time of reinfection. Other cases of [illegible] culosis result from the reactivation of preexisting but latent foci of primary infection or reinfection. The former or childhood type was

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described under the first group above. The second [illegible] caused by the subject who enters the silica industry with an imperfectly healed focus of adult type tuberculosis, usually in the top of the lung. Such lesions occur in perhaps 4 or 5% of young adult males in our city populations. In ordinary environments most of them remain healed and never spread; it is these which do that are responsible for most of the cases in our statistics. In a silicon environment the chances of reactivating such foci are much greater. Since they are most apt to develop before age of 25 years, they are much more liable to harbor living organisms in young persons than in those over 40. Hence, in the young, manifestations of apparently healed tuberculosis are a proper cause for rejection for work in a silica industry, in persons over 40 they are much more likely to be sterile and hence safe as far as the possibility of producing an active tuberculosis. Such lesions may, however, be the nucleus about which a massive conglomerate fibrosis will develop if exposed to high concentrations of silica dust for a sufficient period.

The manner in which reactivation and the development of silicotuberculosis develops is probably somewhat as follows. Inhaled silica accumulates in unusual amounts in the fibrous investment of a partially healed tuberculous focus. The silica reacts with the scar tissue and the normal connective tissues in the vicinity to produce a still heavier zone of silicotic fibrosis on the surface of the old scar. In time phagocytes transport silica particles into the interior and deposit them in contact with living tubercle bacilli. The organisms begin to multiply with renewed vigor but they cannot spread very rapidly because of the fibrosis about them. As they invade it they enter a tissue whose blood supply is very limited and consequently the oxygen tension is low and nutritive materials from the blood are scanty. These conditions would not permit the organisms to remain alive if it were not for the silica, but because of its action enough organisms continue to form to maintain activity. They are too few to detect by ordinary methods of examination. The combined action of a small number of tubercle bacilli and the silica in the lesion maintains reaction and keeps the focus metastasizing in size by a very slow peripheral expansion. Clinical symptoms of intoxication are absent because the lymphatic and blood vessels do not permit absorption of products of degeneration. For the same reasons it takes many times the usual dose of tuberculin to produce the positive reaction indicative of the tuberculous nature of the process. Bacilli

rarely escape from the focus to enter the blood stream and the organs are in the process. But eventually the time comes when a caseous area reaches the surface of the lesion and begins to soften. Most commonly this occurs in the apical part of the lung. Freed from the restraint of the dense fibrosis they multiply with increased rapidity, and the terminal picture is that of chronic ulcerative tuberculosis, with its usual symptoms of cough, expectoration and hemoptysis. Cavity formation may destroy much of the original area of fibrous tuberculous fibrosis and the process dies of the tuberculosis. In less fair outcome mechanisms are responsible for the slow development of the disease has never been evaluated. The *maintenance* of a very chronic, subclinical type of infection by the stimulation of the development of connective

REACTIONS TO MIXTURES OF SILICA AND OTHER INORGANIC DUSTS

The type of the reaction varies with the relative [illegible] silica and the adulterant dusts. The higher the percentage of the more nearly do the lesions approach those produced by pure silica. The greater the amount of non-siliceous or silicate admixture, the more bland (peribronchitic) is the reaction. Usually both types of tissue change are present.

The nodular focus is, in structure, [illegible] of a well defined patches of cellular connective tissue between nodules of hyaline fibers. In the adulterant dusts, the connective tissue may be dense and hyaline from the beginning. There may be a small pale central core of [illegible] connective tissue filled with the non-siliceous dust. The hyaline zone may form a thin investment about larger nodules. Septa of hyaline connective radiate from its surface into the general pulmonary tissues. As a general rule the margin of the siliceous nodule of pure silica fibrosis is much less sharply defined than that of the mixed dust type and it is pronounced if the adulterant dust has little silica and it is pronounced if the adulterant dust has little silica.

It is not impossible that in the presence of a [illegible] some of the other dusts may accelerate the deposition of connective tissue cells. Certain inorganic dusts [illegible] fibrous zone of the nodules and the hyaline connective tissue septa between them may in time fill in and replace most of the nodular tissue with pigmented cellular connective tissue. Such a process [illegible] possible for the so-called massive conglomerate silicosis

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lesions of this type are especially common in [illegible] Conducted experiments such as those involved in mining [illegible] certain degree of importance this hypothesis deserves consideration. It may be the cause of some such lesions with or without the focus of healed infection.

In roentgenograms it is not often possible to differentiate between the shadows of nodules produced by pure silica and those caused by mixtures of silica and other dusts. The recognition of the nodular shadows themselves gives trouble enough to-day but the time may come when careful comparisons will enable the experienced observer to determine them. The frequent occurrence of associated, massive, conglomerate fibrosis in some industries makes one suspect the nature of the occupational exposure. Such, for example, are the dust of granite cutters, anthracite miners and some of the foundry group.

The susceptibility to tuberculosis is probably as great in cases of modified silicosis as it is in the pure forms but there seems to be a greater tendency for the infection to be healed again after some degree of extension has occurred. This was true of tuberculous guinea pigs exposed either to ferruginous chert or to artificial mixtures of silica and gypsum. Clinical and autopsy study of hematite miners indicates a high frequence of healed tuberculosis. Whether the same holds true for all forms of modified silicosis awaits demonstration.

ASBESTOSIS

For the sake of completeness this subject will be given brief consideration but there is not enough information on the condition to present a full discussion.

Early cases rarely come to post mortem examination but in the few that have been observed one finds the same collars of fibrous tissue about the terminal bronchioles that were described for experimental animals. Many of the advanced cases were complicated by obvious infection which produced a picture too complex to analyse satisfactorily. In others there was a question as to whether all of the fibrosis encountered had been produced by dust alone or whether there was an element of healed infection.

The advanced cases present a considerable variety of gross appearances, one of the reasons for suspecting a variable complicating

factor such as infection. Most of the cases [illegible] fibrosis, usually associated with some [illegible] modified silicosis. The outstanding [illegible] gross appearance gave little or no [illegible] the pleura or enclosing [illegible] tissue. In the fibrous [illegible] there are [illegible] millimeters in diameter [illegible] In other cases the pleura [illegible] apparently thickened but they are [illegible] The latter is more prominent in the regions of the [illegible] deposit along the branching walls of the [illegible] [illegible] pronounced. The vascular and bronchial trunks [illegible] visibly thickened. The lymph nodes are generally [illegible] pigmented.

The microscope reveals large differences [illegible] seem to have no general pattern or relation [illegible] or lymphatics. An attempt to prepare [illegible] the relationship between the patches of fibrosis and the [illegible] abandoned after studying serial sections because [illegible] lack of any form to the lesion. The fibrous [illegible] [illegible] the air spaces in considerable [illegible] of the lung. All the spaces are not filled, however, for [illegible] a considerable number that are still patent. The [illegible] blood vessels in their walls are compressed. Occasional [illegible] bodies are numerous in some cases they are [illegible] done both in the scar tissue and in the patent air spaces. The [illegible] bronchial lymph nodes and intrapulmonary lymphoid tissue [illegible] considerable black particulate matter but only where [illegible] a lesion is to be detected. There seems to be [illegible] inflammation for these fibrosis found only in the presence of infection.

The roentgenograms characterized by a diffuse [illegible] called referred to as a ground glass appearance of [illegible] of the lower third of the lungs. Anatomically [illegible] dust and reaction to it in the upper lungs as the [illegible] flat roentgenogram would indicate more extensive [illegible] Dr. Simpson has called attention to the fact that [illegible] of the film one has projected a much greater thickness [illegible] with fine patches of fibrosis than on the upper. The [illegible]

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shadow cast by the fibrosis of asbestosis [illegible] the fact that everywhere throughout it are numerous [illegible] attended with it.

There is some debate as to the influence of asbestosis upon susceptibility to tuberculosis. Merewether reports that 35 %, of his English cases of asbestosis that have died and come to autopsy, show complicating tuberculosis. In this country there have not been enough post mortem examinations for statistical analysis, but Lanza's success of having persons working in the asbestos industry have failed to reveal any excess of active tuberculosis. Animal experiments incline the speaker to feel that tuberculosis tends to heal in the presence of asbestosis and that in consequence some of the more extensive areas of scar formation seen in so-called simple asbestosis may be of tuberculous origin. Other infections may behave in a similar fashion.

F. SILICOSIS COMPLICATIONS IN OTHER ORGANS

Simple Silicosis, Discrete Type.—The pleura is slightly thickened over every nodule that impinges upon its surface but no adhesions have been observed.

The heart is probably not involved. The cases that have been reported are more likely to be independent of the pulmonary condition but of increased frequence because of the age group that is involved.

The kidneys are never involved in silicosis.

The abdominal lymph nodes frequently show evidence of specific silicotic fibrosis. The hepatic node, lying along the common bile duct and other structures in the anterior wall of the foramen of Winslow, the esophageal nodes and perhaps a group along the lesser curvature of the stomach are the most common ones. In certain cases there may be a whole chain of nodes on either side of the abdominal aorta, down to a point 5 or 6 inches above the bifurication.

Retrosternal lymph nodes and other smaller nodes and lymph vessels throughout the parietal pleura are not infrequently pigmented and fibrous.

The liver and spleen in rare cases may show occasional silicotic nodules as large as 1.5 mm. in diameter. Perhaps associated infection in the lungs is partially responsible for this dispersion and relocalization of the silica from the respiratory tract.

F. SIMPLE COMPLICATED SILICOSIS [illegible] SILICOTUBERCULOSIS

The pleura is thickened and fibrous with adhesions to the visceral wall of the pulmonary lesions extend to the surface.

The lungs show a secondary hypertrophic emphysema [illegible] The evidence is conflicting and [illegible] the Chinese [illegible] is undependent [illegible]

Other organs are involved as in simple silicosis [illegible] but with greater frequency because of the infection [illegible] more deep [illegible]

[illegible] the infectious nodules [illegible] the [illegible] because the [illegible] Because they do not enter the blood stream [illegible] lymphatic and intestinal [illegible] complications.

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Discussion of Dr. Gardner's Paper

Dr. Gardner reported that he had been informed that Dr. McCord had finished some most interesting experiments on the action of ethyl silicate, a substance which was being used in the manufacture of paints and varnishes. He asked Dr. McCord if he would outline his results to the group.

Dr. McCord's summary:

Ethyl silicate, (Tetra ethyl ortho silicate) an organic compound of silicon was found from animal experiments to possess highly injurious properties bearing no proved relationship to the action of silica, (silicon dioxide), in the causation of silicosis.

In these experiments 218 small animals (rabbits, guinea pigs, rats), were subjected to the action of ethyl silicate in various amounts and at various intervals administered intravenously, intraperitoneally, subcutaneously, orally, and to a limited extent through inhalation and skin contact.

The minimum lethal dose per kilo for rats is approximately 0.16 ml. per 100 grams of body weight. When administered intraperitoneally, death takes place within three days. When introduced intravenously into rabbits, the minimum lethal dose is approximately 0.21 ml. per 100 grams of body weight, death usually takes place within one hour.

Regardless of the method of introduction, the chief action of ethyl silicate is centered in the pulmonary tract. Rupture of terminal capillaries is accompanied by pulmonary hemorrhage which fact stands in relation to all other observations thus far made, such as anemia, hematuria, secondary pneumonia, air emboli, etc.

Pulmonary hemorrhage associated with ruptured capillaries may be demonstrated within ten minutes after intraperitoneal injection and in the absence of proved inflammatory action within the peritoneal cavity.

The brains of rabbits dying with profound convulsive seizures within a few minutes after injection, have, whenever examined, presented air emboli. It appears tenable that such air emboli find origin through aspiration of air from the ruptured air sacs and ruptured [illegible] constituting the foremost injury from ethyl silicate.

Although ethyl silicate possesses [illegible] proved [illegible] of a type characteristic of silica, the substance in animal experiments has demonstrated a high degree of toxicity of [illegible] nature.

Dr. Gardner commented that Dr. McCord's animals [illegible] symptoms which reminded him of those produced by intravenous injection of colloidal silica. He had confirmed Dr. McCord's observation that the metal silicon is [illegible] inert and [illegible] reaction in the tissues.

In reply to the question as to whether intravenous injection of dust would produce the same effect as inhalation, Dr. Gardner stated that the intravenous test was a method for eliminating the inert types of dust which were incapable of provoking a [illegible] reaction. For such purposes the method was apparently [illegible] though it was still debatable whether it could be applied to [illegible] the particulate dusts. The lack of reaction [illegible] asbestos still necessitated such reservations [illegible] dust intravenous injection seemed to duplicate the [illegible] reaction produced by inhalation. The arrangement of [illegible] actions and their relationships to normal structure [illegible] different because of the varying anatomy [illegible] the organs involved. At the present time there was nothing to indicate that intravenous injection did not constitute a satisfactory method for evaluating the effect of particulate inorganic dust particles upon the living tissue.

Dr. Gardner was asked his opinion of the relative value of chemical and petrographic analysis of gross tissue and of [illegible] incineration of sections. He replied that he believed [illegible] whether there was an appreciable amount of silica in the tissue but that he himself relied most on the gross and [illegible] appearances of the tissues. A high content of silica in the lungs could not warrant a diagnosis of silicosis if the tissue changes characteristic of that condition were absent. The chemical analysis might [illegible] including if the tissue was not properly selected. Generous portions of the lungs from diseased areas should be employed. The lymph nodes, if they were analysed, should be treated separately. Low values would obviously be obtained if only the [illegible] marginal portions of the lung were given the chemist, [illegible] high results would be produced by including lymph nodes.

In the Saranac Laboratory all lungs submitted for study were

PLAINTIFF'S EXHIBIT
PENGAD-Bayonne, N. J.

ically and histologically that a correlation between the amount and type of dust present and the character of the disease might be observed. Perhaps such a study would show only that in exceptional cases involving a history of exposure to an unusually intense dust. Perhaps more such cases would have been made if the field had confidence in its own ability to identify particles by petrographic methods. Since Dr. Fairhall's presentation, he advocated all such complex or unrecognized industrial dusts. Professor Larsen of Harvard and Dr. Eckhardt of the Bureau of Mines. Dr. Gardner then revealed a table of particles in sections of tissue. He said he was now using it only on occasional cases. Identification of particles here again depended upon more knowledge of petrography than he professed. Since the experts in the field he cited were prepared to make definite opinions on the nature of very fine dust particles, he would be presumptuous for the type to do so.

It was asked whether the caustic hydrochloric and chromic acid mixtures used in preparation of histologic sections would interfere with the petrologic identification of dust particles. The pathologist replied that he had no idea. Particles, he felt he believed, they did not. Only the resistant mineral particles in inhaled dust were concerned and theoretically they would not be attacked by a tissue reagent. He formaldehyde mixture included acid. The heat used to concentrate a section would evaporate it. Electron films of such substances absorbed on the surfaces of the particles so that their appearance in polarized light would be affected. Heating at too high a temperature would alter the hydrous substances, a possible source of error in the identification of mineral particles.

In reply to a question as to the value of x-ray diffraction analysis of mineral particles in the sputum of persons being exposed to dust, Dr. Gardner stated that he believed it possible to identify such particles with considerable accuracy. He recalled, however, that in exceptional cases which Dr. Fairhall had had analysed, the petrographer had failed to report results consistent with the reported exposure of the individual. He also mentioned the possibility that selective inhalation and retention of the different components of an inhaled dust might influence the relative number of the different elements in the sputum.

Dr. Gardner was asked whether he believed that silica was a poison. He replied that it undoubtedly produced toxic effects in

the tissue which were proportional to [illegible] stroyed. He stimulated the cell which [illegible] there was a concentration of cells [illegible] necrosis death. Dispersed cells [illegible] a poison like potassium cyanide or [illegible] cell activity when ground very finely and [illegible] concentrated in localized areas [illegible] dissolve through the body [illegible] observable evidence of injury in [illegible] of particles. Animals [illegible] internal evidence of their [illegible] the experiments mortal. They [illegible] of their disease but rather from [illegible] the tissue unduly susceptible, or from [illegible]

Incidentally, it was mentioned [illegible] accepted chronic passive congestion [illegible] of heavy human cases of silicosis. Dr. [illegible] in guinea pigs resulted from exposures to [illegible] concentrations of pure silica which [illegible] type of fibrosis in the lungs. The [illegible] walls of the capillary bed of the [illegible] tissue might in turn affect the [illegible]

Dr. Gardner concluded by [illegible] that he would [illegible] a person but that the concentration [illegible] the usual case of silicosis were [illegible] silica. If one could accept the [illegible] would offer no difficulty.

Dr. Lanza asked whether the [illegible] unmasked action of silica could be [illegible] acid silicic nodes or larger [illegible] time. He stated as his own belief [illegible] of individual nodules and that [illegible] early areas of necrosis due [illegible] of blood supply). All other [illegible] soon.

Dr. Gardner concurred in [illegible] and replied [illegible] sections with illustrations. [illegible] in the interior of individual [illegible] ended and replaced by fibrous [illegible]

PLAINTIFF'S EXHIBIT 1577 (63)
SYNCRO-Ravenna, N. J.

[illegible] tuberculous infection. He [illegible] a common [illegible] silica and [illegible] Cochrane [illegible] almost pure [illegible] Many cases [illegible] form after such [illegible] the material [illegible] and become [illegible] the surface of the [illegible] be evacuated. Larger areas of degeneration were seen in the centres of areas of more fibrosis, particularly in coal miners and in certain magnesite miners. Such lesions responded [illegible] to roentgenograms because of the density of the surrounding tissues. He pointed out that such areas did not have definite walls like a tuberculous cavity, they consisted of elongated, irregular shaped pieces of softened tissue in the heavily pigmented dense connective tissue. Such areas had no definite wall like a tuberculous cavity but the softened tissue blended imperceptibly with the unmodified fibrosis about it.

In commenting upon the protective action of hematite against silica Mr. Cummings reiterated that the Ishpeming group had been examined 6800 iron miners during a period of 5 years and that the incidence of nodular fibrosis detected by roentgenogram was 30%. This approximated the figure obtained in other large groups of men exposed to silica had been studied. He inferred that the men with the silica had apparently not prevented the development of the unexpected number of cases. He was much more impressed with the fact that the incidence of infection, chiefly tuberculous, was so much lower in this group of silicosis subjects than in other localities such as Barre and Picher. In the hematite miners the mortality from tuberculosis was only twice that of the industrial male population for the same age period while in the other localities it was much greater. It was his opinion that the protective action of hematite manifested itself chiefly in reducing the frequence of progressive tuberculous lesions, commonly associated with silicosis.

Dr. Gardner admitted that Mr. Cummings' statements constituted conclusive proof that men who had chronic silicosis when exposed to exposure of hematite dust only. He wondered, however, how the results on animals could be reconciled with these observations. The rats were exposed for much longer periods of time than the animals and this might explain the apparent discrepancy. On the other hand, many of the men had been employed in development work from time to time before the preventive measures were in force, in such exposures their exposures had been to much purer silica dust. Since

[illegible] occupational history was at best [illegible] the [illegible] the [illegible] were [illegible] to doubt the significance [illegible] [illegible] [illegible] [illegible] [illegible] protection disclosed by [illegible] [illegible] [illegible] [illegible] [illegible]

...would be at the same moment more. The ... was almost equal to that in animals exposed to quartz dust for one year; the number of nodules was less.

In reply to a question as to whether it would be useful to allow a man to continue in his occupation after silicosis had developed, the speaker stated that his decision would be governed by the age of the workman and the conditions under which he was working. If the man in question were young and if the dust exposure could be greatly reduced by the simple precautions mentioned, he should be placed in some other department of the plant. If the man were an old employee who would have difficulty in learning a new trade, he could undoubtedly continue at his previous work. Presumably the employer who was sufficiently interested to discover the good men of his workmen would take precautions to make certain that the dust hazard was eliminated or at least reduced.

Dr. Bell was asked whether there was any evidence to prove that the inhalation of non-siliceous dusts, which interfere neither with pulmonary ventilation nor with the function of the lymphatic system, affected susceptibility of the lungs to subsequent non-tuberculous infection.

Dr. Gardner replied that to his knowledge there was no proof of such action. In some groups an increased incidence of bronchitis had been reported but this was not true of the cement or gypsum workers examined by the Saranac Laboratory. Five autopsies had been obtained on members of such groups; three of these had been of men who were never often from the selected individuals who had happened to die of pulmonary infection. He mentioned two cement-mill workers who died of tuberculosis. Their lungs showed such extensive manifestations of infection that it was hard to determine how much of the reaction was due to the dust. Incidentally, he mentioned that in the lungs of these cases there were large numbers of atypical asbestosis bodies, a finding not surprising in view of the fibrous nature of this mineral and its chemical composition ($CaMg_3Si_4O_{12}$). He also stated that experiments were now in progress to determine whether some of the silicates, which acting in normal tissues were inert, might be capable of producing more reaction in the presence of tuberculous infection.

Some observers had maintained that the incidence of unresolved pneumonia was greater in individuals exposed to dust of any type

than in the general population. Proof of this statement was hard to...

Cummings pointed out that the ... from ... in laboratory workers was ... rest of the ... population in the same locality. Dr. Lynch inquired how many had come to autopsy and Dr. Cummings replied that there were several. Dr. Lynch then stated that he felt that ... caution should be exercised in making any elaboration about ... pneumonia" for the term included a variety of infections, from acute influenza to lobar pneumonia. He had had frequent experience in the accuracy of clinical diagnosis. Partly because ... and partly because of the speed by the different types of pneumococci, he could make such correction and he cited the different periods ... by the pneumococci of types, 1, 2 and 3. He felt that a statistical analysis of "pneumonia" was, therefore, fraught with ... were applied to a specific background of ...

Dr. Lynch said that his experience ... tions had impressed him in two respects: (1) that there ... occurred in silicotic lungs in which there was still a large ... of alveoli functioning visible. The ... of the alveolar ... such that it must have normal lung in ... alveolar ducts; (2) that the absence of lymphatic obstruction ... of silicosis was ... complete but that none of the lymphatics could be forced ... and distended by pneumonic exudate despite the state of ... Dr. Gardner concurred in these observations.

It was asked whether tool wood ... provide ... have and whether, like asbestos, it should be classed as a ... dust. In reply Dr. Gardner mentioned that ... experimental experiments had failed, with one exception, to ... evidence of irritation by this substance. When injected ... into rabbits, rock wood did not cause progressive ... kind and it did not cause fibrosis. Subcutaneous and other ... of injection had been similarly inert. Injection of large ... rather mixed with the food or by stomach tube, had failed to ... gross or microscopic changes in the alveolar tissue. The ... also result occurred when the Sayers-Miller compound was ... tempered. Most of the animals had also been fed ... portion. Since similar results follow the injection of pure ... calcium silicate he was inclined to believe that the results were ...

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wool were produced by similarities in chemical [illegible] any such result, of course, could never be experienced by human beings handling glass wool.

The speaker did not feel that the crumbly particles of wool used for experimentation acted because of their fibrous structure. For all important purposes the particles were 3-micron or less in length. The wool, while it had a gross fibrous structure could not be broken down into finer and finer fibrils by grinding as was the case with asbestos.

Asked whether it would be advised in commercial use in appreciable quantities he replied that he believed that most of it was too coarse and that the finer particles would be *subconsciously swallowed* into the upper respiratory tract so that they would be coughed out and expectorated before many of them reached the lungs. Those which did penetrate the lungs would be incapable of producing fibrosis from the evidence afforded by the injection experiments.

It was then asked whether the speaker actually believed that the effect of asbestos upon the lung was due to the fibrous structure of this substance. He replied that he was uncertain but that his animal experiments had thus far failed to indicate that either a bestos, if ground sufficiently fine, or serpentine particles which had the same chemical composition were capable of producing fibrosis in organs other than the lungs. The experiments were not yet completed and even when they had been the evidence might not be conclusive but he believed that it now suggested a mechanical type of irritation which acted specifically upon the lungs. He pointed out that the control still lacking was the production of fibrosis in the lungs by some fibre similar to asbestos but of *entirely different chemical composition*. He had thought of some cotton fibres but they were not sufficiently divisible into smaller units, each having the same fibrous structure. *Furthermore*, the roentgenologic studies on cotton operatives with which he was familiar had failed to disclose evidence of pulmonary reaction to this dust. He recalled that a few cases of silicosis had been reported among such workers but he was inclined to accept the explanation of the authors of the reports that such nodular fibrosis was due to appreciable *quantities* of silica which had adhered to the cotton.

Dr. Hogeland stated that he had recently heard, with some scepticism, a series of papers on the reaction to inhaled cotton dust but

[illegible] that Dr. Vorhaus had just shown him some [illegible] specimens which revealed what appeared to be [illegible]

Someone asked how *frequently* [illegible] bodies [illegible] Dr. Lanza their detection in the sputum was [illegible] In answer to [illegible] large or small numbers unless [illegible] fortuitous process. Often concentration of the sputum also [illegible] with antiformin was necessary [illegible] associated with a history of exposure to [illegible] roentgenographic evidence, the presence of an appreciable number in the sputum would be *sufficient* to establish a diagnosis. The [illegible] one or two in routine examination of [illegible] [illegible] prepare [illegible] but such a confirmatory [illegible] obtained one would hesitate to make a diagnosis [illegible] There were so many instances on record of accidental [illegible] occasional bodies in routine examination [illegible] sections that under rare circumstances [illegible] the sputum. Dr. Lanza related the story of their detection in the sputum of a *chemist* whose only contact with [illegible] was in the preparation of the mats for Gooch crucibles. He had no [illegible]

PLAINTIFF'S EXHIBIT [illegible]

PHYSIOLOGY OF THE FIBROTIC LUNG

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The mechanisms by means of which the respiratory [illegible] effected is tremendously complex. The lungs together with the right heart may be likened to a tonometer in which the blood in the capillary bed of the lesser circulation is equilibrated with the alveolar air. Normal function of the respiratory mechanism tends to maintain the blood leaving the pulmonary capillaries at a relatively constant composition, as regards oxygen, carbon dioxide and hydrogen ion concentration, in spite of tremendous variations in the extent of the internal respiration of the tissue cells.

The mechanism by means of which a steady state is established in the tonometer involves coordination of the circulatory and ventilatory minute volumes with the utmost precision. This coordination is accomplished principally through the mediation of the nervous system rather than through changes in the composition of the blood, though marked changes in the latter may modify the reactivity of the nervous mechanism.

The circulation through the lung is very largely influenced by ventilatory activity. The normally negative pressure which exists in the pleurae is a great factor in the return of venous blood to the right heart, and we know that the volume of the venous return is the principal determining factor in ventricular output.

The regulatory mechanism governing respiratory movement has recently received a very great deal of attention. Over a century ago Legallois showed that section of the vagi resulted in a slower and deeper respiration. This was followed by the discovery of the Hering Breuer reflexes. If action currents in the vagus nerves are studied it has been shown that showers of impulses pass up the vagi during inspiration reaching a maximum frequency when the distension of the lungs is greatest, and a minimum frequency when the deflation is greatest. The accumulation of stimuli on vagus receptors in the lungs as they become distended results in a reflex deflation. When deflation is complete the inspiratory cycle begins again.

Efferent impulses which may be detected in the phrenic nerve occur in rhythmic showers representing periodic discharges from

higher respiratory centers. The [illegible] after section of the vagi. The character of the breathing [illegible] be greatly modified depending [illegible] which phase of [illegible] the higher centers the showers of afferent vagal impulses [illegible]. If the maximum vagal effect falls in the active phase of the [illegible] center the rate of breathing is accelerated and the depth [illegible], and conversely if it falls in the inactive phase the respiration becomes slower and deeper.

The activity of the respiratory center may be greatly [illegible] by many extra-pulmonary influences: afferent stimuli [illegible] from skin, from muscles, from [illegible] and larynx, from the carotid sinus and aorta, and from the emotional and higher psychic centers. The [illegible] which these stimuli affect the respiratory center is largely determined by the chemical conditions in the blood. [illegible] the effect [illegible] the carotid sinus is heightened by oxygen want or by CO_2 excess. The excitation [illegible] of a peripheral nerve is [illegible] total of effects of CO_2 excess [illegible] CO_2 lack to shallower respiration approaching apnea.

As to oxygen relatively large changes must occur in [illegible] pressure before the oxygen saturation [illegible] affected. In severe anoxia the tendency is toward an acceleration of respiration.

[illegible] we come to consider the character of the tonometer [illegible] with which the blood in the pulmonary capillaries [illegible] the alveolar air. This air lies in the deeper [illegible] bronchial system. Its volume is an undetermined moiety [illegible] capacity of the lungs. By "mid-capacity" we mean the functional residual air, the volume of air remaining in the lungs at the end of a normal expiration. This is the air with which the [illegible] of a new inspiration must mix.

The tidal air and the rate of breathing determine the respiratory minute volume. Its magnitude is determined by the mechanism regulating ventilatory activity and the demands of the internal respiration of the tissues.

The vital capacity is the limiting or maximal value of the tidal air. As the respiration increases during work the [illegible] and the tidal air is enlarged by increasing use of the [illegible] air. The maximum effective ventilation of the lungs [illegible]

PLAINTIFF'S EXHIBIT
1525 (4)

accumulated in a rubber bag which is [illegible] and expired with the aid of [illegible] per minute. These values are calculated in terms of 20 normal inspirations corrected to the base of their capacity. The vital capacity is an important index of the maximal ventilatory capacity, even though it is a purely artificial value.

The residual air which remains in the lungs after a forced expiration is likewise a purely artificial value which has considerable functional significance under pathological conditions. In normal men it has a value of about 22 per cent of the total pulmonary capacity, the vital capacity making up the other 78 per cent. Slightly different proportions are observed in women. The residual air at the end of a forced expiration probably consists entirely of alveolar air. The difference between the functional residual air or end-capacity and the ultimate residual air is known as the [illegible].

The capillary bed of the lung. Estimates have been made that this extends over a surface of 140 square meters according to Hofmeier, but only 90 square meters by Zuntz, who estimates [illegible] square meters alveolar. At rest the capillary bed of the lungs probably contains 6.9 per cent of the total blood volume, 6 per cent at inspiration and 9 per cent at expiration.

The right ventricular output of normal individuals at rest may be measured by the Grollman acetylene method, and by various methods the velocity of circulation time through the lungs may be estimated. It is very difficult to secure unquestionable data regarding blood flow during effort, or under pathological conditions, or to make the correlations between the velocity, volume per minute, and capacity of the capillary bed of the lesser circulation which would be accounted as a good index of the soundness of its mechanics.

There is considerable evidence that during rest many of the pulmonary capillaries are inactive, and that the capacity of the capillary bed is capable of considerable expansion during increased functional demand.

Much doubt exists as to the role that vasomotor nerves play in regulating pulmonary blood flow, though it seems certain that a vasomotor mechanism exists.

In fibrosis of the lungs, such as that which occurs in pneumonoconiosis, a very incomplete picture of the disturbance of respiratory function is available. The pulmonary capacity is altered as measurements have shown. A patient with [illegible] and cyanosis [illegible] a decrease in vital capacity, as measured by the spirometer, and a marked decrease in total capacity. These observations have been found to be more marked in the more advanced cases of fibrosis. In those cases in which dense areas of fibrosis are accompanied by considerable emphysema considerable increase in residual air occurs.

In the previous discussion the functional significance of the vital capacity was referred to as an index of the maximal capacity of the lungs. In fibrosis, as we shall see later, the reduced vital capacity is accompanied by diminished maximal ventilatory capacity.

The significance of the increased residual air on the arterial blood is not to any effect upon ventilatory capacity, but upon the efficiency of the mechanism as an equilibrator for the maintenance of normal saturation of the hemoglobin with oxygen, and the regulation of the CO_2 content of the blood. When the residual air reaches the proportion of 50 per cent of the total [illegible] changes in the gas content of arterial blood [illegible] oxygen saturation falls below 94 per cent, the CO_2 [illegible] and this tends to be compensated for as a rule by an increase in the reserve of the plasma (Fig. 1).

It is very difficult to determine by experimental means the exact effect which pulmonary fibrosis produces upon the capillary bed. It seems reasonable to suppose that the [illegible] of the bed would be diminished in a manner [illegible] which in many cases [illegible]. Moreover, the [illegible] become greatly enlarged [illegible]. Both of these processes would favor the separation of [illegible] capillary capacity, since in one case [illegible] connective tissue, and in the other case they are [illegible] much smaller than normal relative to the volume of [illegible] dilated alveoli.

If these assumptions are correct, then it follows that as the capillary bed grows smaller it will require a smaller output of the [illegible] to fill the bed to the limit. The volume of [illegible]

In cases of fibrosis the smaller vessels of the pulmonary [illegible] the heart would encounter an increased peripheral resistance. [illegible] would come a stage at which the right ventricle would [illegible] to hypertrophy, the beginning of the [illegible].

Since there is normally a large margin of reserve in the [illegible] of

PLAINTIFF'S EXHIBIT 1305 (a)

valves in this capillary bed one would expect a considerable shunt, we might never before metrazol peripheral resistance would develop across the output of the right ventricle decreases. One criterion, however, of importance would seem to prove equally as soon as the increased pressure output begins to tax the capacity of the capillary bed.

Cobbett, Hopkinson, and Churchill occluded the pulmonary artery gradually with a clamp. They found no fall in arterial pressure or rise in venous pressure recorded until the occlusion amounted to 60 per cent of the cross sectional area of the pulmonary artery. A reduction in cardiac output accompanied by a fall in arterial and a rise in venous pressure occurred when the occlusion equalled 60-85 per cent.

Moore and Binger injected potato starch into the pulmonary arteries of animals. When enough of the capillaries and arterioles had been obliterated they were able to demonstrate dilatation of the pulmonary artery and of the right auricle and ventricle by x-ray and by direct inspection through a thoracic window.

Moore and Binger estimate that the left pulmonary artery supplies about 40 per cent of the capillary bed and the right pulmonary artery 60 per cent. When the left artery alone was ligated effects upon the right heart were not apparent. The right heart was affected by ligation of the right pulmonary artery, so that we can conclude that a reduction in the capillary bed of 50-60 per cent is certain to result in a *cor pulmonale*.

We have reviewed the electrocardiographic findings in 60 cases of pulmonary fibrosis and emphysema of various types, but largely composed of pneumonoconioses and tuberculoses. In a group of 28 cases with nodular fibrosis left axis deviation was found in 7 (25%) and right axis deviation in one case.

In another group of 16 cases of subacute pneumonoconiosis right axis deviation was found in 6 and left deviation in 3. The pulmonary capacity values and oxygen saturation of the arterial blood of the six cases showing right axis deviation are given in the following table:

Table I

Name	Total capacity percent of normal	Vital capacity P.A.T.C. percent	O₂ saturation arterial blood percent
Per	58	30	87
Wa	42	63	80
Fra	100	69	99
Bal	86	65	84
Wh	76	57	87
Ge	69	61	—
Average	73	62	82

These figures will give an idea of the degree of alteration of the pulmonary capacity associated with cor pulmonale.

These findings may be compared with those of 10 cases of fibrosis in which the electrocardiogram showed no evidence of cor pulmonale, given in the following table.

Table II

Data concerning 9 cases of pulmonary fibrosis, not complicated without right axis deviation in electrocardiogram.

Name	Total capacity percent of normal	Vital capacity P.A.T.C. percent	O₂ saturation arterial blood percent
	85	39	96
H. H.	115	49	97
A. F.	85	53	93
D. V.	84	50	
D. E.	61	45	87
H. B.	71	24	97
J. S.	85	47	99
F. S.	89	58	93
I. F.	59	53	8
Average	81	46	9

PLAINTIFF'S EXHIBIT 123 (60)

Thus we see that right axis deviation has not been observed in any case in which the residual air is less than 40 per cent of the total capacity or the arterial saturation more than 90 per cent. It should be borne in mind, however, that hypertrophy of the right ventricle may occur in pulmonary fibrosis and emphysema *without* a right preponderance in the electrocardiogram, owing to concomitant changes in the left ventricle or to changes in posit[illegible]

As one would suppose from the low val[illegible] of [illegible] saturation *with oxygen*, cyanosis was the rule in all patients [illegible] during exhalation of [illegible] gas. However, polycythemia was relatively uncommon. Kaltreider, Hurtado, and Boothby determined the blood volume in 25 normal men, 16 cases of emphysema and 14 cases of pulmonary fibrosis. In 3 of the 14 cases of fibrosis there were marked increases in total blood volume, cell volume, and hemoglobin. The other 11 cases gave normal values.

The effects of pulmonary fibrosis on the pleura and on the venous pressure are important. The fibrotic lung tends to increased rigidity and lessened elasticity. Pleural adhesions are frequent. As emphysema develops all three of these factors tend to diminish the negative pressure in the pleura which plays such an important part in the return of venous blood to the right heart.

By direct measurement of the venous pressure in normals, values are observed ranging from 40-110 mm. H_2O with an average of 60 mm. H_2O.

The venous pressure does not depend entirely on the intrapleural pressure, and one may observe cases of advanced pulmonary fibrosis in which venous pressure is within normal limits. It is usually somewhat elevated with values of 80-90 mm. H_2O. However, when the right ventricle begins to fail in a patient with cor pulmonale pronounced elevations of venous pressure are observed (above 110 mm. H_2O).

The effect of pulmonary fibrosis on the movements of the thorax has been studied in roentgenographs. The position of the diaphragm as measured by the height of the pulmonary hilus is not greatly altered, except when an extraordinary degree of emphysema exists. The diaphragmatic excursion, measured on a doubly exposed film which records both maximum inspiration and expiration, is appreciably less. The normal excursion was 6.0 cm., and the average [illegible] for 28 cases of well marked pulmonary fibrosis was 1.3 cm.

A corresponding degree of impairment was noted in the angle of rotation of the 4th rib, the average angle of rotation for normals being 21 degrees and for cases of fibrosis 15 degrees.

If films are exposed at the same distance, one at maximum inspiration made at maximum inspiration, and one at maximum expiration, the pulmonary areas may be measured by tracing their outline with a rotating wheel planimeter. The comparison of these two areas affords a means of studying the expansion of the chest quantitatively.

In normal individuals the ratio of $\frac{\text{area at maximum expiration}}{\text{area at maximum inspiration}}$ gives a value of 62-71. Values of the ratio which are higher than 71 indicate diminished power of expansion. The importance of the measurement is less in cases of pulmonary fibrosis than it is in those with pulmonary emphysema.

When individuals with pulmonary fibrosis perform muscular work the respiratory rate, tidal volume and minute volume ventilation are greater than those of normal individuals performing the same work. This is true whether the volume of breathing is referred to the surface area or to the vital capacity or to the percentage of maximal ventilatory capacity. In this respect their behavior is quite similar to that observed by Harrison in cardiac patients. As in the case of heart disease the augmented breathing in cases of pulmonary fibrosis is attributable to a heightened sensitivity of the Hering-Breuer reflexes rather than to changes in the gas content of the blood, since they occur in early cases of fibrosis before the blood gases are altered.

Dyspnea is the consciousness of effort in breathing. It occurs in normal individuals during strenuous muscular exercise. In diseases of the heart and lungs it occurs during lesser degrees of effort than in normals, and it may occur at rest. This is also true of severe anemias and of acidosis unless consciousness is dulled by [illegible] or uremic coma.

Peabody found that the maximal ventilation of normal men was 9-10 times the resting value. Normal individuals usually were conscious of breathing when the ventilation reached 12-15 times the resting value, and definite dyspnea was generally reached when the ventilation was increased 4-fold or about 70 per cent of the maximal capacity. In our experience in the study of patients with [illegible] and from the experiments of Harrison these appear to be [illegible]

PLAINTIFF'S EXHIBIT 15C (11)

same relation of dyspnea to the fraction of the total ventilatory capacity in use.

The respiratory reserve at any given moment is the difference between the maximal ventilation and that in use. Normal individuals experience mild dyspnea when the reserve is 50-60 per cent. This also holds true for those with heart disease, pulmonary emphysema and fibrosis.

The maximal ventilatory capacity [illegible] dicted from the vital capacity by the formula:

$$\frac{42 \times \text{vital capacity} \quad 37}{100} \quad \text{liters per min.}$$

Individuals with pulmonary fibrosis have a lowered vital capacity and correspondingly a more limited maximal ventilatory capacity. At the same time, owing to the disturbance of the Hering-Breuer reflexes, they hyperventilate as compared with normals performing the same amount of work. In consequence dyspnea develops on less effort than in normals. This is the basis of the exercise tests for disability in pneumonoconiosis which the writers have employed.

In carrying out these tests they have used an ergometer which is shown in Fig. 2. This ergometer is mounted on a heavy base. A shaft driven by a chain and sprocket is mounted on excellent bearings. A heavy flywheel is used to give stability and uniformity of speed. The braking mechanism consists of a copper disc mounted on the shaft and made to revolve between the poles of four large electromagnets. The braking force exerted by the pull of these magnets on the copper disc varies with the amount of current flowing through them. This current is varied by means of rheostats.

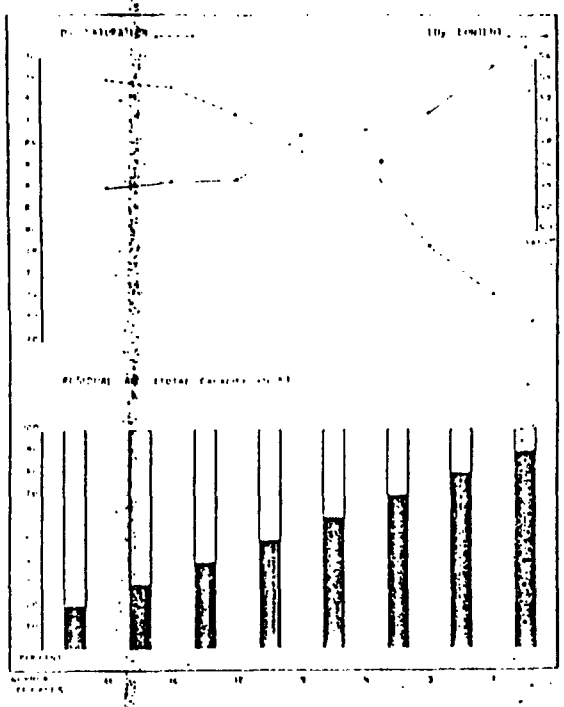
The subject sitting on the seat of the bicycle pedals at a constant speed which he can control by means of a view through a tilted mirror in front of his eyes. The amount of work done is calculated from the amount of weight suspended on a lever arm and its distance from the center of the shaft. As the subject pedals at constant speed the current is adjusted in the magnets so as to keep the lever arm swinging in a narrow arc symmetrically on either side of a fixed point. Thus the mechanical work done is equal to the braking power which will exactly balance a weight on the end of a lever of known length. The length of the braking lever is the distance from the center of the magnets to the center of the shaft. Revolutions of the shaft are counted by a mechanical counter. Allowance is made for friction, which has been accurately measured.

The subject breathes through valves by means of a mouthpiece. The nose is closed with a clip. The expired air is collected and measured in a spirometer, the movements of which are recorded graphically on a kymograph. Values of the ventilation per minute are determined for the subject at rest and during the last minute and a half of a five minute exercise period at different levels of work, sufficient time being allowed for complete recovery between each work period. The maximum ventilatory capacity is actually determined whenever possible by observation during the maximal effort which the subject can maintain. When it is not possible to measure it calculation is made according to the formula given above. Fair agreements are obtained between observed and predicted values as one may see by referring to bottom of Fig. 3.

In Figure 3 we have shown graphically the results of such tests on a variety of abnormal subjects compared with those obtained in 20 young vigorous normal subjects. The values for the ratio Maximum Minute Ventilation have been plotted in each case against the work done expressed in kilogram-meters per minute. The ventilation of normal men fell within the shaded area on the graph. Observations on various pathological individuals in the majority of instances showed higher values than did normals at the same rates of work. Dyspnea begins to be felt in all types of individuals when the minute ventilation is from 55 to 60 per cent of the total ventilatory capacity.

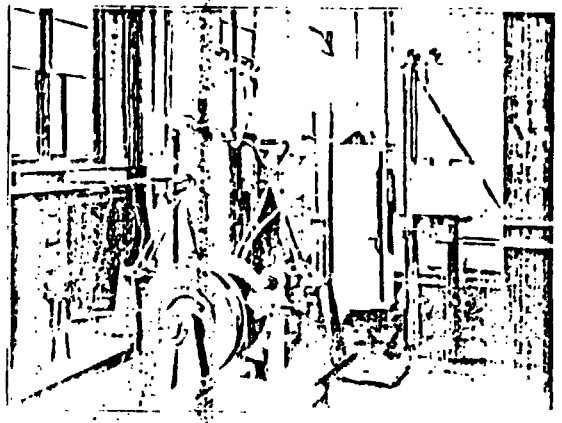
Such a test is not specific for cases of pulmonary fibrosis since the behavior of patients with heart disease and emphysema is essentially the same as those with fibrosis. Diagnostic differentiation of the etiological factors in disability must proceed along the [illegible] clinical lines. The test does permit of determining relative degrees of cardio-respiratory disability by accurate measurement of the amount of work required to produce dyspnea. It is not necessary to have the subject perform maximal amounts of work, since the comparison can just as well be made at rates of work which are within the safe range for the majority of subjects ([illegible] kgm./min.). For complete details see "Respiratory Response During Exercise in Pulmonary Fibrosis and Emphysema", Kaltreider and [illegible] Cann, J. Clinical Investigation, 16:23-40, Jan. 1937.

PLAINTIFF'S EXHIBIT 1508 (?)



LEGEND

FIGURE 1. Relationship between the ratio of residual air to total pulmonary capacity, RA/TC, and the oxygen and carbon dioxide content of the arterial blood



LEGEND

FIGURE 2. Photograph of Ergometer used in Exercise Test [illegible] Investigation 16:23-40, 1947

Reproduced by permission of the Journal of Clinical Investigation [illegible] Avenue, N. Y. Published in [illegible] 16:23, Jan. [illegible]

PLAINTIFF'S EXHIBIT 1508(22)

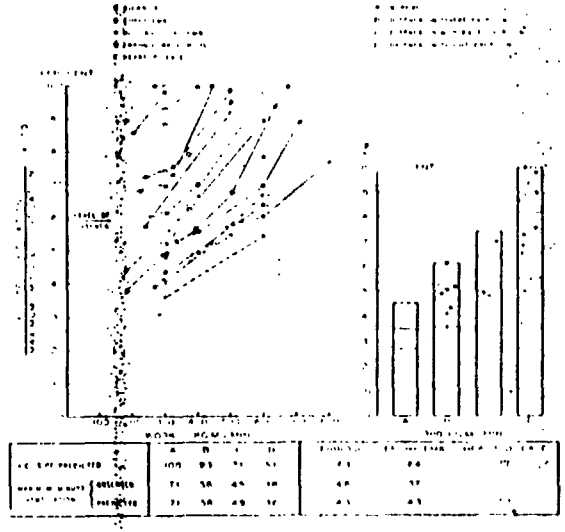


FIGURE 3.—Respiratory responses during exercise of various pathological subjects compared with that of 20 normal men (Hedley [illegible]). See J. Clin. Investigation 16:23-40, 1937.

Discussion of Prof. McCann's Paper

Dr. Enzer stated that Dr. McCann had presented [illegible] of several years of well-conceived and well-executed research into the problems of human pulmonary physiology. His presentation raised a number of points that he would like to have clarified and amplified.

He asked whether right axis deviation was necessarily a sign of right ventricular hypertrophy. His own experience at the post-mortem table had disclosed that out of 130 examinations on individuals dying with evidences of silicosis, but not dying necessarily of the silicosis or its complications, there had been two cases of death from right heart failure. In one of these the right ventricle was [illegible] centimeter thick and in the other 0.7 centimeters thick. These were the only two cases dying of what might properly be termed right heart failure. There were other cases of death from cardiovascular disturbance which did not present such striking right ventricular hypertrophy. These two cases, he felt, definitely were the result of a very massive pulmonary fibrosis. In these two cases, very nearly [illegible]% of the functional lung tissue was involved by massive conglomerate fibrosis. In a few other cases of very severe and far-advanced pulmonary fibrosis, there was some degree of hypertrophy of the right ventricle. On the other hand, there had been many cases of very considerable fibrosis without any evidence of right ventricular hypertrophy. It had occurred [illegible] that the explanation for this might arise out of the anatomic fact that pulmonary arterial circulation appeared to have been completely blunted from the massive fibrotic areas. Emphysema might also be responsible for the cardiac pathology, although he had not been impressed with right ventricular hypertrophy in uncomplicated pulmonary emphysema. Theoretically, pulmonary fibrosis of the silicotic type should be associated with right side heart pathology. Practical experience did not seem to support this theory to the degree one would expect.

Generalized systemic cardiovascular disease, mostly of an arteriosclerotic nature, and sometimes hypertensive, had been a frequent finding. Clinical investigation had impressed him with the high incidence of this type of cardiovascular disease in men of these age periods, and apparently it was entirely unrelated to pulmonary

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pathology. Serial electrocardiographic studies had disclosed a comparatively high incidence of coronary disease.

He asked Dr. McCann whether he felt that the performance of a strenuous muscle effort in the electrocardiographic laboratory could be adequately controlled. He and his associates had tried to evolve methods of testing pulmonary efficiency by tests not employing muscular effort. The method which they had used by Dr. Barach was the progressive reduction of the supply of oxygen in the breathing apparatus and the employment of external resistance to the act of breathing. A brief preliminary statement of their results was given. The fact seemed that if emphysematous patients of the kind had been encountered cardiovascular disease, especially mitral pulmonary valve disease, responded to the test very unfavorably. In fact, with pronounced hypertension, severe arteriosclerosis or extensive pulmonary emphysema responded poorly. They were not entirely satisfied that these tests had been sufficiently extensive to carry out to the laboratories in pulmonary physiology from cases of uncomplicated pulmonary fibrosis, and it might be that they would have to resort to some form of muscular exercise to study this problem.

He then asked whether or not Dr. McCann had studied blood lactic acid during these examinations, and what results such studies had disclosed.

To the question on right axis deviation of the electrocardiogram, Dr. McCann replied that he had gone over the autopsy findings in the cases in which the pathologist had diagnosed pulmonary heart disease and found that only about half of these cases showed right axis deviation. He stated that he had not intended to lay too great emphasis on the electrocardiogram but that the procedure had necessarily been the basis of his operation.

As to whether a portion of the bone contracted and imposed an added resistance on the right ventricle when a branch of the pulmonary artery was entirely cut off, he felt that it did. If the arterial occlusion was primary the capillaries would still be filled with blood by the bronchial arteries and anastomoses of the long-standing occlusion. There might be emphysema and fibrous changes of a severe degree. If the arterial occlusion had been produced by emboli which become more and more avascular as it progressed then the capillaries filled would be reduced in both volume and area by two processes: (1) by an actual decrease in capillaries which had been permanently closed and (2) by emphysema in areas surrounding the occlusions. The arterial occlusions set up which had the effect of increasing the load of the right heart.

Concerning muscular effort, he referred to Dr. Barach's previous work conducted on the efficiency of the human organism. He said that a large part of Dr. Barach's work for the past several years had been devoted to a study of means of measuring the physiological response in relation to external work produced. Dr. Barach's object was to measure the human machine as an engine, his ventilation as a complicated carburetor, and the amount of work that is his oxygen consumption, would be a measure of the engine's efficiency. This in turn would disclose the point at which he developed dyspnoea. Such tests were very difficult to carry out, but he felt they were worth contemplating and that they were worth doing.

He pointed out that there was a certain proportion of patients who deliberately failed to cooperate or to comprehend the purpose of exercise tests. It was hard to find any method of study which was 100% fool proof. But he felt that the tests had been satisfactory and that there was always room for improvement in the technique of such exercise tests and a more functional approach to the problem. That tests at rest, such as Dr. Drinker had mentioned, were inadequate for appraising for exercise might be more valuable for one reason or another.

He stated that an investigation of lactic acid in relation to the problem for the summer in connection with the determination of the rate at which cases of advanced fibrosis progressed [illegible] monthly.

Dr. Dozer asked what criteria were used to [illegible] in differentiating between so-called chronic diffuse fibrosis of pulmonary fibrosis against those with marked emphysema.

Dr. McCann replied that cases classified as simple fibrosis were those not secondary to tuberculosis. They had been classified as obstructive emphysema. They were not caused primarily by fibrosis due to posture, to scoliosis or other conditions and had been put in a separate category from cases of fibrosis of the lungs.

PLAINTIFF'S EXHIBIT

ROENTGENOLOGIC ASPECTS OF THE NORMAL AND THE SILICOTIC LUNG

Dr. E. P. Pendergrass
University Hospital, University of Pennsylvania

It is generally conceded that the roentgenographic examination, when properly carried out, is the most accurate means at our command of determining the existence of the lung, in any case of suspected pneumoconiosis, or of differentiating the condition and any other one which may be present.

The ability to interpret roentgenograms of cases of pneumoconiosis properly must be based upon a knowledge of many important factors which may be enumerated as follows: (1) a knowledge of the anatomy of the chest and many of the physiological problems associated with its anatomical constituents; (2) a thorough familiarity with normal roentgenographic and fluoroscopic appearances and permissible variations therefrom within normal limits; (3) a knowledge of the histology of the lungs and especially of the lymphatic system; (4) a clear perception of the pathology of the condition of pneumoconiosis and of all conditions which may simulate it in roentgenographic appearances; (5) an experienced familiarity with the roentgenographic appearances of the condition in question and of those which may bear a resemblance to it, based upon a fundamental knowledge of the pathology represented; (6) a knowledge of the physical factors involved in the production of the suspected or alleged pneumoconiosis and, (7) the employment of the proper technic to show to full advantage any or all of the abnormalities present, for technic may fully enlighten, may so modify appearances as to be confusing or may be quite misleading.

In studying the roentgenograms or in making fluoroscopic observations of the chest one should take cognizance of every anatomical structure. Otherwise some important abnormality is found to escape notice now and then. The bony framework should receive its share of attention. Anomalies of ribs occur in about one per cent of individuals. Evidences of old fractures should be recorded. Scoliosis may have an important bearing on interpretation for or

against heart and aortic abnormalities. The appearances of the soft tissues of the chest wall may sometimes lead to valuable knowledge of the condition of emaciation. It is well to examine all of the above mentioned structures in some definite order.

The heart and aorta are the most obvious for present observations. The normal heart shadow varies considerably with the position of the individual, his size, the amount of abdominal fat and fluid, the phase of respiration and the distance of the source of radiation when the exposure is made. The various effects upon the size and outline of the cardiac shadow under the influence of pneumoconiosis lesions must be understood. The effect of the fibrosis in the more advanced phases and of the accompanying emphysema with its variable degree of emphysematous change in the lung tissue as well as in the mediastinal tissues. Heart displacements due to the contraction of the fibrosis caused by the emphysema. Severe cases of pneumoconiosis in individuals must usually be excluded from the Army because of the chest. Any estimation of heart size in the roentgenogram must be made with the individual in the erect position and the focus-to-film distance of 6 feet. Focus-target-film distances of less than 6 feet give erroneous information as to size.

Especially the arch of the aorta has not been given the attention it should be. The shadow of the spine of the patient may simulate it. Scoliosis may make it appear dilated. Tortuosity of the aorta should be made evident in the roentgenogram. Especially should abnormal dilatation of the emphysematous chest and the ascending and the horizontal arch. All of these possible errors should be eliminated by the fluoroscopic examination. The aortic knuckle on the left should always extend beyond the second rib and the left edge of the spinal shadow.

The position of the trachea is often of great value. In bronchial adenosis. It should be studied also in the fluoroscopic examination in all properly exposed films made in both the anteroposterior and lateral directions. In the former the shadow of the trachea will be seen to cross the supra-sternal notch with the bifurcation to the right below this point if the patient is normal. Scoliosis may be misleading.

A thorough study of the degree of the diaphragmatic excursions, should be made in every case examined. Especial attention should be given to the diaphragmatic sinuses. The functional

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position or contour of the dome may be altered by a great many conditions of the phrenic nerves, lungs, pleura, air passages, the diaphragm muscle itself and subphrenic lesions. A study of the movement and contour plays a most important part in the diagnosis of silicosis and the estimation of disability. Dome excursion is very variable in healthy individuals. It is more extensive in athletes and older individuals, barring excessive emphysema, and in men than in women. Broad, regular inequalities in level are usually not abnormal in the absence of localized restricted movement and preceded subphrenic conditions, such as localized enlargements of the liver, can be excluded. Sharply peaked contour are abnormal and are due to pleural adhesions or fibrotic changes in the lungs, often enhanced by emphysema. Pleural adhesions are apt to be at the locations of the interlobes, but may be anywhere.

The lung markings are very important in many respects. Misinterpretations of normal variations are responsible for many incorrect diagnoses, and a knowledge of the normal appearance and of all conditions which may alter this is very essential. The normal lung markings are the shadows of the structures which accompany the bronchial tree, namely the arteries, the veins in the more proximal portions of the tree and the supporting structures. In reality they represent the bronchial tree. The heavier shadows are called the trunk shadows and their ultimate and finely visible terminations are familiarly known as linear markings. In cases in which there is unusual prominence of the interlobular septa, as from fibrosis or lymph blocks, these shadows are confused with the linear markings.

Naturally the trunk shadows, and sometimes the linear markings, can become greatly intensified by many abnormal conditions which cause more dense or wider shadows to be cast because of fibrosis, lymphatic engorgement, circulatory states or inflammatory conditions or excessive amounts of secretion or fluid or air in the air passages. Hence it will be seen that the peribronchial, perivascular-lymph node type, or what is used to be called the first stage of pneumoconiosis must be differentiated from a large number of conditions, such as the normal fullness of age, passive congestion from cardiac decompensation or left ventricular, but temporarily, after coronary thrombosis, polycythemia, acute bronchitis, chronic tracheobronchitis, especially associated with upper respiratory tract infections, bronchiectasis acquired either in adult life or in early childhood as a result of interstitial lung changes from pneumonia, abdominal operations, or the unusual type of pulmonary tuberculosis or other malignant metastases. Mild emphysema accentuates the trunk shadows by contrast, but marked emphysema apt to partially wipe them out unless exposures are made with full penetration. Too little penetration greatly intensifies the normal shadows, especially if there is a moderate degree of emphysema present. Some of the faces at times are said to show a characteristic pneumoconiosis appearance when viewed by roentgenoscopy alone, without evidence of right heart enlargement or pulmonary pneumoconiosis in addition. Excessive exposure of the chest wall and film during exposure will also cause the shadows to be too light.

It is highly essential for the experienced interpreter of chest roentgenograms to be familiar with and be able to identify the various prominent trunk shadows just as he must know the normal appearances. This amounts really only to a knowledge of the course of the major branches of the bronchial tree, which are most commonly seen.

The left bronchus, at least part of which can usually be identified in the average normally exposed stereoroentgenogram, is identified clearly, especially by contrast with the overlying dense shadow of the left pulmonary artery and vein, comes off at a more acute angle and is longer than the right for the reasons that it has to pass the left auricle to give off and must pass under the right pulmonary artery and vein and the aorta. It divides into the upper and lower lobe branches. The upper lobe bronchus crosses the course of the lower bronchus and continues under the pulmonary artery and aorta. Its branches must be designated by the trunk shadows as follows: (1) the apical trunks paralleling the aorta; (2) the anterior and posterior trunks which take the general direction of the second rib space; (3) the lingual tip trunk to the lingual tip of the left upper lobe on the left of and partially overlying the heart. The lower lobe bronchus is longer than on the right and has two main branches, the superior plus the right lower lobe bronchus. It can be identified in the sagittal view as a hollow structure. It divides into two main branches which have to be separated from the pulmonary vessels. There is first a single trunk which is given off to supply the apical portion of the lower lobe. The terminal branches are

PLAINTIFF'S EXHIBIT 15(c) (1)

usually divided into four main divisions, the anterior, mesial, lateral and posterior. These are best seen in three divisions in the lateral view as anterior, mesial and posterior divisions. Named designations go no further than these main divisions.

The right bronchus comes off at a more obtuse angle and is shorter than the left, because of the lack of arterial relations. The upper lobe branch passes over instead of under the pulmonary artery and is not so well seen. It has, of course, no luminal or branch or corresponding trunk. The other trunk have approximately the same relations as on the left side. That portion of the right bronchus between the upper and middle lobe branches is usually designated as the right stem bronchus for purposes of special identification. The right pulmonary artery passes over it. The artery and its main branches cast very prominent shadows on the right side and are separated from the right cardiac border by a semicircular space and help to form the right hilum shadow. Opacity of this space in properly exposed roentgenograms is evidence of some abnormal condition. The right middle lobe bronchus, visible as only a trunk shadow, comes off from the anterior aspect of the stem bronchus and passes anteriorly, and hence seems very short in the flat roentgenogram because of foreshortening. Its true relations and direction are better seen stereoscopically. The air passage below the middle lobe branch becomes the lower lobe bronchus, also usually represented by a heavy trunk shadow. The branch of the lower lobe bronchus to the apex of the lower lobe has a somewhat unusual relation, not generally realized, in being given off at the same level as the middle lobe branch, and these two trunks are apt to be confused in the flat roentgenogram. The terminal lower lobe trunks have approximately the same relations and distributions as in the left lower lobe.

The hilum shadows, though altered appearance, contribute their share in the picture of pneumoconiosis in many instances, as when the perivascular peribronchial lymphocytic type of aspect is predominant. Normally they are of a homogeneous darkness, irregular and indistinct in outline but fairly constant in extent. They are composite shadows made up of the right and left bronchi, pulmonary artery and vein, lymph nodes and the supporting connective tissue around the root of the lung. Their prominence depends largely on the absence of *any air containing* lung structure. They are subject to considerable variation in appearance within normal bounds and differences in x-ray penetration. The only practical use of distinguished by the Normal Clinic Committee is that the [illegible] root or vessel shadows should be seen through [illegible] should not be made up of any solid mass of homogeneous density [illegible] abnormal exposures. The right hilum was designated [illegible] *tendency from the top of the right main bronchus* [illegible] of the root of the right lower lobe bronchus into the [illegible] The left hilum shadow was preceded [illegible] from the upper border of the pulmonary artery shadow [illegible] a variable point below. Posterior and lateral [illegible] The proper conception of the normal appearance [illegible] of the hilum shadows in cases of suspected pneumoconiosis [illegible] In the perivascular peribronchial type [illegible] the enlarged and fibrotic pulmonary lymph nodes [illegible] causes an enlargement and increased homogeneity of the [illegible] the prominence will be further increased [illegible] the [illegible] of the contained vessel shadows. The appearance [illegible] portion of the hilum shadows is [illegible] upper lobe seems a factor in the [illegible] lower and the fibrotic consolidation of the [illegible]

The approximate locations of the hilum shadows [illegible] are often of extreme importance. In flat roentgenograms of the chest one cannot determine the lobar location of [illegible] with a high degree of accuracy unless the [illegible]

The height of the apices of the lower lobes [illegible] high in some individuals. The height of the left [illegible] variable room that of the the right.

The lateral view of the chest is always important [illegible] in advanced cases of pneumoconiosis. A pronounced [illegible] gives the best index as to cardiac size in a case of [illegible] with emphysema. The anterior aspect of the cardiac [illegible] normally in close contact with the sternum [illegible] distance. Backward displacement by [illegible] of the lower lobe and lateral [illegible] lower lobe atelectasis. The contact with the sternum [illegible] will vary with the amount of inversion of the [illegible] upper lobes, being much less in [illegible] *severe than in thin* individuals. The most reliable [illegible]

PLAINTIFF'S EXHIBIT 565 (71)
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sensa, as stated in the Normal Chest Committee Report, is to be based upon observations of the lateral roentgenogram of the chest. The great increase in lung structure between the anterior cardiac border and the anterior chest wall is due to the hypertrophy of the lingual tip of the left upper lobe and the right upper lobe seen there. In addition to this there is a considerable increase posterior to the cardiospinal space, also represented by lung tissue of the lower lobes. Both of these elaborations may be much in excess but in the emphysematous chest, and especially that of the athletic, there is apt to be a forward bowing of the anterior chest wall and a marked posterior curve of the spine, which tend to increase the lung space in front of and behind the heart. The more obtuse angles of the ribs with the sternum are of little value in the diagnosis of emphysema. The abdominal type in the athletic is quite similar to that of the emphysematous subject, and in fact, it may not be abnormally affected at all in the latter. Increased lung radiotransparency is the rule in advanced emphysema, but is usually to be regarded as a hinderance in correct interpretation of lung shadows rather than an aid in diagnosis. Depressed diaphragm domes, while common in emphysema, are not reliable as a diagnostic sign. The depth of the posterior costophrenic angle is extremely variable. It is usually deepest in thin individuals and children. This depth has been one of the most valuable findings in the lateral view, because in the posterior costophrenic sulcus many a lesion or effusion which was hidden by the tops of the diaphragm domes in views gone by is now readily detected by the lateral chest roentgenograms.

No one can understand the pathology of pneumoconioses without a basic knowledge of the histology of the lungs, and especially of the primary lung lobule with its terminal air passage system and alveoli and of the lymphatic system. We are indebted to W. S. Miller for much of our knowledge of these structures and whose we must quote at length. The final division of the intralobular bronchi of the secondary lobules are into respiratory bronchioles which enter the primary lobules or lung units. These passages differ from the more proximal ones in not being lined by ciliated epithelium and in having some alveoli opening directly into them. Within the primary lobule the respiratory bronchiole divides into two or three alveolar ducts (ductuli alveolares), each of the latter soon dividing, each atrium opens into several saccular alveolar ducts, each of which has a circumference of alveoli, similar to those in communication directly with the respiratory bronchioles and alveolar ducts. Probably the majority of pathologists now believe that the alveoli are lined by connective tissue and not epithelium. Each primary lobule receives a small pulmonary artery branch which accompanies the bronchiole. The veins are found thus far only entering at the periphery.

There are four sets of lymphatic vessels to its respective lobule, starting in the primary lobule, those of the bronchi, those of the arteries, of the vein and of the pleura. The peribronchial lymphatics communicate within the bronchial wall and form a network which ramifies centrally throughout the entire air passage system to the hilar pulmonary lymph nodes. Their most distal origin is in the peribronchial region of the respiratory bronchiole. The perivascular lymphatics, single trunk in the primary lobule, communicate with the peribronchial. The lymphatics accompanying the veins in the septa are the largest because their ultimate radicles communicate with the pleural plexus. In addition, each vein receives a lymphatic trunk which joins the more superficial lymphatic trunk which joins the accompanying vein. Each venous radicle joins the vein. The radicles arise from the periarterial and peribronchial vessels. The lymphatic vessel of the pleura communicates with the vessels of the septa. A knowledge of the lymphatic system is necessary for a proper understanding of the distribution of the anthracotic pigment and of inspired dusts.

Almost equal in importance to a knowledge of the primary lobule is the location of certain small lymphatic aggregations in the lung structure. They are found at the bifurcation of the bronchi, at the junction of venous radicles and the pleural plexus, at the junction of venous radicles with the vein in the septa, at the angle of division of the respiratory bronchioles and alveolar ducts and in the divisions of all branches of the bronchial tree. They become larger and larger and at more central points they become true lymph nodes. The direction of drainage is toward the hilum except the lymphatics of the pleura covering the lower half of the lower lobe, which empty into the lymphatics of the lung parenchyma over the diaphragm.

PLAINTIFF'S EXHIBIT
PENCARD-Exposure, N. J.

unable to reach the pneumonic nodes and not the lobule. The lymphoid deposits are important in pneumoconioses and in some pulmonary infections as tuberculosis, because they are taking place for phagocytosing dust particles. Silica particles or hydrated colloidal silica deposited in these areas cause what are probably the initial lesions in silicosis, with cell infiltration, cell proliferation, cell destruction and ultimate fibrosis. The fulness and theft of silica laden phagocytes, no doubt in relays, continue until the lymph channels become blocked by enlargements at the sites of the lymphoid deposits. These seats become larger and larger and finally coalescence nodules are formed as the forerunners of the ultimate macroscopic nodules, or the nodular aspect of the condition. The blocked vessels with smaller nodules along the course of the bronchial tree, together with enlarged and more or less fibrous pulmonary lymph nodes are responsible for the appearances of the enlarged lobule. Shadowy and very ill-marking. A complete lobe-wise block causes a decrease in lymph flow toward the pleura, with pleural changes and the following characteristics of advanced cases of silicosis. A block in both directions leaves no course open to the phagocytes except to pass through the walls of the lymphatic vessels into the intercalendar interstitial tissues, as Gardner has shown, with the resulting interstitial fibrosis.

There are definite characteristic events in pneumoconioses that represent pathological phases of the condition. Dr. Gardner will discuss these with you later and detail for you certain descriptions that have been accepted by the Committee of the Kennedy Lab.

Among the conditions that may simulate silicosis are passive congestion of the lungs as a result of cardiac decompensation, pulmonary congestion associated with coronary thrombosis, bronchiectasis, metastases, polycythemia, diaphragmatic infections and miliary tuberculosis. In order to differentiate these various conditions from silicosis it is necessary to have available a complete clinical record, and even with complete clinical records one sometimes confronted with real difficulty in arriving at a definite diagnosis. Slides were shown of a number of these conditions simultaneously to illustrate the similarity of the appearances on the roentgenogram.

In conclusion, I would like to discuss for a few moments the question of silicosis and tuberculosis. There is ample clinical evidence to suggest that silicosis predisposes to a tuberculous infection

Tuberculosis or any other infection does not produce the typical clinical manifestations when it is complicated by silicosis and therefore oftentimes necessary to depend upon the roentgenologic examination for a differentiation between pneumoconiosis and tuberculosis. This is not always a simple matter and may be difficult or impossible. Serial studies, however, are valuable in arriving at a correct diagnosis.

In infections the process is more likely to extend to contiguous places. If preexisting silicosis is present the mediastinal structures including the trachea, will probably not be displaced. If on the other hand, tuberculosis was present as a primary condition there would be displacement of the trachea being displaced to one or the other side, provided there is a unilateral lesion.

In the massive lesions without infection the appearances of the pneumoconiosis is discretely that which one would expect. However, if there is a superimposed infection the lesion is more homogeneous as one would expect in an exudate. Furthermore, occasionally there is calcium formation and even cavities which can be recorded as evidence of an infective process.

The Soft Parts:
- Fat musculature—pectoral muscles.
- Loss or gain in weight.
- Oblique positions causing difference in density of lungs.
- Shadows of soft tissue masses—nodes—calcified, etc.

The Bones:
- Symmetrical position of the clavicles.
- Rhomboid fossae.
- Bicipital and cervical ribs.
- Bone destruction in parathyroid disease and [illegible] tumors.
- Concretion.
- Ossification of cartilages not necessarily abnormal—[illegible] calcification increases generally with age.
- Ribs more at right angle in expiration.
- Scoliosis obscures lung structure on both lateral views—normal both views to same [illegible].
- [illegible]
- Encysted pleurisy is thin [illegible] and [illegible]

PLAINTIFF'S EXHIBIT 45-3 (71)
PENGAD-Bayonne

Normal reflections over vertebra [illegible]. Abnormal shadows in disease.

Subcostal muscles cast shadows suggestive of pleural thickening.

Companion shadow of the first and second ribs, can be identified because they are symmetrical on the two sides.

Visceral pleura -- not seen normally except when it covers accessory lobes.

Thickening of visceral pleura will cause increased density.

Visceral pleura rarely, if ever, becomes as thick as the parietal pleura. Pleura thickens more in lower chest due to movement of chest wall and heart.

Position of interlobar fissures.

MEDIASTINUM:

Trachea -- midline above, slightly to right below.

Divides at disc between 4th and 5th thoracic vertebra.

Angle -- right bronchus 20 degrees.

" -- left bronchus 40 "

During inspiration, the trachea moves downward and forward, unless fixed by a mediastinitis.

Right border mediastinum made up of superior vena cava and innominate artery, ascending aorta mesial to vena cava.

Left border mediastinum formed by subclavian and left common carotid arteries.

HEART:

Not considered in this presentation.

Look for calcification.

DIAPHRAGM:

Right dome [illegible] cm. higher than left.

Occasionally left is higher, even without transposition of viscera.

Costophrenic sulcus depth varies.

Peaking due to adhesions or inelasticity of lung.

Waviness due to individualization of costal components.

Most normal movements are during ordinary respiration, forced inspiration sometimes produces abnormal types due to conflicting costal, abdominal, and diaphragmatic muscle movements.

HILUM SHADOWS:

Bronchi -- arteries -- veins -- lymph nodes -- nerves and connective tissue.

Greater number of lymph nodes on the right and [illegible] upper lobe bronchus than with lower and [illegible] lobe.

One should see varying densities.

Limits of hilum shadows -- 2 inter paces and [illegible]

CALCIFIED NODES:

Homogeneous -- healed.

Mottled -- not necessarily healed.

Dense circular shadows due to vascular trunk.

THE ZONES:

Hilum.

Middle or trunk (vascular).

Peripheral zone.

Expansion is greater in the peripheral than in hilum [illegible] zones.

THE LOBES:

Normal lobation.

Azygos and inferior lobe variants.

VASCULAR MARKINGS:

Bronchi may or may not accompany vessels.

Artery and vein on different sides of bronchi [illegible] during phases of cardiac cycle.

THE BRONCHI:

Upper -- *right* *left*

vertebral

2nd interspace [illegible] lingual tip

Middle

Lower

LUNG FIELDS:

Primary lobules.

Muscular sphincters about channels leading into [illegible]

In asthma they impede egress of air leading to emphysema.

Secondary lobules -- made up of 50 to 250 of primary lobules. These larger ones are found in the base and peripheral portions of the lung.

Pulmonary artery -- follows course of bronchi.

Pulmonary vein -- follows periphery of lobule.

Lymphatics -- superficial and deep.

Bronchial lymphatics in primary lobule [illegible] present in the air spaces as vessels.

Lymphatics of artery anastomose with bronchial [illegible]

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Lymphatics of vein follow veins except that there are no lymphatics along veins arising from walls of the air sacs.

Pleural lymphatics drain toward the hilum.

Some drain through the diaphragm into the abdominal pre-aortic nodes.

Valves in superficial lymphatics point in no particular direction.

Valves in deep lymphatics allow flow in one direction only -- near the hilum and opposite direction near the periphery.

Lymphoid deposits rare before 30 years.

" " prominent at 60 years.

Lymph flow enhanced by massage, passive motion, capillary wall injury, increased venous and capillary pressure, decreased blood proteins.

Lymph flow negligible when the part is at rest.

Rapid appearance of foreign particles in hilum nodes not explained entirely by phagocytosis. Perhaps there are alveolar pores.

Lymph flow decreases 45% in pneumothorax.

Respiration, etc., therefore, plays important role in pulmonary lymph flow.

Rate of lymph flow may vary in different portions of lung because of differences in expansion.

No proof on amplitude of lymph flow. If we know so little about this, there is reason for lack of knowledge of the function of the lymphatics.

EFFECT OF THE PHASES OF RESPIRATION ON THE ROENTGEN APPEARANCES IN THE CHEST

INSPIRATION:

Upper lobe maintains constant relation with ribs.

Lower lobe does not.

Pigment found in upper lobe adjacent to the intercostal spaces.

Not so in lower lobes and next to heart.

Explanation is the massage effect.

Working against inspiratory mechanism are the heart, vertebral column, and portions of the ribs. Therefore, apex of upper lobe does not expand so much as lower lobe. One can conclude that more air goes in lower portion than upper portion of the lobe. Could one deduce, if adequacy of lymph flow and degree of respiratory mobility go hand in hand, that this may explain upper lobe lesions in tuberculosis and

central lesions in silicosis, with clear areas around heart and along periphery?

SILICOTIC LESIONS:

Beading --

Nodulation --

Conglomerate nodulation -- all three seen best in the upper 2/3 of lung fields. Periphery and lower 1/3, relatively free.

Explanation: Emphysema and greater respiratory movement in lower lobes.

Emphysema in the lower lobes due to compensation and lack of pleural adhesions.

Peripheral clear zone due to massage action of ribs.

Pericardial " " " " " " " heart.

When infection is superimposed, the part becomes relatively fixed and the infection extends to the periphery.

ASBESTOSIS:

The opposite obtains.

Lesions are largely in the base and the preponderant lesions are peripheral because of superimposed infection.

The pleura becomes involved early and emphysema, instead of being basilar, is in the upper portion of the chest.

The pleura is thickest around the diaphragm and heart, the areas where there is the greatest massage movement.

DISCUSSION:

Roentgen lesions in silicotic patients have been divided into two large groups for ease of description:

Simple silicosis and silicosis with infection.

Asbestosis causes a different type of pathological process.

Do lesions which we have ascribed to simple silicosis really occur or do they occur only where there has been a superimposed infection which has healed?

Do individuals working in dusty atmospheres develop beading or nodulation without infection?

Are lesions thought to be due to silicosis with infection, all silicosis with tuberculosis or are there other numbers of lesions that are chronic and active?

Do not contaminating dusts modify silicosis to such an extent that our terminology should be augmented properly to identify the industry or contaminating dust, which has been done in the coal industry?

PLAINTIFF'S EXHIBIT 1973 (9)

Discussion of Dr. Pendergrass' Paper

Dr. Gardner commented upon the value to those familiar with the subject of a contribution like Dr. Pendergrass'. He recalled that to furnish a background he himself had presented the story as if there were no doubts or uncertainties but that many of the questions which had been raised in the morning's paper had troubled all investigators of the problem. To those who were hearing about silicosis for the first time it might appear that Dr. Pendergrass and the other speakers were so uncertain about it all that they could not be expected to diagnose the most obvious case. This was hardly true. Dr. Pendergrass had raised questions as to the mechanisms involved in the production of the disease; he had asked his audience to reconsider some of the evidence to be certain whether all of the accepted explanations were the only possible ones; but he had not disturbed the fundamental criteria on which all diagnosis rested. To ask such questions was valuable because it compelled one to think more deeply, the only way in which progress could be made. He warned that no one should get the impression that it was impossible to differentiate between silicosis and other conditions which might simulate it for in practice Dr. Pendergrass and others were doing it every day.

In his presentation Dr. Pendergrass seemed to indicate that the lymphatic system might not actually perform as effectively in removing inhaled foreign bodies from the lung as had been described. He said that in sections of pneumoconiotic lungs which he had seen he had not found dust-laden phagocytes inside of the lymphatic vessels on their way to the lymph nodes. He had looked at some of Dr. Clark's preparations at the University of Pennsylvania and had seen, through a transparent window in the ear of a living rabbit, phagocytes filled with red blood corpuscles that had stayed in the same position inside the same lymphatic for weeks at a time. The only movement of the phagocyte was a slight oscillation without progress. Since Dr. Gardner had stated that the lymphatics seemed to [illegible] the silica so that it could produce reaction, he would infer (facetiously) that the body would be better off without a lymphatic system.

Dr. Gardner replied that it was difficult to observe the phagocytes in transit through the lymph vessels; only rarely were they

detected except in the case of the thrombi in [illegible] one had to infer that such transport occurred [illegible] a gradual increase in the amount of pigment in the lymph nodes at the root of the lung as more and more dust was inhaled. Since the lymph vessels constituted the only direct pathway [illegible] and the nodes, it was a natural inference that they were the [illegible] even though chance sections failed to [illegible] the act of passing. In Dr. Clark's preparation of [illegible] the insertion of the transparent windows [illegible] the normal function to such an extent that the [illegible] interrupted.

He said that he had often asked himself [illegible] accumulations of dust-laden phagocytes had [illegible] connective tissues which surround the lymphatic [illegible] passing through into the vessel to be eliminated. He had [illegible] the conclusion that the cells accumulated because they were too numerous or, in other words that the individual had been [illegible] more dust than his lungs could eliminate by physiologic [illegible]. The same was true of individuals who developed silicosis; their lymphatic systems were inadequate to handle an abnormal amount of silica dust. The dust that did enter the lymph channels and was carried to lymph nodules was irritating enough to injure the filtering mechanism and then a vicious circle of reaction began.

Dr. Schulz, who had worked with Dr. Cecil Drinker [illegible] the lymphatic system was asked to comment. He admitted that the function of the lymphatics had been a bugaboo to everyone who had studied them. It was unknown, for example, how material passes from a blood vessel or a bronchus into a lymphatic. He was [illegible] certain of the validity of the common assumption that the lymphatics constituted a sort of sewage system. Their primary function, to his mind, was to return soluble materials to the blood from the tissues. He had been trying to satisfy himself whether the lymphatic vessels constituted an open or a closed system. The rate at which various substances passed to and fro through the walls of a lymphatic suggested that there were open pores in its walls but these openings, if they existed, were too small to be visualized. He pointed out that bacteria and relatively large foreign particles could enter a lymphatic without being first engulfed in a phagocyte. If they had come from a pulmonary air space one would assume that [illegible] had been an important factor in their movement.

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Dr. Gardner agreed that many dust particles entered lymph vessels without the help of phagocytes although he had some doubt as to the relative frequency of the two mechanisms.

He discussed the generally accepted theory that the flow of lymph was maintained passively through the moving tissues of the lung in respiration. In order to bring out the extent of such motion he asked Dr. Pierson to speak of his experimental observations on human lungs.

Dr. Pierson reported that in the treatment of tuberculosis it was desirable to know how much movement of the lungs occurred under various conditions and whether movement could be influenced by posture. By taking films of normal subjects lying on their sides it was found that the diaphragm moved more on the down side than on the upper one. To determine whether a greater amount of work was done by the lower lung, whose diaphragm was moving further, comparative measurements were made of the air passing in and out of both bronchi in dogs lying on their sides. It was found that more air was moved through the bronchus on the down side and hence more work was being done. To determine how far out upon the lung such movements took place, films were taken in expiration and in inspiration of patients with calcified subpleural tubercles and it was established that on the down side the calcified tubercles actually moved further than when they were on the up side. Dr. Pierson's explanation for the greater movement of the diaphragm on the dependent side was the tension exerted upon it by the elastic lung above and the pressure of the solid abdominal viscera below. To immobilize the lung completely it was necessary to paralyse the diaphragm by phrenicectomy (cutting the nerve supplying the diaphragm) and to have the patient lie upon the operated side. This was proved by observing a patient so treated in whose pleura some silver clips had been left at a former operation. On fluoroscoping this patient it could readily be demonstrated that there was absolutely no movement of the dependent lung. These observations proved that the lung moved appreciably in respiration but that such movement could be practically stopped by appropriate measures. (The effect upon lymphatic circulation was not discussed but it may be recalled that when motion in a tuberculous lung is limited by collapse therapy symptoms of intoxication often decrease quite suddenly. It is assumed that this is a result of closure of the lymphatics with a decreased absorption of toxic products from foci of tuberculosis. *Dr. Gardner*)

Dr. Gardner called attention to the presence of [illegible] small circular shadows seen along the blood vessels in roentgenograms of the chest. He said that he, with Dr. Simpson, had been among the first to visualize the earliest demonstrable evidence of the presence of silicon nodules located in lymphoid tissue along the course of the blood vessels. Sometimes in very good stereoscopic films a [illegible] could be detected appearing in exceptional cases as evidence of the involvement of a small portion of the lung. But he felt that [illegible] to be certain whether beaded thickenings of the septa were due to minute silicon nodules. He cited a case of a foundryman of whose films had long been shown to Dr. Pendergrass and himself. The films presented the most pronounced and unmistakable beaded appearance and he had not hesitated to ascribe the appearance to minute silicon nodulation in view of the occupational history. Dr. Pendergrass, however, was of the opinion that the appearance was caused by heart disease. A second film, taken a few weeks later, had then been exhibited which proved Dr. Pendergrass right. The beading had entirely disappeared, which could not have happened had the appearance been due to silicon nodulation. Dr. Gardner stated that he did not believe that beading was a sole diagnostic criterion but that it still interested him in his attempts to correlate the shadows seen on the films with the lesions that produced them. He asked Dr. Riddell his opinion and the latter stated that beading did not constitute a safe basis for a diagnosis of silicosis and that he felt that all beading should be taken with more than a [illegible] of salt.

Dr. Gardner was asked as to the size of the lesion necessary to produce a shadow visible on a roentgenogram. He replied that it depended upon its density in relation to that of the lung tissue. A very small calcified focus would be visible on a background of normal lung where as a patch of such pneumonic tissue as large as the thumb nail might escape detection until the air spaces in it were well filled with exudate. Dense silicon nodules would [illegible] could be seen when they were only a few millimeters in diameter. [illegible] reaction of the type we found as an allergic reaction. The subpleural tubercles produced in guinea pigs by [illegible] rarely exceeded 2 mm. in diameter and they could [illegible] in the dust arsenal [illegible] and bury about them

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Dr. Pendergrass then commented on Dr. Pierson's demonstration that the periphery of the lung moved further than the central portions and stated that this observation to him indicated a more rapid flow of lymph at the periphery. Pressure of the expanding lung against the more resistant heart, vertebral column and portions of the ribs would tend to massage dust out of the superficial lymphatics and into the deeper parts of the lung just as in the top of the boys it was apparently massaged away from those portions of the pleura that are constantly applied against overlying ribs. He inquired whether accelerated lymph flow and massage against resistant structures might not explain the paucity of silicotic nodules seen at the periphery of the lung in the roentgenogram. He asked Dr. Gardner to comment.

The latter replied that the massaging action of the lung against overlying ribs was the time-honored explanation for the freedom from pigment in this location and its accumulation in the intervening areas beneath the intercostal areas. Where the dust accumulated it undoubtedly occurred along the lymphatics. In the white intervening zones it might have been forced into the lymph vessels or it might never have accumulated about them. But in the case of silicosis he was not prepared to grant the validity of Dr. Pendergrass' fundamental thesis that there were fewer nodules in the periphery than in other parts of the lung. Perhaps the roentgenographic evidence was deceiving although he assumed that Dr. Pendergrass had based his opinion on stereoscopic appearances. Flat films would of course give a false impression because the edges of the lung were so thin that comparatively few nodules would cast their shadows on the roentgenogram. But his observation of the lungs themselves had convinced him that there were just as many silicotic nodules in a unit volume of lung at the periphery as in the central portions and that, in the absence of distorting infections there were as many in the upper as in the middle and lower lungs. The first nodules to appear were situated immediately beneath the pleura. They originated in the lymphoid tissues present at the junction of the pleural and the communicating lymphatic trunks. These nodules could be visualized in the roentgenograms but they were not very numerous. As more formed in the parenchyma of the lung their distribution was quite uniform, involving both the peripheral, middle and hilum zones. As Dr. Sampson had pointed out, contraction of the fibrous tissue in such nodules then drew the lung inward but as a result the peripheral air spaces expanded to fill the thoracic cavity, resulting in the so-called compensatory emphysema. After this change had developed the advanced case of silicosis did show a peripheral zone relatively free of nodules. The change was most marked at the lower and outer angles of the lower lobes where the increase in translucency in a film permitted the interpretation of emphysema. In the mid and upper portions of the lung such changes were less marked and one did not ordinarily recognize it as emphysema but it was nevertheless present. To check this observation anatomically one should examine the lung after distention with air for in the collapsed state the peripheral air spaces were closed; this part of the lung had shrunk and the nodules appeared to touch or almost touch the pleura. On distending the lung, however, a surprisingly wide zone of pulmonary tissue made its appearance peripheral to the nodules.

In the case of conglomerate fibrosis the reaction frequently extended into and was imperceptibly fused with the thick and fibrous pleura. Coarse or "bullous" emphysema developed in other parts of the lung because no air spaces were generally left to dilate peripheral to the fibrous mass.

Dr. Pendergrass then said that he thought he had allowed for the factor of distention but he still wondered why the conglomerate shadows should extend entirely across the lung field and thought that a disturbed lymphatic circulation might be the cause.

Dr. Gardner replied that he thought that the massive areas of fibrosis developed in portions of the lung injured by infection. Perhaps the obstruction of lymphatic vessels in the area of infection at first helped to retain an unusual amount of dust. But he believed that extension of such areas of disease occurred directly, the scar formation advancing directly through all of the tissues at the periphery of the area.

Dr. Pendergrass asked whether silica alone, without concomitant infection, would produce changes visible on a roentgenogram. Dr. Gardner replied that he was convinced that it would cause discrete nodulation and others had generally accepted the experimental and post mortem evidence on human beings as convincing. He thought that conglomerate fibrosis would have to be considered in a separate

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category as indicated in his talk on pathology. While some believed that conglomeration resulted from an apparent fusing together of an unusual number of nodules in a limited portion of the lung he had asked himself why more nodules should have happened to form in one location than another. There was no special area of the lung constantly selected for this type of change and consequently it did not appear to result from some local anatomic peculiarity. The fact that it occurred now in one place and now in another suggested some accidental anatomic disturbance and the most likely cause of such disturbance was infection. Study of the conglomerate fibrosis did not suggest a fusing or a pulling together of nodules that had once been separate and discrete; it revealed nodules embedded in a matrix of diffuse connective tissue. He then went on to demonstrate how silica could react upon the young connective tissue cells of an incompletely resolved pneumonia to produce such a diffuse matrix as a background for the nodules. He commented that after most pneumonias in the general population, the inflammation subsided and the tissue returned to an apparently normal condition, but that sometimes, even without a known exposure to silica the inflammation did not disappear but became organized and formed scar tissue. He believed that Dr. Riddell's point, that one should clearly differentiate between infection in its etiologic sense and its clinical sense was most helpful. In the former, infection had merely altered the soil and then disappeared as an active condition; in the latter, infection was still injuring the tissues locally and was giving rise to constitutional symptoms.

He reiterated that some cases of "conglomerate" fibrosis might be produced by collapse of a portion of the lung due to pressure on the bronchus supplying it but he had not seen them. Or again, the process might be irritated by other dusts inhaled along with silica, which produced streamer-like strands radiating from the surface of the original silicotic nodules. More silica accumulating on these strands could in time cause them to thicken and ultimately obliterate all of the lung between the nodules.

He mentioned one case which Dr. Sampson would undoubtedly use to illustrate his talk in the afternoon session where the successive films demonstrated more and more nodules in a given area of the lung which might be used as an example of conglomeration. But

[illegible] did not prove that all massive fibroses formed in this manner, it still remained to [illegible] the cause for the increased number of nodules in this particular part of the lung.

Dr. Riddell commented that roentgenologists were describing only the shadows seen on films. The shadows appeared to have run together but such an appearance did not necessarily mean that the nodules causing the shadows had fused. In his opinion conglomerate shadows should never be interpreted as indicative of simple silicosis without an exhaustive study of the case; most often they were produced by an element of infection which he implied would often break down and manifest itself by symptoms in the course of time.

Dr. Gardner stated that both he and Dr. Riddell had been members of the committee which wrote the report on the "Roentgenologic Appearances in Silicosis and the Underlying Pathological Lesions" published in the Public Health Reports for August 2, 1935. There they had tried to differentiate between lesions produced by infections that were healed and only operative in an etiologic sense and those that were still clinically active though generally chronic. Changes of the first type were catalogued under simple silicosis roentgenologically, as "conglomerate shadows"; pathologically, "as the result of coalescence of discrete nodules". Dr. Gardner adhered to his responsibility for the latter but said that further study had now convinced him that this interpretation was incorrect. The second type of clinically significant infections were grouped under the heading of "silicosis with infection" and had been termed "massive shadows of homogeneous density". The Committee had recommended a distinction when one was at work upon its classification but if experience should prove that all lesions of this type would ultimately break down and become active such a distinction would be unjustified. He believed that the classification, which was only proposed tentatively, would ultimately have to be modified.

Dr. Pendergrass asked whether only tubercle bacilli could produce the changes resulting in conglomeration. Dr. Gardner replied that he believed bronchopneumonias of other origin might have similar effects. He hoped to observe some develop and progress to their terminal stages in the annual serial examinations of our miners. It might prove to be the case that the non-tuberculous pneumonias would be responsible for the conglomerate fibrosis that remained healed permanently and that those caused by tubercle ba-

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cilli would be the ones that ultimately broke down. He had no data to prove such a hypothesis but there was a little evidence in favor of the converse, i.e.: that tuberculous lesions could be healed completely even in the presence of silicosis. As an example he cited the experimental tuberculosis in animals inhaling chert, which had healed so completely that it was hardly recognizable as tuberculous and the similar findings in human cases of sidero-silicosis.

Dr. Pendergrass asked whether conglomerate fibrosis was as disabling as the more active silico-tuberculosis. Dr. Gardner said that both would cause dyspnoea and decreased capacity for work but that symptoms of intoxication due to activation of the infection would sooner or later add to the seriousness of the latter type of condition.

Dr. Riddell expressed his gratification that Dr. Pendergrass had pointed out one error that had crept into roentgenographic readings and had probably caused more trouble than any other single factor. This was the habit of referring to exaggerated linear shadows as evidence of fibrosis when, as a matter of fact they were not caused by a fibrosis at all (see pathology). He then recalled certain young men who had been rejected for employment because of such shadows but who had returned later and were accepted because the exaggeration had disappeared. A true fibrosis could hardly have vanished in such a short period of time.

Dr. Pendergrass then stated his belief in a basal localization of lesions in asbestosis with a tendency to subpleural distribution when infection complicated the picture.

Dr. Gardner said that here again there was an apparent difference of opinion due to the method of observation. He could not agree that his anatomic specimens showed any more evidence of asbestosis in the base than in the apex of the lung. He could not count the number of the lesions for they were too poorly defined. He had grave doubts as to what constituted the characteristic anatomic picture in asbestosis. There were nearly 40 specimens in his museum and no two of them were alike nor did they show any common feature which was sufficiently characteristic to have made him suspect asbestosis at autopsy unless he had been forewarned by knowledge of the occupational history. Confronted with 500 films and told that among them there were 10 of persons with asbestosis, he doubted whether he could have picked out more than 2 or 3. He felt

that it was absolutely essential to have much more correlation of pathologic and roentgenologic evidence before drawing many conclusions about this condition.

Very little effort has been made to discuss various manifestations of silicosis as that was amply covered by Dr. Simpson. Prominent points, however, in which there is some question as to the correlation of pathologic and roentgenologic findings were raised in order that these subjects could be discussed by Dr. Gardner and others present.

PLAINTIFF'S EXHIBIT 1505 (Sc.)

ROENTGENOLOGIC DIAGNOSIS OF SILICOSIS

DR. H. L. SAMPSON
Trudeau Sanatorium

Too many individuals are today being classified as silicotic or pneumoconiotic on the basis of roentgenograms of such poor quality that normal pulmonary markings are mistaken for evidences of pathologic states. Therefore it is fundamentally important to recognize the essentials of a proper roentgenogram of the chest.

The characteristics of the suitable roentgenogram are not difficult to define. It should reveal, in proper balance, the shadows of the various physical structures of the chest wall and the enclosed pulmonary tissues.

The *density* of the film should be such that one can see the shadows of the blood vessels, both in the more exposed portions of the lungs and in those areas that lie behind the heart. One should also just be able to discern a black line between the bodies of the vertebrae. Films that are too light or too delicate destroy the identity of the pulmonary markings and their pattern may simulate a pathologic condition. Such films fail to reveal the vascular shadows behind the heart or diaphragm; they produce misleading exaggerations of the root shadows and they often present a false appearance of pulmonary congestion. In films that are over dense certain delicate shadows are not seen although if they are not too dense stronger illumination will compensate for this defect. Very dense films cannot be depended upon except those purposely taken to reveal the internal structure of heavy fibrous areas.

Contrast is important to enable one to differentiate between shadows of varying quality but it may be exaggerated to the point where false impressions are created of the physical structure of the underlying anatomy. Excessive contrast tends to make the shadows of inactive tuberculosis foci simulate those of active lesions. A good guide to proper density is the ability to visualize both the fine blood vessels in the periphery of the lungs and those behind the heart.

Definition is the third important essential. Upon it depends one's ability to differentiate between the detailed characteristics of

the shadows cast by different anatomic structures. In a film with proper detail, the shadows of the heart, diaphragm and larger blood vessels radiating from the root into the lower lung will be sharply defined and clear cut. Loss of definition may be due to movement in the subject, film or tube, the use of a tube with too large a focal spot, too short a tube—object distance or poor screen contact.

Two kinds of roentgenograms are ordinarily used; stereoroentgenograms, which permit visualization of shadows in their proper perspective or depth and single or flat roentgenograms, in which the shadows of all the structures in the lung, both the normal and those due to disease, are projected onto one plane. The comments to follow apply equally well to making either kind.

Four prime factors are involved in taking a roentgenogram and it is necessary to understand the effect of each of them upon the final result. Voltage, milliamperage, exposure time and tube-object distance (T.O.E.) are fundamental. The tube, screens, films, photographic processing etc. are, of course, assumed to be adequate for the purpose.

Voltage primarily affects the contrast of a film. The higher the voltage, the less the contrast; the lower the voltage, the greater the contrast. Ordinarily the voltage is changed to compensate for the thickness of the chest of the individual being examined.

Milliamperage is varied to compensate for necessary changes in other factors. The amount of current used is determined by the output of the x-ray apparatus and by the capacity of the x-ray tube. The smaller the focal spot of the latter, the less its capacity.

Tube-Object Distance affects primarily the detail, magnification and degree of distortion of the shadows. The recommended distance is 5 feet although satisfactory results can be obtained at 5 or possibly even 4 feet.

Time is reduced to arrest motion but nothing may be gained beyond certain speeds. The time should not exceed 1/10 second and exposures of 1/20 to 1/30 second are common objectives.

While an x-ray apparatus with an output of less than 100 milliamperes is capable of producing fairly good roentgenograms, it is not recommended because of the length of exposure required. With apparatus of more than 100 milliampere capacity it is essential to employ a tube with the smallest possible focal spot to [illegible]

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definition. For distances greater than 6 feet it is necessary to use tubes with larger focal spots so that no advantage is obtained by exceeding this limit.

INTERPRETATION

To interpret roentgenograms satisfactorily one must know the characteristics of a good film, which have just been described, the normal pattern of the healthy lung and its permissible variations, the type of shadows cast by tissue reactions to different kinds of dust and the pa[illegible] [illegible]ting from infection and other pathological conditions as they exist independently or in combination with reactions to different dusts.

Ever since the Trudeau X-ray Laboratory was started, twenty-six years ago, an earnest attempt has been made to keep the interpretation of films upon a purely objective basis. The films are read without knowledge of the patients symptoms or occupational history. A brief description of the shadows is given with the statement that the findings are consistent with some pathologic condition. The final diagnosis is rendered after this information has been correlated with data from other sources. By maintaining such a standard of objectivity one avoids the influence of the knowledge that the patient is complaining of pulmonary symptoms or exhibits physical signs over a certain part of his lung. In viewing a possible case of pneumoconiosis one does not have to ask himself whether the particular dust which the individual claims to have breathed is capable of producing pathologic reaction and whether he can detect evidence of such reaction in the film; he merely asks whether the film before him shows shadow patterns that are abnormal.

To maintain such a standard it is of paramount importance to be thoroughly familiar with the shadow patterns of the healthy lungs. The tree-like system of the arteries, veins and bronchi cast shadows on a film which vary somewhat in different functional phases but are invariably characterized by branching linear shadows. After this pattern ever so little, by a focus of disease in the pulmonary parenchyma, by an exudation of cells or by a congestion, and in the well-taken roentgenogram a reflection of this change in physical structure will be revealed. Interpretation of such changes is not dependent upon a history nor upon a knowledge that physical signs

have been elicited over a certain area. The d[illegible] [illegible] the pattern is altered does not constitute a diagnosis; it merely calls attention to a change in physical structure in a particular [illegible] of the lung. Having satisfied himself that the change [illegible] in [illegible] determine the reason for such changes.

It is obvious that after years of experience in examining thousands of films one should develop standards and that certain patterns which are constantly associated with particular types [illegible] should be interpreted in the light of that experience. The pathologist has such standards and uses them daily in making his gross and microscopic diagnoses on tissues. Only when he encounters pictures that are unfamiliar does he find it necessary to resort to [illegible] sources of information before he interprets the tissue changes before him. It is reasonable to assume that the experienced roentgenologist should develop similar standards.

By carefully following several thousand cases of [illegible] tuberculosis it has been amply demonstrated that [illegible] manifestations of disease too slight to register on a well-taken roentgenogram are exceedingly rare and that the shadows which are registered, can be visualized with no knowledge of the patient's condition.

The study of films of persons exposed to different types of dust has been approached in the same objective manner. Several hundred roentgenograms of groups of men employed [illegible] [illegible] granite, foundry and steel industries [illegible] without knowledge of the particular conditions of exposure. Serial films of over 6000 men from a group of hematite [illegible] have been followed for a period of four years, again without knowledge of whether the subject was an office executive or a [illegible] operator underground. Incidental films, also numbered in hundreds, from men exposed to a great variety of dusts, such as a [illegible], fluorspar, talc, feldspar, etc., have been reviewed and [illegible] [illegible] reactions of one general type or [illegible]. The findings have [illegible] [illegible] with [illegible] of exposure, with the data obtained from [illegible] and in a considerable number of cases with [illegible] the main the correlation has been good. [illegible] no change have actually had little pathologic reaction, [illegible] silicotic fibrosis have been correctly catalogued. [illegible] with linear types of reaction have been shown by [illegible]

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a thickening of the blood vessels from the development of chronic inflammatory reactions in the walls of these structures.

The only exception for defining the standard patterns that is lacking is a careful study of an appreciable number of men exposed to relatively pure silica dust. The sporadic cases with such exposures that have been reviewed have generally been complicated by so much tuberculosis that the picture has been obscured. This deficiency is keenly appreciated and we have hesitated to generalize from an experience confined largely to the action of mixtures of silica and other substances.

THE HEALTHY CHEST

Obviously, if the roentgenogram is going to be placed upon an independent footing the interpretor must have a thorough knowledge of the shadows cast by the normal structures of the lungs and thorax. Dr. Pendergrass has already described them in great detail. Here it is only necessary to recapitulate the essentials.

It is necessary to bear in mind that a chest which is "healthy" may present a variety of appearances. Age affects the lungs just as it does the other organs of the body. The shadows cast by the various structures in the child's lung are quite different from those of the adolescent or the aged, and yet all of them may be healthy subjects. The phase of respiration or of the cardiac cycle when the exposure is made, modifies the condition of the structures in the lung and the type of shadow that they cast.

The bulk of the shadows in the roentgenogram of a normal lung are due to blood vessels and in particular to the blood inside these vessels. The latter can be demonstrated by taking roentgenograms of the excised lung before and after the blood has been washed from the vessels. Such shadows are arranged in the form of a leafless tree, with the larger branches radiating from the roots of the lungs and the finest twigs at the periphery. In suitable stereoroentgenograms one has no difficulty in visualizing the smaller branches and hence the discovery that "the markings reach the periphery" is not indicative of a pathologic condition as is often stated. The vessels to the lower portion of the lung are larger because of the greater volume of tissue to be supplied. Consequently their shadows appear heavier and longer than those to the upper lung but this again does not indicate disease.

As age advances the pattern of vascular shadows becomes increasingly distinct and clearly defined, perhaps because of a gradual thickening of the vessel walls and perhaps because of changes in the surrounding air spaces. Whatever the cause, the vascular shadows increase in prominence with age both in persons exposed to dust and in others. Hence interpretations based solely upon the appearance are of dubious value.

The shadows of the bronchi play only a minor role in the normal pattern of the lung. These tubes, like the alveoli or air cells, contain air and only the larger branches, near the root of the lung, have walls thick enough to cast an appreciable shadow. Even between the main branches of the pulmonary artery and vein, the walls of the larger tubes can be recognized but at the periphery they are not visible.

Dr. Pendergrass has demonstrated the anatomy of these and other pulmonary markings.

THE BEAR EXAGGERATION

In the study of films from individuals exposed to the various forms of dusts there has been a general assumption that one must see some evidence of this dust or of reaction to it in the lungs. As a consequence emphasis is laid upon shadows, which in any case under examination for other causes such as tuberculosis, would even be mentioned in reporting the roentgenographic findings. Findings of "linear exaggeration," "feathering of the bronchovascular," "linear prominence" etc. have become routine practice in many laboratories. Not only have such shadows received unwarranted reports but they have been interpreted as evidence of fibrosis. This has led to great confusion for it is becoming obvious that not all dusts are capable of causing fibrosis. The writer has characterized the degree of so-called linear "exaggeration" in pneumonoconiosis as the "stage of imagination."

There can have come to autopsy who [illegible] died before death by [illegible] of them will be discarded by way of diagnosis [illegible] been employed for 15 years in a gypsum mill [illegible] roentgenograms were said to have a moderate [illegible] lower lung markings which were only slightly [illegible] He died [illegible] from his cardiac condition [illegible]

PLAINTIFF'S EXHIBIT 12 (12)

a moderate pigmentation of his lungs and lymph nodes. Microscopic sections demonstrated that the pigment was collected in clumps in the walls of the blood vessels and in the interlobular septa. About the deposits was a very slight chronic inflammatory reaction but there was no fibrosis. Possibly these changes cast some shadows in the roentgenogram but the same picture could have been produced by an engorgement of the blood vessels associated with the existing cardiac changes. Certainly one could have allowed his imagination free play and if the history of exposure to dust had been unknown it would have been natural to ascribe the slight changes to this cause.

But there are more definite degrees of exaggeration of the linear shadows cast by the vascular tree which are apparently produced with prolonged exposures to dusts of various kinds. The roentgenogram in such cases reveals a generalized thickening of the shadows cast by all the blood vessels and by the connective tissue septa between pulmonary lobules. The accentuation of these fine markings in the periphery of the lung fields produces a net like confusion of interlacing shadows. Study of histologic sections reveals that the thickening is caused by somewhat more extensive zones of chronic inflammatory reaction in the vascular trunks around the lymphatic trunks. The change is not always a fibrous one except in so far as the microscope may reveal a few strands of collagen, but it does not involve the parenchyma of the lungs. Consequently there is little or no interference with respiratory function.

The frequency of this more marked stage of linear exaggeration is determined by the character of the dust which the subjects have inhaled and by the duration of the exposure. However, one cannot always be certain that inhaled dust is the cause; chronic infections and cardiac lesions produce similar effects. In reviewing several thousand films taken during preemployment examinations at a large steel plant a very considerable number with first or second grade linear exaggeration have been discovered. This material probably represents the average picture in an American industrial population.

In groups of men exposed to mon-chromic dusts or to dusts with less than 10% of free silica, one is more apt to discover the advanced stages of linear exaggeration after prolonged employment but the frequency of this condition does not increase regularly. For example, a roentgenographic survey of over 1300 employees from a

remote plant in various parts of the country [illegible] employed. The data in the case records of [illegible] concentrations of [illegible] appreciable amounts of free silica [illegible] 8% of the films were read as negative; nearly 12% showed the first stage of linear exaggeration (the stage of "suggestion") and [illegible] the second or more marked condition. Analysis of these films revealed [illegible] employment periods, but a increasing incidence [illegible] degrees of linear exaggeration [illegible] period; thereafter until the 50 to 60 year period [illegible] exception. Had there been more men in the group employed from 30 to 60 years the figures would undoubtedly have been more significant. Nevertheless there were 110 of the 1300 men [illegible] and when they were grouped together the combined percentages of both grades of linear exaggeration were less than three [illegible] members of the 25 to 30 year group.

Another example may be cited from the group of [illegible] miners. These men had been exposed to varying concentrations of [illegible] silica and iron ore. About 35 percent of their roentgenograms showed no change; a little over 30 percent revealed first degree linear exaggeration and approximately 13 percent revealed the second or more marked linear stage.

Several autopsies have been obtained from men [illegible] had been interpreted as marked linear exaggeration. Gross examination of the lungs revealed the red brown pigmentation known as [illegible] Microscopic sections demonstrated that the dust was present chiefly along the lymphatics of the blood vessels, septa and pleura. There was associated with a more pronounced chronic inflammatory change than was encountered in the man exposed to gray iron dust and [illegible] there were a few minute silicotic nodules in the lungs [illegible]. In other words, the linear inflammatory change was not [illegible] but an admixture of some silica in the dust that had entered the lungs.

It has naturally been assumed that the linear type of [illegible] the case of persons exposed to [illegible] would progress [illegible] silicosis. It was hoped that the roentgenograms [illegible] the kymatic mottle would reveal [illegible] which would progress [illegible] stage to another. Many of the workers have been [illegible] roentgenograms taken at intervals of one year. [illegible] been found in which such a progression had occurred. [illegible]

PLAINTIFF'S EXHIBIT 1569 (Cr)
PENGAD-Bayonne, N. J.

posures will be necessary to determine the frequency and the average time required to affect the truncation.

An attempt has been made to determine whether the presence of linear exaggeration alters susceptibility to infection. Analyses of the cases among iron miners has revealed more infection in the two groups with roentgenograms of these two types, than in the normal group. However, it is not always certain whether the infection was not itself the cause of the thickened blood vessels. Likewise, it is inconceivable that the presence of such reactions could throw any added strain upon the right heart.

The experience has demonstrated that linear exaggeration may be caused by faulty technique in making the roentgenogram or by the physiologic changes incident to advancing age or by pathologic changes such as right heart failure, chronic bronchitis, asthma and bronchiectasis. In the case of persons exposed to mixed dusts containing free silica, linear accentuation presumably precedes nodulation but the actual transition has yet to be observed in our cases. In individuals exposed to non-siliceous dusts only the slighter grades of exaggeration are usually found. The more advanced stages are occasionally seen but their failure to coincide with the duration of exposure suggests that they are often due to other causes. In all cases, except those due to definite disease like bronchitis and bronchiectasis, the absence of clinical symptoms and decreased working capacity makes it seem obvious that linear exaggeration is in no sense a serious condition.

SIMPLE SILICOSIS, DISCRETE NODULATION

The inhalation of sufficient quantities of free silica over a sufficient period of years results in the formation of discrete fibrous nodules, never more than 4 mm. in diameter, uniformly distributed throughout all parts of both lungs. In the very advanced stages a few nodules may fuse together forming small conglomerations but in the early stages such collections have not attained any great size. In the advanced case of discrete nodulation there are frequently areas of emphysema, particularly in the lower and outer margins of the lungs. Since this reaction develops after the nodules such areas are free of these foci. In the roentgenogram the shadows of the nodules are visualized as a generalized speckled or spotted appearance throughout both lungs. The term nodulation has been coined to describe it.

In the early stages of nodulation, when the nodules are very small, the normal linear pattern of the lungs is obliterated and somewhat overpainted by a veiling about the lung fields superimposed upon it are the fine speckled shadows of the nodules. The appearance has been aptly described as the "ladies' fan" shadow.

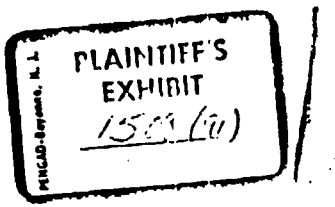
As the nodules increase in size these shadows tend to obliterate the linear pattern of the lungs. The [illegible] nodules, now 2 to 3 mm. in diameter, demonstrate [illegible] perhaps, the most typical appearance of silicosis. The nodules tend to remain discrete in most places although there is some [illegible] slight tendency to coalescence.

In the most advanced stages each nodular shadow [illegible] in diameter the tree may be considered in full leaf. Now there is no trace of the vascular pattern. In the immediate [illegible] are [illegible] separated by normal lung [illegible]

A definite tendency toward coalescence in localized areas [illegible] the costophrenic angles now become [illegible] and [illegible] lucent areas.

In any of the foregoing stages of nodular fibrosis the [illegible] shadows may exhibit well marked modification. The lymph nodes at the hilum enlarge and increase the reaction about the nodules which converges into this area produces considerable [illegible] on the mechanical tissues. In relatively early cases one may [illegible] to visualize on the film individual nodes which have enlarged. Later these contract and often contain foci of calcification. The generalized fibrosis in the surroundings to the produces a "[illegible]" appearance which runs out from the root into either lung field.

Although it has never been possible to follow the [illegible] and care successively through a [illegible] second [illegible] lation, it is customary to class the [illegible] Because of the greater clinical significance of the [illegible] It is [illegible] that tuberculous complications [illegible] in the case when the disease becomes [illegible] simplex [illegible] merely that there has been a greater opportunity for [illegible] to be superimposed as the lesions grow older.



Stereoroentgenograms are of great help in deciding whether early nodulation is present. In the proper perspective fine scattered nodules are more clearly visualized but they do not seem to be as numerous as when all of the shadows from the entire thickness of the lung are projected upon one plane. As a consequence roentgenograms which might be read as possible first degree nodulation on a flat film are frequently interpreted as advanced linear exaggeration when viewed stereoscopically.

The films which we have thus far studied have been of men exposed to mixtures of silica and other substances. The histologic reaction in these cases consists of atypical nodulation with very considerable linear change produced by the associated non-siliceous elements in the dust. While the roentgenograms reveal a picture which is essentially nodular, the shadows of such foci are not always clean cut and sharply defined and there is a background of interwoven linear shadows. It is not yet possible to diagnose such pictures as characteristic of sidero-silicosis as distinct from pure silicosis but further experience may enable us to do so.

Progression of nodular fibrosis. Our opportunities to observe progression of silicotic nodulation have been limited almost exclusively to the above mentioned group of hematite miners. Practically all of these men are still employed and consequently exposed to some inhaled dust. However, the institution of preventive measures has reduced the concentration of dust to very low levels. During the four years that this large group of miners have been under observation the progression rate from a linear to a nodular stage of fibrosis was of the order of only 2 or 3 cases per thousand per year. If all the cases in the study are included the progression rate is less than 1 per thousand per year. In other industries, where the composition and concentration of the dust is different the rate will probably vary correspondingly.

We have not had an opportunity to follow any cases in roentgenograms who have been removed from their dusty environment for any period of time. Britten and Heed reported a considerable group who showed no evidence of nodulation on ceasing work but when reexamined after a period of several years had developed such changes. However, Dr. Campbell has loaned us a series of films of a Polish coal miner whose fibrosis steadily increased during a period of 12 years after he ceased mining. His disease was of the

conglomerate type and he finally died. Autopsy revealed no evidence of active infection but a conglomerate fibrosis with intense anthracosis probably modified by a limited amount of silica.

The presence of complicating infection appears to accelerate the development of nodular fibrosis. In the hematite miners the statistical evidence of such progression has been observed about 3 times as frequently as in miners without infection. The actual number of cases progressing is so insignificant that with careful precautions they might be overlooked.

Complications are not infrequently encountered, the most common one is compensatory emphysema. This change may be associated with advanced, discrete, nodular fibrosis but is almost invariably present in cases with conglomerate disease. Emphysematous bullae occur at the base of the lung but more developed elsewhere. Evidence of bullae is rare. It is manifested by a dark or cystic area in the lung field from which most of the pulmonary markings are absent. Nodular shadows are either entirely absent or scarce. In a case with developing emphysema now under observation several fibrous strands [illegible] with displacement of over an inch in certain of the vascular structures which [illegible] are discernible. The change in their position from month to month [illegible] that the fibrous lung is contracting and the increasing amount of lucency and peripheral rarification constitutes evidence of the frequent, developing emphysema.

Pleurisy does not accompany uncomplicated silicosis with associated infection, either in healed or active phases, with proved fibrous adhesions developing over any lesion which reaches the surface of the lung. The slight thickening of the pleura which occasionally nodule impinging on that membrane is not sufficient to cause changes to [illegible]

Changes in the other thoracic organs have not been of common occurrence in cases with silicotic nodulation. Enlargement of the cardiac shadow has been only an occasional finding in the hundreds of films that have been reviewed. The age of the individual has seemed to us to have a more direct relation to its occurrence than the presence of nodulation. While we have not taken the opportunity to tabulate our findings it is our impression that enlarged heart shadows have been more common in the cases without nodulation

PLAINTIFF'S EXHIBIT 150 (iii)

than in those with this change. We have frequently commented that the enlargements which were associated with nodulation involved the left as well as the right side of the organ.

Only rarely has the diaphragm been elevated and the ribs depressed in cases of discrete nodulation. More often the compensating mechanism was that of emphysema which causes no dislocation of the [illegible] viscera. In most instances the shadows of the dome of the diaphragm have been smooth.

SIMPLE SILICOSIS OF CONGLOMERATE TYPE

The roentgenogram generally presents the picture of a second or third degree generalized nodulation with localized areas of more dense reaction. These occur most often bilaterally in the upper or middle thirds of both lungs. Rarely are they seen at the bases. The shadows of such conglomerate foci are very dense and their borders are sharply defined. Some of them radiate from the roots of the lung, others lie entirely surrounded by parenchyma more or less heavily strewn with nodular shadows. If they reach the pleural surface this structure is involved and casts its own distinctive shadow. Evidence of associated emphysema, often of the [illegible] type, is more readily detected.

Gardner has speculated that such lesions would be most likely to develop in positions of the lung previously injured by infection. We have several cases now under observation in which it would appear that conglomerate foci are developing as a result of local contraction of the lung. Nodules which were previously separate and discrete seem to have been drawn together like chips of wood floating upon a pool of water. While definite evidence of an underlying infection cannot always be made out the fact that the movement of the nodules is always toward the apex makes it seem probable that an element of tuberculous infection is responsible. The development of an associated emphysema about the area offers further support of this view.

On the other hand, very occasionally we have seen small localized collections of nodules which appear to be in the process of coalescence without associated evidence indicative of infection. Whether, in subsequent films, these foci will result in the formation of areas of conglomerate fibrosis remains to be demonstrated.

DIFFERENTIAL DIAGNOSIS

Differentiation of silicosis from several other conditions on the basis of films alone is sometimes difficult and always requires the best grade of stereoroentgenograms. Clinically, the difficulty is not nearly as marked because many of the conditions likely to be confused give rise to well marked symptoms. Miliary tuberculosis, for example, could never be present in a man well enough to remain at work and the same would be true of miliary carcinoma. The question is often asked, however, whether one can differentiate silicotic nodulation from other pathological conditions on the basis of the film alone. In general the question can be answered in the affirmative although, of course, a certain number of mistakes will be made.

In the case of miliary tuberculosis good stereoroentgenograms, or in many instances good single films, will reveal the collateral inflammatory zones about each tubercle which differentiates these lesions from the "dry" appearance of the silicotic nodule. The blood borne tubercles extend into the costophrenic regions but this is not usually the case in nodular fibrosis. Compensatory emphysema does not occur in miliary tuberculosis. In adults most miliary spreads are associated with foci of older tuberculosis in some portions of the lungs, a complication which need not be present in silicosis.

In metastatic carcinoma of the lungs, the tumor nodules are generally larger and their shadows have a softer quality than those of fibrous nodules. The scirrhous forms are apt to be confused with massive conglomerate disease. Pulmonary congestion secondary to cardiac lesions is usually most prominent at the bases of the lungs. It is manifested by a generalized haziness with an accentuation of [illegible] along the latter may be confusing unless the change is restricted [illegible] of the reaction and its localization tend to differentiate [illegible] generalized nodulation of silicosis.

Certain forms of fungus infection may prove difficult to differentiate. Generally, however, the shadows are not nodular and not generalized throughout both lungs. Such infections tend to produce diffuse shadows with an exaggeration of all of the [illegible] resulting in a pattern not unlike that of [illegible]

PLAINTIFF'S EXHIBIT 1505 (93)

With a knowledge of the age, sex, medical and occupational history, and clinical findings in the case, the type, arrangement and location of the shadows in the roentgenogram generally permit one to make an accurate diagnosis. Serial films taken over a considerable period of years are not necessary for diagnosis but yield valuable information as to the evolution of the disease process.

SILICOSIS WITH INFECTION

Since tuberculosis infection constitutes the form most frequently encountered as a complication of silicosis, its manifestations will be discussed in greatest detail. As stereoroentgenograms have been recognized for years as the most accurate means of detecting evidences of tuberculosis in the general population, it is somewhat natural to expect that they might occupy a similar position for silicotic subjects. In persons not exposed to dust it can be conservatively estimated that this method will reveal about 99 percent of tuberculous pulmonary lesions. Their patterns are varied.

The healed foci of the "primary complex", which represents the residual scars of childhood infection, consist of one or more subpleural foci of fibrosis, generally associated with calcification together with similar areas of reaction in one of the tracheobronchial lymph nodes. Either one or the other member of the group may heal so completely that evidence of it is not visualized in the roentgenogram. These lesions have been observed in thousands of cases with active or inactive adult type tuberculosis who have passed through Trudeau Sanatorium and it has never been possible to detect development of new disease about the old healed foci. For this reason we cannot believe that the childhood focus becomes reactivated and furnishes the organisms which produce the new adult reinfection.

Adult type tuberculosis is most often observed in the upper third of one or both lungs. In all but its completely healed and fibrous forms it is characterized by localized clusters of "mottled" shadows. These are cast by clusters of tubercles, either exudative or proliferative in type having the distribution of a bronchopneumonic process. They frequently conglomerate to form larger masses but at their borders one always finds the mottled shadows of more isolated tuberculous foci. The familiar areas of rarefaction in their centres, sometimes containing semifluid exudate are interpreted as cavities.

The visualization of the fluid level, which does not change with position, is helpful in diagnosis. As the disease becomes more proliferative in character and fibrosis develops, the latter contracts and produces distortions of the lung and adjacent viscera. The upper lobe may contract as visualized by the elevation of the fissure between the upper and middle lobes on the right. The trachea is pulled to the affected side often with marked distortion. Associated pleurisy is manifested by a diffuse cloudiness and by visualization of the thickened membrane in the antero-posterior view obtained along the lateral borders of the lung.

Tuberculous lesions, unless they are essentially healed (inactive) are characterized by constant change. The disease progresses in a characteristic fashion, usually by aspiration with the development of the new foci of bronchopneumonia in the lower portion of the same or the opposite lung. Pleural effusions are common complications.

The fibroid form of tuberculosis is characterized by string-like shadows of fibrous tissue radiating throughout the upper lung fields with associated groups of clear cut mottled shadows and perhaps the well defined annular shadows of fibrous walled cavities.

Miliary tuberculosis is less common. Its manifestations, consisting of minute nodular shadows extending to the extreme periphery of the lung with associated areas of haziness from collateral inflammatory reaction have already been described under the heading of differential diagnosis.

The recognition of tuberculous infection in the presence of silicosis presents a somewhat different problem but while it is more difficult most of the infectious foci are probably being detected. A detailed comparison between roentgenograms taken before death and the gross and microscopic appearances in many autopsy specimens forms an accurate basis of interpretation. The different types of tuberculous lesions that are found in ordinary subjects have occur in the silicotic but in addition there are two varieties that are peculiar to the latter.

The primary complex can generally be recognized upon a background of silicotic nodulation. Although other observers have reported human cases in which the primary focus of infection has become reactivated in the presence of silicosis we ourselves have failed to detect evidence of such reactivation in the thousands of cases that

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PLAINTIFF'S EXHIBIT
1508 (71)

we have reviewed. For this reason we have not regarded such lesions as a source of danger.

The scars of chronic, apparently healed apical tuberculosis are not infrequently encountered in persons who also have silicotic nodulation. Evidence is at hand to indicate that the apical tuberculosis antedated the reaction to the silica. While we have seen very few of the scars which we have regarded as thoroughly healed become reactivated by subsequently inhaled silica, such a possibility always exists for it is difficult to affirm that a tuberculous focus is completely healed.

Three courses of evolution are open to such lesions, (1) they remain healed and merely accumulate unusual amounts of silica in the scar tissue, (2) they may reactivate and spread by aspiration as they do in non-silicotic subjects, or (3) they may reactivate and spread atypically, producing the condition designated as "silicotuberculosis."

In the first case, where the focus of infection remains healed, presumably because it no longer contains living bacilli, the accumulated silica merely accentuates the scar tissue already formed. The result of such reaction is the conglomerate type of silicosis already described. The healed infection merely alters the tissue in which the silicosis develops. When the infection spreads by aspiration, as it does in non-silicotic hosts, the clusters of mottled shadows can be seen lying between the more clearly defined and uniformly distributed nodular shadows of the silicotic reaction. Early cavity formation, fibrosis with associated distortion and chronic pleurisy are later developments. Such cases are much less common than the next type.

Silico-tuberculosis is the most frequent outcome. This is the new condition which is neither tuberculosis nor silicosis, but the product of the combined action of both tubercle bacilli and silica. This is the lesion in which there is no evidence of aspiration into other parts of the lungs, little or no evidence of the mottling characteristic of infection, merely a large localized shadow with evidences of compensatory emphysema and pleurisy. It simulates the conglomerate fibrosis already described under simple silicosis but in serial films taken over a period of four or more years this kind of focus slowly increases in size. In the cases which we have seen break down, the centre of a large fibrous area may soften and an annular shadow, indicative of cavity, develop. Perhaps a purposely over-exposed

film is necessary to reveal it. The slowly expanding border of the area may in time develop evidence of mottling as the infection finally reaches the more normal lung about the old fibrous scar. Such lesions are slow in evolution; for years they may persist without much change as if the tuberculosis were being resisted by the surrounding capsule of fibrous tissue which is itself constantly becoming thicker from the silica that it contains. But in time, often after years of almost imperceptible progression, the infection breaks away, progresses more rapidly, perhaps with the development of cavity, and then behaves like tuberculosis in an ordinary host. When this has happened the outcome is almost invariably fatal.

In addition to these evidences of chronic tuberculosis in the upper lung, which in the great majority of instances seem to develop from latent foci already present before the silicosis developed, there are also more active manifestations of new infections. These occur in the infraclavicular, middle and basal portions of the lung. They are much more apt to be exudative in type; they are much less prone to resolve and disappear as so frequently happens in non-silicotic subjects. The shadows characterizing such lesions again consist of non-symmetrical clusters of soft, fluffy mottling which spread quite rapidly to involve contiguous portions of the lung field. The shadows of the silicotic nodules in the involved areas lose their sharp definition and each becomes surrounded by an ill-defined hazy zone. Inflammatory reaction in the intervening air spaces also results in a diffuse haziness which tends to obscure the shadows of the normal pulmonary markings and those of the nodular lesions.

A similar picture of so-called "permodular tuberculosis," sometimes develops quite uniformly throughout most all parts of a silicotic lung. Usually the process is initiated by the development of one or more localized areas of mottling which is followed quite rapidly by the appearance of zones of fluffy shadow about all of the hitherto sharply defined nodular shadows. The latter become larger and lose definition. The shadows of the original nodules might be compared to those which would be cast by kernels of dry corn; those of the foci with permodular reaction might be produced by corn that had popped. Permodular tuberculosis can, of course, have no counterpart in uncomplicated cases. The process runs an acute course and changes develop rapidly in serial film. The original localized foci of mottling often coalesce and thin-walled cavities de-

PLAINTIFF'S EXHIBIT 155 (45)
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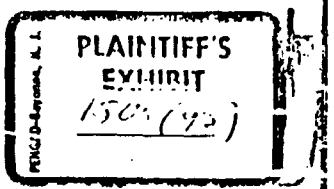
velop, the perinodular shadows extend to involve most of the lung field between the nodules and the picture then simulates that of a tuberculous pneumonia.

All of these manifestations of tuberculosis in the silicotic host are being encountered in routine examination of roentgenograms. Except in the acute perinodular type, the end stages of silicotuberculosis and the rather unusual cases of apical tuberculosis which spread by aspiration, the clinical picture and physical examination are not particularly helpful. Therefore it is incumbent upon the roentgenologist to be familiar with these various manifestations, to follow enough of them serially to establish criteria by which he may hope to prophesy their behavior. In individual cases, seen for the first time, it is often very difficult or impossible to be certain whether an area of massive fibrosis has resulted from an old infection which has completely healed and will never progress very far or whether it is merely a latent infection gradually evolving as silicotuberculosis which is ultimately going to break down and manifest itself as chronic tuberculosis. All such massive shadows should therefore be regarded with suspicion and should be followed with serial films taken at intervals of at least one year. In time we may establish some basis of determining which are permanently healed.

The detection of non-tuberculous infections in the silicotic lung is not particularly difficult but it sometimes requires several roentgenograms to determine the nature of such infections. Most of them are associated with clinical symptoms which establish the diagnosis but there are some changes that have been assumed to be due to infection which appear and perhaps disappear again without demonstrable symptoms. The pneumonias and abscesses are revealed as localized areas of cloudiness without the mottling which is characteristic of tuberculosis. Serial roentgenograms of course reveal the rapid evolution of such processes. While resolution is the usual outcome of pneumonia the incidence of organization is said to be higher in the silicotic than the non-silicotic individual. Our own experience with such conditions is confined to a very few cases. Bronchiectasis has been a very rare finding, probably not more than half a dozen cases have been discovered in all of the films of all men exposed to different kinds of dust. The same is true of pulmonary cancer whose incidence has been even less. For these reasons we cannot ascribe either lobar pneumonia, bronchiectasis or cancer

to the presence of silicosis or any other form of pneumoconiosis.

As mentioned above, there are some cases with silicotic nodulation in which rather ill-defined, hazy shadows of considerable extent make their appearance in the course of routine serial examinations without any symptoms admitted by the subject. Such shadows often appear in the bases, cover areas several centimeters in diameter and are almost never bilaterally symmetrical. They do not obscure the costophrenic adhesions or apparent contraction of the lung. Sometimes they persist for a few months and then disappear, others that are still under observation, have remained without much change over a period of years. If, as we suppose, they are caused by infection the complete absence of symptoms must be due to long-continued absorption through the obstructed lymphatics. These cases clearly need more study.



THE CLINICAL PICTURE AND DIAGNOSIS OF SILICOSIS WITH CONSIDERATION OF DISABILITY

DR. A. R. RIDDELL
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There appears to be a growing tendency on the part of physicians to depend almost entirely on the x ray for the establishment of diagnosis in diseased conditions of the chest. Radiography offers undoubtedly the most important and accurate means at our disposal for this and many other purposes. Complete reliance on it however, to the exclusion of other diagnostic methods, may lead to error or partial error, which may be quite as unfortunate for the individual under investigation. In silicosis failure to recognize the presence of the pathological condition may actually be to the affected one's advantage, when the x ray only has been the source of information.

In any disease the clinical picture develops only after due consideration has been given to all the data which can be assembled regarding it. In this respect silicosis is not an exception. In developing this clinical picture in silicosis the occupational and clinical history may be considered as representing the background, the roentgenological findings the foreground, while physical examination and such accessory aids as the laboratory offers supply the perspective and high lights necessary to its completion.

Thus in silicosis diagnosis depends on—

(1) *History—particularly a history of the inhalation of adequate quantities of dust containing silica.*

(2) Information obtained through the appraisal of symptoms and clinical signs.

(3) Information resulting from laboratory investigation.

(4) Specific x ray appearances.

As occupational history will be dealt with in detail during these meetings I shall not refer to it except to stress its extreme importance in the clinical picture of silicosis, and in fact in many other conditions of occupational origin. It is surprising that with the intense

present day industrialization so little interest is often evidenced in academic circles in this important avenue of enquiry.

It is obviously unnecessary to stress the importance of chest radiography in the diagnosis of silicosis. This is the subject of special discussion here. Let me remind you, however, that the x ray picture in silicosis supplies information which will be of the greatest value only when the shadows it presents are related back to the underlying pathology which occasioned them.

Silicosis may be considered as arising in two main forms—simple silicosis and complicated silicosis, or properly silicosis with infection. The latter, having regard to the type of infection most frequently encountered, is generally termed silicosis with tuberculosis. In this connection it is important to remember that the x ray appearances occasioned by silicosis are not in every case exactly similar but show minor differences which apparently depend on differences in exposure.

As might be expected these two forms of silicosis are not equally important. Simple silicosis, if one may be sure of its simplicity, is relatively innocuous. It may be present in an individual presenting no indication of serious disablement and whose ability to perform his accustomed work may be little, if at all, impaired. Of equal importance is the circumstance that such an individual does not constitute a danger to his fellow workmen as silicosis in itself is not infectious. Complicated silicosis on the other hand is always serious. It is apt to occasion severe disability in the affected workman. If the infection be the tubercle bacillus he may be a serious menace to those with whom he is in contact. Careful attention to physical examination, particularly when this is augmented by data from the clinical laboratory, affords in general more useful information in regard to the differentiation of types than does the x ray film alone. It is well to remember, however, that no single case of simple silicosis can be looked on as final in respect to type until the affected individual is dead. Simple silicosis as progress occurs always has the tendency to pass into the complicated form.

The terminology applied in describing silicosis is sometimes apt to be rather confusing; particularly is this so when it is under consideration as a pathological condition. The clinical picture presented by an individual case during life may differ somewhat from the pathology disclosed at autopsy. In some quarters a feeling is grow-

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ing that tuberculous infection, but not necessarily tuberculous disease, plays an intimate part in most instances when silicosis occurs in the human subject. It is therefore necessary to be clear when considering the various types whether the term is used in a purely etiological sense or in the sense of representing actual disease. When, therefore, one speaks of simple silicosis one does not mean that some[illegible] the etiological picture tuberculous infection may not have played a part in its occurrence. One does mean, however, that obvious tuberculous disease has not been present. This point is of importance and may well be kept in mind when the subject of silicosis is under consideration.

Silicosis gives rise to no symptoms nor clinical signs which are pathognomonic, except perhaps in so far as those presented by the x-ray film may be considered in this light. With it are associated however signs and symptoms sufficiently suggestive to enable an observer to determine its presence and differentiate it from other lung conditions with an approximation to accuracy. This will be facilitated if a clear history of exposure to silica dust has been first obtained. With the increasing application of the x-ray in diagnosis careful physical examination may not be as important as it once was. Even now it is possible that under certain conditions the physician may be called upon to form his opinion when recourse to radiography is not immediately available.

SYMPTOMS

Dyspnoea.—Dyspnoea is the commonest symptom. It is almost always present in those whose chest radiograph discloses characteristic appearances. It may be very slight, noticed only on unusual exertion, or it may amount to extreme shortness of breath which embarrasses the sufferer even at rest. The dyspnoea of silicotics generally presents a peculiarity of special significance which contrasts it from that associated with many other conditions of which dyspnoea is a feature. It is not accompanied by orthopnoea. Thus in silicosis the sufferer is able to lie flat in comparative comfort if no other condition producing dyspnoea is present. Orthopnoea is only found in silicotics when lung fibrosis is massive and has replaced large sections of the lung tissue. Shortness of breath in silicotics is characteristically associated with unusual muscular effort. It does not occur spasmodically and does not resemble true asthma. However the silicotic may also suffer from asthma. When this is the case the cause of the asthma should be searched for independently, for no evidence exists that the two conditions are ever related.

Chest pain.—Most silicotics experience chest pain at some time during the course of the condition. This may be very slight, amounting to little more than a sensation of restriction, but is often severe and tends to be increased when effort gives rise to breathlessness. It is commonly referred to the anterior mid portion of the chest but is quite frequent at the lateral bases.

Dizziness.—Dizziness is quite common. It may not be advanced as a complaint but can frequently be brought out by enquiry. As the dizziness generally occurs when increased muscular effort has also produced dyspnoea and disappears when the dyspnoea subsides it should probably not be considered a separate symptom. It may however be experienced on change of position or when the upright position is assumed on arising in the morning.

Weakness or fatigue.—Many silicotics complain of being weak. It would appear that they use this term to indicate that they become tired easily because of shortness of breath. It is not generally noticed when they are at rest. True fatigue occurring in a silicotic independent of effort always indicates a complication or the presence of some other independent condition.

Sleeplessness.—Sleeplessness is quite prevalent among silicotics. It is not a favorable symptom as it adversely affects the general condition. It is often found to be the result of excessive cough. Sometimes it apparently indicates severity.

Cough.—Cough is a symptom which may actually constitute a sign. It has been referred to as the commonest symptom in silicosis which I think is an error. It may be dry but is often associated with more or less sputum. Cough in the silicotic who has been in recent exposure may be entirely due to the irritating effect of the dust he has breathed. It is very common in men who work in dusty atmospheres whether they are affected by silicosis or not. It is occasioned by dusts incapable of producing silicosis as well as by those associated with its production. It has been found that even in the silicotic cough often clears when he has been moved from contact with dust, or has been supplied with an effective mask. Nevertheless cough independent of exposure is a common symptom. When silicosis is well established, or in fact at any time in the course of its progress,

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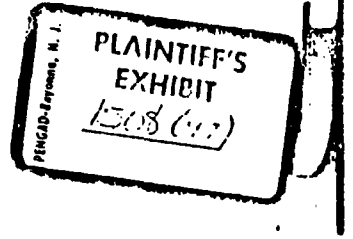

the development of cough, particularly when excessive and productive, should be regarded with suspicion. It is then an indication for further investigation as it suggests an infective process.

Night sweats.—Night sweats are not experienced by those affected with simple silicosis. The occurrence of night sweats have the same significance in the silicotic as they have in those not affected.

Haemoptysis.—Not uncommonly in silicosis a history of blood stained sputum or the occurrence of small haemorrhages will be obtained. Although these do apparently occur in seemingly simple cases haemoptyses warrant careful consideration and no case giving this symptom should be classed as simple until the presence of tuberculous disease has been ruled out.

SIGNS

Weight changes.—As simple silicosis develops the affected individual tends to increase in weight. This weight increase is probably associated with a slowing up process of which he may be quite unconscious. Thus in silicosis significant and progressive loss of weight is very suggestive of a complicating tuberculosis. Frequent weight observations are important in the supervision of exposed individuals, particularly in those who have already become silicotic, and variations from the normal trend should be looked on as the indication for further investigation.

Expansion.—Few who have developed or are developing silicosis have good chest expansion. In them this rarely exceeds 2" at the nipple line and is often much less in those frankly silicotic. Occasionally one may see a case which presents the radiological evidence of silicosis with an expansion of 3". I once saw it as much as 4". Restricted chest expansion is thus the rule. This restriction may often be better appreciated through palpation of the chest than by actual measurements with the tape. By the palpating hand one frequently senses a lack of resilience and poor elasticity in the chest wall. In simple silicosis the restriction is uniform. Regional limitation is not characteristic of the simple condition. When expansion is very limited the anterior chest wall tends to be lifted forward and the accessory muscles of respiration are actively used in the respiratory effort. Sometimes it appears as though the diaphragm is the only muscle employed. In most cases with limited expansion shortness of breath is the rule while at the same time the respiratory rate is increased. These individuals breathe rapidly. The respiratory rate generally falls in the 20's, even with the patient at rest. [illegible] in the 30's.

Percussion & Tactile Fremitus.—Tactile fremitus affords [illegible] [illegible] of value and that obtained through percussion [illegible] ally disappointing. Nevertheless the percussion note is frequently altered. It is as a rule impaired. Impairment in silicosis, particularly in simple silicosis, is apt to be overlooked as it occurs on both sides of the chest in an equal degree. Sometimes the note is hyperresonant. This probably relates to the occurrence of extensive emphysema. Hyperresonance may be so marked that it [illegible] the demonstration of extensive consolidated fibrotic areas.

AUSCULTATION

Breath sounds.—Definite changes in the breath sounds [illegible] generally be detected by the stethoscope. The [illegible] of intensity. In silicosis breath sounds are characteristically [illegible] blanketed. They may be emphysematous in character with the [illegible] pirator phase prolonged. In many instances a rather [illegible] shortening or pinching off of the inspiratory phase can be [illegible]. This shortening of inspiration is commonly associated with a change in pitch which is higher than normal.

Adventitious sounds.—As might be expected the adventitious sounds elicited in silicosis are those common to chest [illegible]. They carry the significance usually associated with them and generally indicate associated infection. With certain reservations therefore when rales are heard in the silicotic the possibility of tuberculosis should be suspected unless this can be ruled out by [illegible]. Rales demonstrable in the lower third of the chest have relatively greater significance in the silicotic than in those not so affected because in silicosis tuberculosis very often makes its appearance in the neighborhood of the hili or toward the lung bases. Of particular significance in regard to the presence of tuberculous disease [illegible] small, rather high pitched musical sound sometimes heard which occurs about half way through inspiration, explosive in character, [illegible] taking somewhat of the nature of a rale and a rhonchus.

Blood pressure and heart rate.—Blood pressure does not seem to be materially affected by the presence of simple silicosis. [illegible]

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be slightly raised in cases with extensive fibrosis of long standing. Low blood pressures occasionally occur in complicated cases. It is surprising however that even when tuberculous disease is extensive and is accompanied by toxæmia blood pressures are seldom low. Acceleration of the heart rate is the rule in silicosis. Most sufferers present rates between 80-90. Occasionally the rate is consistently maintained above the 100 mark. In a few prolonged observation in hospital has failed to indicate any explanation for this increased rate other than the lung fibrosis.

Fever.—The temperature is not elevated in simple silicosis. An elevation, unless during abnormally hot weather, suggests infection. This infection, however, may not be tuberculous. It has been frequently noted that silicotics are apt to run a temperature for several days following the administration of a general anæsthetic. Temperatures as high as 105° Fahrenheit have been noted under these conditions. In addition the occurrence in the silicotic of attacks of lung congestion accompanied by slight fever are quite common. Observation of a temperature range over a period will be found valuable in the supervision of silicotics.

Cyanosis.—It might be expected that cyanosis would be a feature of silicosis but such is not the case. Neither does clubbing of the fingers appear except perhaps in long standing cases with massive areas of dense fibrosis. When cyanosis is present or the fingers are clubbed it is wise to suspect some other disabling condition, probably of cardiac origin. Particularly is this true when such an individual also suffers with orthopnœa. Care should therefore be exercised to eliminate such possibilities before ascribing associated disability to silicosis. Curving of the fingers suggests tuberculous involvement. It is not seen in the simple case.

Clinical Laboratory Aids.—When the component parts of the clinical picture are being assembled laboratory information will be found of great importance. It is on the laboratory side that one may expect further advances in the study of this condition to be made. At present much information is afforded from this source and the clinical laboratory should be in constant use in connection with the observation of exposed individuals. The most important laboratory procedure is sputum analysis. These analyses should be made frequently, at spaced intervals. No case of silicosis can be classed as simple merely because two or three specimens of sputum

have failed to show the presence of bacilli. In addition to the examination of direct smears the practice of culturing the sputum for tubercle should be encouraged. This is common practice in Ontario. In connection with sputum culture it has been found that a variety of media [illegible]. In practice we use four. Each potato medium is placed on the [illegible] media Petragnani, Dorset egg, Wolfsen and a Silica media, which [illegible] seems to be to such a selective [illegible] media have shown any particular advantage over the others but it has been found that for one unperched reason or another specimens tend to show a preference for one in particular. Two tubes of each type of media are inoculated. These are examined after an interval of [illegible] weeks and often prove to be the first to yield bacilli appearing in sputum reported through the media specific confirmation is not a test of virulence. If no growth appears for [illegible] days or if the growth is scanty, the tubes are further incubated and examined after eight weeks have elapsed. Positive cultures are obtained generally at the end of four weeks.

The examination of blood for sedimentation rate is a routine procedure. In simple silicosis the sedimentation rate is not affected, and although an increased rate does not prove the presence of tubercular disease it is very suggestive and serves to direct further investigations. We have found this procedure of value, particularly in the case of men in employment.

Disability.—Perhaps the most difficult part of the problem in silicosis is the estimation of the disability occurred by the individual in the simple form or when it is combined with tubercular disease. I can advance no method for measuring disability for workmen's compensation. Disability must be estimated and, as in other disabling conditions, such estimation is an approximation which will approach accuracy only with experience and practice. In the estimation of [illegible] consideration of the whole clinical picture is of primary importance. Only the trained physician and one prepared by practice [illegible] has any right to attempt such an estimation. All [illegible] should be applied to the case under consideration and they should [illegible] careful attention to what the electrocardiograph discloses for [illegible] indicates in some degree the extent of the fibrosis which has occurred. It has been demonstrated through the injection of the pulmonary artery in excised silicotic lungs that silicosis is accompanied [illegible]

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marked diminution in the bed of the pulmonary artery, and that this bears a relation to the degree of fibrosis.

Frequently the estimation of disability may be narrowed down to the extent. Active tuberculosis immediately serves to elevate the case as a total disability and the demonstration of tubercle bacilli in the sputum has the same significance, at least temporarily. Where less severe disability occurs exercise tolerance tests will be found of some value. It must be remembered however that as applied these tests measure cardiac rather than respiratory efficiency. Nevertheless it is possible through them to get some information regarding respiratory function. Such tests should be as natural as possible. That advised by Dr. Sayers is as useful as any other and is easily applied. It consists of stepping up onto a chair of ordinary or standard height and then down a given number of times. During these exercises, in addition to the observation of heart response, the respiratory response should be gauged. In general it will be found that with exercise the respiratory rate rises and parallels the heart rate at a different level, but after exercise it returns to normal slightly more slowly. Thus if the heart returns to its pre-exercise rate in 2 minutes respiration will steady down in 2½ minutes. In silicotics the respiratory rate increase after exercise will frequently be prolonged up to 5 6 minutes, even though the heart response may have been good. Not infrequently silicotics are unable to complete the twenty chair steps we use in this test because of severe respiratory distress. In applying this test it is of importance that a uniform procedure be used. The twenty steps should be completed within seventy seconds. It is well to restrict the speed of performance to this period of time.

In arriving at a disability rating due regard must be given to other factors than those supplied by the clinical picture and mechanical tests in the specific case. Disability may be related to reduced earning power at a given piece of work or it may be estimated in terms of employability in the general labor market. It will often be found that silicotics in the older age groups are able to perform their accustomed work because they have acquired proficiency at it, whereas they are unable to perform other work involving an apparent comparative expenditure of energy. It is therefore important that due consideration be given to the age of the individual and the duration of his employment in the present occupation. In general it would appear unwise to interfere with occupation in older individuals, unless they show serious incapacity. In severe silicosis however, particularly those showing even slight disability, it may be found advisable to discontinue exposure to silica dust. In such cases assistance is often necessary either by fixing a disability rating on which compensation may be based or by industrial placement or through social organizations to provide work suitable to their condition.

It must be appreciated that the classification of individuals in terms of disability serves no good purpose. For practical purposes those whose disability is assessed at 25%, or less can be classified as having no significant disability. In the medium disability group for convenience disability may be estimated by roughly following groups of 10%. They may thus be classified as follows: 30%, 40%, 50% and so on. In the higher ratings for practical purposes 10% increments may be used. Each individual case however may be considered to differ from the standard. The actual method employed in reconciling estimated disability with [illegible] seems to be of primary importance preceding the method used to arrive at

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Discussion of Dr. Riddell's Paper

Dr. Riddell was asked whether it was his practice to make routine examinations of the urine for silica. He replied that it was not. Such analyses had been made only on selected cases as a research problem and he had reached the conclusion that the determination of urinary silica had no diagnostic value, at least at the present time. The output varied so much with the diet that it was difficult to assess its value in relation to silicosis.

In reply to a question as to the legal and the clinical significance of quantitative and qualitative chemical analysis of the lung ash he stated that lung a lung was routine practice in his department. He thought that quantitative determinations were of little value because no relationship between the amount of fibrosis and the percentage of silica in the ash had been discovered. The test had as much medico-legal value as the determinations of lead in the urine of persons with suspected lead poisoning.

He was asked whether there were any characteristics of the blood that were of diagnostic significance and replied that they had found basophilic stippled cells in the blood of nearly all silicotics. He said in reply to further questions that this finding suggested a toxic action by silica. He had no information as to the number of such cells and had not followed their behavior throughout the course of silicosis.

On being asked whether he had made determinations of silica in the sputum of cases with simple silicosis or silico-tuberculosis, he replied that his own department had not done so but that they had been made in one of the sanatoria in London, Ontario. He did not feel that the results were of diagnostic significance and stated that a man might have plenty of silica in his sputum without ever developing silicosis and, conversely, that the sputum of the individual with simple silicosis might or might not contain silica. Someone commented that an investigator in Chicago had reported that the detection of more than 2 mgs. of silica in the sputum was indicative of probable silicosis. Dr. Riddell replied that he did not believe that this relationship was necessarily true. The quantity present seemed to be related to the recency of exposure. Those in recent exposures tended to have more silica in the sputum than those in whom the exposure was more remote.

Dr. Riddell was asked to amplify his remarks about the detection of tubercle bacilli in the sputum of silicotic subjects and in particular whether he regarded culture as more satisfactory than guinea pig inoculation. He replied that the director of laboratories of the Provincial Department of Health in Ontario had run a parallel series of tests with both methods and had found that more positive diagnoses were obtained by culture than by guinea pig inoculation. Therefore they had standardized on the less expensive method, guinea pigs were inoculated with all growths of acid fast bacilli thus obtained to prove the identity of the organisms. They had found it advantageous to plant each specimen of sputum upon at least 4 different media because some types of organisms seemed to grow better on one medium and some on another. They had been using Petragnani's, Loewenstein's, with and without Na$_2$SiO$_3$ and Wooley's media. Price's medium which consisted of Dorset's egg plus 1% of sodium silicate or silicic acid had received particular attention for a time because it had in their hands uniformly [illegible] growth earlier than controls with the addition of silica. It had not yet been demonstrated that this medium developed any more positive cultures than others but it was a good medium and [illegible] ing used in comparison with the others.

Dr. Gardner commented upon a further study of this problem being carried on by Dr. Vorwald and Mr. Steenken at the Saranac Laboratory. Some years ago Mr. Cummings and Mr. Redlin had shown that finely powdered quartz and colloidal silica added to [illegible] [illegible] a celerated growth but the results were not constant and the amount [illegible] with the silica was not greater than on control media. More recently Dr. [illegible] had demonstrated that on certain media the weight of the growth was at least 4 times as great with the addition of silica as without it.

Dr. Vorwald was asked whether his experiments were made with more than one strain of bacilli and replied that he had used 3 stock laboratory strains, two of them avirulent and one of standard virulence (H. v.). All of these strains had grown luxuriantly on Long's synthetic medium to which particulate silica had been added [illegible] Proskauer and Beck's similarly modified. Mr. Steenken was [illegible] continuing this study in the hope of determining [illegible] anisms involved. The fluid cultures permitted a determination of the amount of soluble silica liberated in the culture as time pro-

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[illegible]d. Apparently increasing amounts were dissolved as the cultures [illegible] in the incubator. The results were not sufficiently defined to report more fully at the time.

Dr. Gardner was asked why the sputum was frequently negative in cases of silico-tuberculosis. He replied that he did not actually know but that the proof seemed indisputable that the number of organisms was decreased in old cases of long standing. He cited 4 sidero-silicotic cases which on autopsy had shown typical gross and microscopic manifestations of associated tuberculosis with large cavities. The contents of the latter had been removed in test tubes at the time of post mortem examination and sent in for guinea pig inoculation. Two animals were injected with each specimen and none of them had exhibited any evidence of infection. Even the diagnostic tuberculin reactions were negative in these animals. These observations proved that the organisms which had once been alive and produced the tuberculosis in the sidero-silicotic lungs had subsequently died. That these were not exceptional cases was indicated by the paucity or absence of tubercle bacilli in stained sections of the lungs of all such lesions and by the tendency for tuberculosis to heal in experimental sidero-silicosis. All of the examples cited were from cases of silicosis modified by iron. Not so many inoculation tests had been made with silicosis of other types but there had been a few and the sections in all of the cases had likewise revealed very few tubercle bacilli.

He felt that there were several possible reasons for the paucity of organisms in the lesions and in the sputum. As [illegible] talk on pathology of the disease, the dense fibrous tissue of silico-tuberculosis had a very limited blood supply and consequently its oxygen tension and nutritive materials were so reduced that bacteria barely remain alive. Bacilli were not often expectorated because most of them were locked up inside the fibrous mass.

Dr. Gardner was then asked whether toxic substances from the bacillus accentuated the reaction of the tissues to silica. He replied that it was apparently true that persons with infection even in latent form tended to react more vigorously to silica, in fact many observers from the time of Watkins-Pitchford to the present day had believed that a certain element of infection was essential to the development of silicotic nodulation. It was the speaker's belief that experimental observation had disproved the latter; he had shown lantern slides of

typical fibrous nodules produced in animals with no trace of infection, by both inhaled and injected silica. He was in perfect accord with the view that infection accentuated the reaction to silica, however, and had many times placed himself on record to this effect. He recalled his experimental observations with mixtures of free silica and other substances and those with silicates where the addition of the infectious factor had resulted in reaction of nodular type, a change which such dusts alone would not produce. The same was probably the case in the experiments reported by Kettle in which silicotic nodules had been produced by the combined action of clumps of dead tubercle bacilli and kaolin.

He reported that Dr. Vorwald was now attempting to determine whether certain chemical fractions of the tubercle bacillus were concerned in accentuating and modifying the response to both free and combined silica. As might be expected the protein elements of the bacillus did not modify the action of free silica; it was presumed that the lipoid component would be more active although this part of the experiment had not yet been attempted. In this connection he recalled the experiments of Fallon dealing with phospholipids, which were discussed at a previous session.

Toxic substances ordinarily liberated from tuberculous foci which gave rise to clinical symptoms and the development of skin hypersensitiveness to tuberculin are held in check where silicosis complicated the infectious process. He mentioned that Dr. Hanlin of Norway, Michigan had demonstrated that not a few hematite miners with very extensive lesions diagnosed as silicotuberculosis had failed to react to an intracutaneous injection of the first [illegible] purified tuberculin (0.0002 mgm) but injection of 0.5 mg of PPD had produced a positive result in most of them.

The reason for the obviously accelerated and accentuated reaction to siliceous dusts in the presence of tuberculous infection had still to be disclosed. It might be a mechanical effect due to obstruction of lymphatic drainage as some writers had suggested. It might be an immunologic reaction of more subtle character. It might [illegible] due to the liberation of non-specific lipoid irritants by both the bacilli and [illegible].

Dr. Riddell was asked to discuss the symptoms [illegible] case with roentgenologic manifestations of advanced silicotuberculosis. He replied that it was difficult to generalize but that [illegible]

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of them whom the Ontario Referee Board had regarded with suspicion had been hospitalized for observation. Some had broken down and developed symptoms of intoxication within a period of 3 months; quite a number remained asymptomatic for two years. Eventually most of them did break. There were also exceptional cases with tubercle bacilli in the sputum who had shown no symptoms, but the same was occasionally true of tuberculous individuals without complicating silicosis. He mentioned that he had examined a man without silicosis, but with an uncomplicated bilateral open tuberculosis who had boxed seven rounds the preceeding night without apparent discomfort.

In reply to a question he said that he had not found morning cough and vomiting a frequent symptom of silicosis. However, it occurred at times.

He was asked how frequently the dyspnoea of the silicotic subject was of neurotic origin and replied that such cases occurred in any group but that he did not encounter many in silicotic subjects. In Ontario the element of litigation did not complicate the picture and hence malingering was uncommon. He did not know of any test to differentiate dyspnoea due to organic disease from that of neurotic origin. Careful study of the individual, in a hospital if necessary, and and evaluation of all the clinical findings was the only method of procedure. In most of the suspicious cases he had been unable to prove the suspected neurotic basis of this symptom.

Dr. Bell nap commented that he had considered the breath holding test of significance in the estimation of disability. He felt that a person who could hold his breath for 50 seconds was not seriously disabled. He asked Dr. Riddell's opinion. The latter replied that the value of any test depended upon the experience of the examining physician. If he had used it long enough he could evaluate its significance for a particular individual. He felt that the breath holding test was useful, and should be followed up.

On being asked for his opinion as to the progression of silicosis after leaving a silica dust exposure Dr. Riddell said that the reaction progressed whether the subject remained in the dust or left exposure. He intimated that lay opinions on this matter had sometimes proved troublesome and cited an experience with 6 cases from which an attempt had been made to prove that miners could continue to work in dust without evidence of progression. The cases had occurred in employees in a mine where the Referee Board had rarely if ever found silicosis from that exposure alone. The 6 men had been classified as silicotic very early in the Ontario experience but he believed that at least three of them never had silicosis and hence their condition could not have been expected to progress. He cited this experience to indicate that the beliefs of employment departments in the industry might be at variance with medical opinion.

He was asked whether he had tabulated the symptoms and clinical signs of the various stages of silicosis as manifested in different age groups and replied that the task was now in progress.

He was asked whether, from a purely medical standpoint and quite apart from the compensation aspect, there were any other dusts whose inhalation reactivated tuberculosis. He did not think there were. He mentioned that his board had examined a number of men exposed in surface jobs to dusts which did not produce pulmonary fibrosis and they had failed to discover an increased incidence of tuberculosis. If such occurred he was of the opinion that there might have been an unsuspected silica hazard. (He did not state whether he had always found it). Even in the case of men exposed to lead, which is reported to favor tuberculosis, he had not been able to show any increased incidence of tuberculosis.

Dr. Riddell was asked to amplify his remarks about the significance of a healed primary complex of childhood tuberculous infection in a silicotic subject. He replied that he believed that such a lesion constituted grounds for rejection for work in a silica hazard because it was so difficult to be sure it had really healed. His opinion was based upon the results of a review of a number of old films of men now at work. He had found that a number of apparently healed primary complexes had subsequently become reactivated with resultant progressive silico-tuberculosis. He also felt that the presence of old lesions of reinfection increased the probability of silicosis. Under comparable conditions of dust exposure the man with a healed infection was more liable to contract silicosis than the one without such complication. He said he would absolutely refuse employment in a serious silica industry to the applicant with demonstrable evidence of healed tuberculous infection.

When asked whether this statement applied regardless of the age of the applicant he admitted that he would be less severe with older persons and when pressed for specific limits, he said that he

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thought the primary complex was probably safe in applicants 35 to 40 years of age or more. In younger individuals he would not hesitate to reject on this finding alone. He admitted, however, that he would not reject an applicant if he had only a positive tuberculin reaction without demonstrable evidence of a primary complex in his lung.

Dr. Sampson then raised the question as to whether the roentgenogram of the chest revealed every primary infection which was responsible for the positive tuberculin reaction. He cited his own 11 year study of school children in Saranac Lake. This group, probably because it was largely composed of children of tuberculous parents, showed a high incidence of positive reactions, 50 to 60%. The incidence of primary complexes visible in the lungs was only 15 to 20%. He inquired whether the discrepancy was due to foci of infection outside the lungs or whether such lesions had been present and then healed to such an extent that they left no visible evidence on the film.

Dr. Riddell commented that this certainly occurred. He had observed a lesion recede and leave a small brilliant nodule and finally disappear.

It was Dr. Sampson's impression, at least in persons not exposed to dust, that the primary complex bore no direct relationship to the adult type of tuberculosis which had developed later. In observations covering perhaps 50,000 adults during the past 25 years, he had never seen disease of the adult type develop in the region of the primary complex. (Dr. Sampson omitted to consider the possibility that the primary focus might have supplied the bacilli which were carried by the blood, or lymph to produce lesions in other parts of the lung. This possibility is a remote one but it should not be overlooked.—*Dr. Gardner*) His observations had led him to believe that most calcified lesions were incapable of reactivation. In substantiation he cited an experience in which an overdose of tuberculin had been administered to a patient with calcified lesions of adult type. A focal reaction developed about the areas of calcification in the lungs of sufficient intensity to be detected in the roentgenogram. But this reaction was soon absorbed and no reactivation ensued. In following the same children over the period of 11 years, no primary focus had ever spread to produce a lesion of adult type in the same area.

In silicotic subjects he had likewise failed to discover any case of tuberculosis which appeared to have developed about a reactivated primary focus, although his serial observations had thus far been limited to a 3 or 4 years study of iron miners. (It might be noted that reactivation would theoretically be less common in silicosis owing to the general tendency toward healing of tuberculosis in the modified form of the disease.—*Dr. Gardner*.)

Mr. Cummings reported that he had compared the incidence of primary complex in iron miners with tuberculosis and in those without roentgenologic evidence of adult type tuberculosis and had found the proportion of the childhood lesions essentially the same in the two groups. If the primary complex were the cause of much adult disease it should have been higher in the latter group. (One might have asked here how many lesions of childhood type are obscured by the wide-spread shadows of adult disease.—*Dr. Gardner*.)

Dr. Riddell was asked whether he had any idea as to the relative number of fatal cases of silico-tuberculosis which resulted from reactivation of preexisting foci of infection and those caused by new infections from without. He said that he had no idea but he recognized that both types might play a part.

He was asked whether the Ontario Referee Board had the power to remove silicotic persons from employment. He stated that the Board could suggest removal but that it had no power to enforce its recommendation. He said that under legislation previously in force in Ontario a man who had developed silicosis was given an award of $500 if he left the exposure. But the scheme failed to accomplish its purpose of rehabilitating the man in a new job. His disease often progressed as rapidly out of exposure as if he had not stopped work, he spent his $500, frequently in riotous living, and then became a candidate for relief. In consequence the idea was abandoned. The impulse to quit work was not nearly as strong today because both the men and their employers had discovered that they could often continue to work after a positive diagnosis had been made. The Board now suggested that only younger men be removed from exposure and their suggestions were generally being followed but there was no compulsion.

Dr. Riddell was asked to discuss the Ontario Commission's attitude toward cases of uncomplicated tuberculosis in industries with a silica hazard. He said that disability from uncomplicated tuber-

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culosis was not compensated in Ontario even when there was a suspicion that dust exposure might have had some bearing on the progress of the infection. Under the law tuberculosis was not a compensable disease unless associated with demonstrable evidence of silicosis.

He was asked whether the Act still specified that in the event a silicotic employee became tuberculous his compensation was reduced. He stated that under the Act as amended in March, 1937, tuberculosis developing within 3 years after exposure to silica dust was considered to be of occupational origin. Before amendment the period was only 2 years. The man totally disabled by uncomplicated silicosis was compensated at 66 2/3% of his wages when last employed. If he should develop tuberculosis in addition his compensation was reduced to 50%, on the theory (originating in the U. S. A.) that 10% of the population would develop tuberculosis regardless of specific silica exposure. A saving feature was a stipulation that no compensation for total disability should be less than a minimum of about $12.50 every two weeks. He commented that personally he failed to see the justification for such reduction.

In reply to a question he said that his Board had the power under the Health Act to force a man with open tuberculosis to leave employment.

Similarly, he said that compensation was granted to the individual who developed silicosis upon a preexisting tuberculosis.

He was asked the attitude of his Board toward pneumonia as a complication of silicosis. He said that he believed the incidence of pneumonia was no greater in silicotic than in otherwise normal subjects but that it was apt to be more serious in the silicotic. It was his impression that the mortality was higher. He admitted the danger of generalization about a disease produced by so many causes, as brought out in one of the previous discussions.

Dr. Riddell stated, in reply to a request, that he had not had sufficient experience with asbestosis to describe its clinical manifestations.

CONTROL — MEDICAL ASPECTS

Dr. A. R. Riddell
Ontario Department of Health

Since this subject was discussed here two years ago much progress has been made in silicosis control, particularly in the United States of America. As elsewhere control methods have been influenced and stimulated by problems related to the compensation of injured individuals. In America occupational disease is at present compensable either through the method of blanket coverage, or under specific schedules in some eighteen states of the Union and in the District of Columbia. Not a few states have made special provision to compensate silicosis. In some [illegible] provision was adopted during the past year. Five states compensating [illegible] disease, namely — Minnesota, Nebraska, New Jersey, Ohio and Rhode Island, do not provide compensation for silicosis. In West Virginia, however, silicosis is the only industrial disease which is compensated. In certain states, where this problem has received favorable attention, the law in addition to providing compensation for those disabled requires the institution of preventive measures. Some provide for a measure of medical control.

It should be realized that the occurrence of silicosis cannot be prevented by any method of medical control, but these methods may postpone its appearance in the individual, and modify its reverse when it occurs. The functions of medical control include the following:

(1) To demonstrate the [illegible] industrial processes, and [illegible] importance in each.

(2) To provide for the selection of working staffs so that unsuitable applicants, particularly those in whom tuberculosis is present, are not placed in dusty exposures.

(3) To detect those already in exposure who develop pulmonary tuberculosis so that such may be removed from exposure and contact with their fellow workers.

(4) To detect the first development of silicosis in the individual worker and arrange his rotation to [illegible]

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gerous work, or his removal from work where this is considered necessary.

(5) To supervise silicotics remaining in exposure at their accustomed work, and finally

(6) To check the effectiveness of general methods of silicosis prevention.

Medical methods embodying these functions have to a greater or lesser extent been introduced and are now operating in all industrial countries. In the past these were generally initiated in connection with mining operations, as silicosis was first recognized to constitute a serious problem in that industry. Since, however, the silicosis hazard has been demonstrated in connection with other occupations such control methods have been more widely applied.

Medical control in silicosis always tends to be linked with arrangements for the compensation of those developing disability, and laws governing the operation of such measures in general recognize this fact. Sometimes medical control is entirely operated through Compensation Boards. As the application of these methods vary considerably, from place to place, it is instructive to consider some specific instances where medical control methods are in operation.

These measures were first applied on a comprehensive scale in South Africa in connection with gold mining operations on the Rand, and the experience there has influenced the institution of similar methods wherever the need for such medical supervision has been recognized. Medical control in South Africa is linked with arrangements for compensation although it actually operates under a separate administrative body from that administering the compensation law. The history of its development was briefly referred to on a previous occasion here and need not be further considered. Let me remind you, however, that it functions under an Act which became Law in 1925. This, although since amended in some minor respects, remains essentially the same as when it was passed. Under the Act two administrative bodies were created—one is charged with the administrative problems arising out of compensation, termed the Miner's Phthisis Board; the other a purely medical body, termed the Miner's Phthisis Medical Bureau, is responsible for the administration of the medical provisions in the Act. These two bodies had been in existence prior to the 1925 Act but at that time a third body, the Appeal Board, was instituted. This Appeal Board was appointed to serve in the capacity of referee in medical matters. They consider only special cases, particularly those arising out of compensation where differences of opinion have occurred. The personnel, [illegible] of the Medical Bureau and the Appeal Board, is on a full time basis. None of its officers is permitted to engage in other work.

Medical control methods in South Africa are operated entirely through the Medical Bureau whose duty it is to examine applicants and decide on their fitness for work as miners, and to conduct periodic examinations on working miners. It also examines all miners, or eligible ex-miners, who present themselves as claimants under the Compensation Act either for simple silicosis or silicosis with tuberculosis. It decides their eligibility for compensation and fixes the disability in each case. In addition the Bureau decides all death claims brought forward by the dependents of deceased [illegible]. In connection with these claims post mortem examination is required when the Bureau considers such necessary. [illegible] in connection with death claims all examinations conducted by the Bureau [illegible] the preparation of a chest x-ray film in the individual case. This film, taken in conjunction with other data obtained through physical examination, is used by the Bureau when cases are under consideration. The decisions of the Bureau, except that they are subject to [illegible] by the Appeal Board, are final.

The initial examination on applicants is rigorous and many of those applying are rejected as unsuitable for work in the mines. Unsatisfactory chests account for 95% of these rejections. [illegible] excluded all individuals who present indications of respiratory disease either past or present, and also [illegible] unusual prominence in the [illegible] prominence may be either general [illegible] applicant for work must be of good [illegible] and in the opinion of the Bureau fit to be engaged as a working miner.

All applicants for work, working miners or claimants for compensation are examined by at least two members of the Bureau but decisions as to fitness for work or eligibility for compensation are arrived at by the whole Bureau or a quorum of the Bureau. Periodic examinations are conducted every six months on working miners. Through these examinations tuberculosis [illegible] detected either separately or combined with silicosis. [illegible] oping tuberculosis in any form are excluded from further under

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ground work and may be debarred from further mining. Miners in whom silicosis in the simple form is detected may leave the mines after being compensated or remain at work. Prior to 1934, when the Act was amended, the majority left the mines as they were then ineligible for further benefits if they continued in exposure and their condition progressed. Since 1934 those affected with simple silicosis are encouraged to remain at work as progression has been found to occur in silicotics whether they remain in exposure or not. Under the South African Act those affected by silicosis associated with disability are paid compensation on a sliding scale. Payments are also made to those who develop tuberculosis without silicosis. These payments are not considered in the light of compensation for occupational disease but as compensation for loss of work. In order to qualify for payments in respect of tuberculosis, not associated with silicosis, the worker must have been in exposure for a period of one year. Prior to 1934 when the Act was amended this period was two years. The change was probably made to facilitate early detection of disease and the removal of those affected from further exposure.

Silicosis is also an industrial problem in Great Britain but the industrial situation there differs from that in South Africa. It resembles rather the one facing us here, where silicosis arises in a variety of occupations scattered throughout the country, and not concentrated as in South Africa in one relatively small area. To meet the situation in Great Britain medical control measures were put in force following an Act passed in 1931. This Act was an Act of Consolidation as the problem of medical control had been separately attempted in connection with individual industries prior to that time.

The 1931 Act provided for the appointment, by the Secretary of State for the Home Office, of Medical Boards to be situated in strategic areas throughout the country. These Boards function under the direction of a Chief Medical Officer whose headquarters is situated in Sheffield. They operate in connection with the Compensation Act of 1925 but their duties are not confined to questions involving compensation. All appointments are on a full time basis. Under the Boards initial and periodic examinations are conducted. The initial examination follows closely that described as occurring in South Africa and the standards required are similar. They are however conducted by a single medical officer. Periodic examinations are carried out at stated intervals and at each of these two medical officers work in conjunction.

In Great Britain tuberculosis is considered the primary problem, and workers in any trade controlled by the Act who develop tuberculosis are compelled to relinquish their work. The Board has discretionary powers in regard to those affected by simple silicosis. Each case is considered separately and the individual may be permitted to remain in, or compelled to leave exposure. Those removed from employment may under certain conditions claim compensation. The decisions of the Board in connection with its findings at any examination, either for employment or the estimation of disability are final. Under the British system individuals who have reached the age of forty or forty five, (the age varies in regard to certain occupations) and have been twenty years in the industry concerned are given the option, in the absence of tuberculosis disease, of remaining at work or claiming an award under compensation. In the case of younger individuals the Board may order removal if it considers this step to be in their interest. No compensation is paid these unless they are considered eligible for such because of disability. Death claims are handled much as under the South African Bureau. Autopsies may be required at the discretion of the Board. In association with the Board arrangements are made for research and observation and through these much valuable information has been obtained since the institution of medical control methods. Under this system since its inception in the neighborhood of 20,000 autopsies have been conducted on individuals exposed to the hazards of dust inhalation.

In the Province of Ontario [illegible] in operation to a limited extent. [illegible] controlled by the Workmen's Compensation Board. Only in connection with the mining industry do the methods in force approach [illegible] South Africa and Great Britain. After the addition of silicosis [illegible] Act as a compensable disease in 1926 examination centres were [illegible] three then principal mining areas, namely, Porcupine, Kirkland Lake [illegible] bury. In 1936 a mobile examination unit was instituted in connection with the newer areas scattered over the Western end of the Province. The increase in this industry during the past few years indicates the necessity for a further appointment. The [illegible]

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under consideration. The three general sheds are supplied with standard x-ray equipment of the stereoscopic type. The mobile unit is served by a portable x-ray outfit. Each unit is in charge of a medical officer on a full time basis and includes a technician and a clerk stenographer. In one area in connection with the medical control methods full time Public Health Nurses have been appointed. These appointments have been made by the industries concerned, and have no direct connection with the official control method. The services of these nurses have proven invaluable and the addition to the usual medical control measures warrants careful consideration.

In addition to the appointment of examining officers a Referee Board, composed of three physicians with special training, acts in an advisory capacity to the Compensation Board. This Referee Board reviews all claims for compensation, supervises treatment, and assesses disability where this is indicated. The duties of the examining officers are similar to those of the English Medical Board and the South African Bureau except that their decisions in every case, other than in individuals who have developed tuberculosis without silicosis, must be confirmed by the Referee Board.

In Ontario compensation is awarded in respect of simple silicosis at 75% a modified scale for silicosis with tuberculosis. No payment is made for tuberculosis when silicosis is not present. All individuals with tuberculosis are removed from underground and none are accepted for work who show any indication of that disease. Rejections on initial examination are moderately high. Tuberculosis is the cause of many of these rejections.

In 1936 an Act to amend the Workmen's Compensation Law was introduced and passed the New York State Legislature. This makes special reference to the compensation, under certain circumstances, of individuals who have contracted silicosis. Under this Act medical control measures have been put in force. These control measures are directed by the Industrial Commissioner, who is been instructed to appoint two or more medical examiners for each of the five districts into which the State is divided for the purpose. The Industrial Board has been instructed to adopt rules of practice and procedure and prescribed methods and standards, under which physical examination, x-ray and other studies shall be conducted. Provision is made for the appointment of three consultants in the employ

ment of the Civil Service. The medical examiners are precluded from being employed by the duties in the employment of the workmen they examine. The consultants apparently serve on a full time basis. It may be presumed that the medical examiners will function in connection with the periodic examination of individuals [illegible] and that the consultants will act as a Referee Board in connection with compensation claims. The Act specifically states that no pre-employment examination is contemplated. This, I think, is unfortunate. It must be remembered, however, that from the legal standpoint the employer must always satisfy the courts. Cases are everywhere found after consideration of what is possible rather than of what is desirable. It will be interesting to watch the operation of this scheme [illegible].

I have referred to a few specific methods of medical control now in operation, not because these are the only ones in existence or necessarily the most effective, but because they illustrate in a general way how the problem has been handled under various conditions. All the methods considered are official and in the best workable they can be expected because the medical aspects of this industrial condition merge so closely with general community interests and Public Health activities. Individual industries have applied [illegible] methods from time to time on a voluntary basis. Some of these have been very effective. Under most schemes devised to the present, particularly as these relate to compensation, the really disabled worker receives consideration at least moderately adequate, but provision for the partially disabled is not so satisfactory. This probably is this so in respect to younger persons who have developed silicosis. Few schemes make definite provision for the rehabilitation of the partially injured who have relinquished [illegible] work [illegible] by compulsion or on the grounds of [illegible] to whom tuberculous disease contributes [illegible] disability, require special handling. They are [illegible] for heavy manual work and this, in most instances, is all they can expect to obtain when thrown on the general labor market.

The Ontario experience in respect to silicosis [illegible] prevention might be interpreted as indicating that [illegible] compensation has been granted [illegible] to benefit [illegible] States to recently published by the Compensation [illegible] every compensation case [illegible] the average [illegible] which compensation was allowed [illegible]

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and the average age at death at 55.6 years. Attempts to trace others admitted to compensation who have died such figures cannot be prepared on those still living. In this group the average duration of life after compensation was allowed was 13.6 years. The experience varies with different industries. In the mining industry, in reference to those who have died, the average age at the time of compensation was 41.9 years and death occurred at an average of 47 years. The foundry worker was admitted at 47.39 years and died at 57.76 years.

General medical examinations are often made which do not include clinical laboratory features and these should be in contrast to examinations made in connection with cases requiring observation and supervision. This is particularly important in respect to those in whom the presence of tuberculosis is suspected for in silicosis control tuberculosis is the real medical problem. Uncomplicated silicosis would be relatively innocuous. In this connection pre-employment examination is one of the most important functions of medical control as it prevents those already tuberculous being placed in silica exposure. The importance of tuberculosis in association with silicosis is illustrated by a review of 491 individuals, claimants for compensation. These included roughly four-fifths of the cases handled by the Ontario Workmen's Compensation Board during the past ten years. Of this 491, 330 were considered compensable as they showed the presence of silicosis. Of these individuals 140 had positive sputum. In addition to the positive sputum cases 29 presented clinical and x-ray indications of tuberculous disease. The known deaths in this series were 123. In 91 the cause of death is known to have been tuberculosis.

The experience with silicosis varies with exposure and no single exposure can be cited as authority in predicting what may occur in another even although the operations may be comparable and the materials encountered similar in regard to total and free silica. This difference in experience is sometimes surprising and it may relate to other substances than silica locally present in the form of impurity. This is strongly suggested in a piece of work recently carried out in Porcupine and reported by Dr. Robson of that camp. His article will appear in a coming issue of the Canadian Medical Journal.

It has been frequently observed that the time required for the production of silicosis in the individual varies in different occupations. This is stressed in the Compensation Board's *Report* previously referred to. It points out that in the mining of base metals among a gang of 24 men working ten years, still only 8 per cent. will show signs of silicosis. In the porcelain industry 9.33 per cent. would be expected to show the same result, while 2.23 would be recognizable in the first five years.

In conclusion it may be observed that silicosis as an occupational condition is declining in importance. Its prevention is not a medical problem. As it becomes evident through the ordinary medical examination that the ultimate result of the continuing silica exposure depends on the clinical course of the tuberculosis rendered probable, the real problem for the industrial physician disappears. Engineers have met the problem successfully, as Dr. Robson points out, by telling us that the occurrence of silicosis in mining operations can be considered to be a still severe outlook during the next fifteen or twenty years.

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Discussion of Dr. Riddell's Paper

Dr. Riddell was asked to discuss his attitude on the mouth breather in preemployment examinations.

He stated that he felt nasal obstruction of a serious degree was a cause for rejection but that slight nasal defects could be disregarded. He believed that a mouth breather was more liable to contract silicosis than an individual breathing normally through the nose. He asked Dr. Gardner to tell the group of some experimental observations now being made in the laboratory.

The latter reported that Dr. Vorwald had proposed to test the effect of mouth breathing by producing a nasal stenosis in guinea pigs and then compelling the animals to inhale dust. He had obstructed the nostrils by cauterization with silver nitrate; as soon as the lesions were healed the guinea pigs with a number of controls had been placed in the hematite dust room. Most of the animals in which the stenosis was nearly complete could not adapt themselves to breathing by mouth. They swallowed excessive amounts of air and died from tremendous distention of the stomach. Others died of pneumonia. Two of them had a complete stenosis of one nostril with only a 1 mm. opening in the other (the maximum diameter of a normal guinea pig is about 3 mm). These two pigs lived a year in the dust room. At the end of this time they were killed and the amount of dust in their lungs was compared with that in the controls. Grossly the latter had much more pigmentation than the treated animal. On ashing the two pairs of lungs the percent of ash in the dried lungs of the two pigs with the nasal stenosis was 24.95 and 22.80 respectively. In the controls the figures were 27.2 and 25.9. The percent of silica showed a great difference. For the stenotic pigs the values were 0.665 and 0.622; both the controls gave 1.135% SiO_2. The significance of these findings in only two animals was unknown. More observations with other species of animals would have to be made.

CONTROL—ENGINEERING ASPECTS

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In all silicosis problems the primary purpose of dust control is to prevent disability. Such being the case, it is obvious that cooperative medical control is of vital importance. Just how extensive this cooperation must be depends upon local conditions and cannot well be formulated in any set of rules.

It often happens that dust control is applied without any clear-cut medical reason. It may never have been proved that the dust is especially harmful but the management finds it is best to keep the working environment pleasant and reasonably clean. Such cleanliness has the added advantage of reducing the possibility of claims for illness caused by dust inhalation and also [illegible] any chance of the firm's being held liable for general pollution of the atmosphere.

The engineer must have some sort of measure for appraising his results. If he seeks to prevent silicosis he cannot wait ten to twenty years to find out whether the working places have been made sufficiently clean for good health. Naturally, he demands figures of permissible dustiness on which he bases his essential calculations. From time to time, he samples the air to determine whether or not his results are keeping conditions up to this arbitrary standard. In many plants and mines today routine dust sampling is carried out in a manner entirely comparable to the sampling of the various products and materials of production. Experience has shown, beyond any reasonable doubt, that the setting up of these standards, as objectives, coupled with routine sampling to determine how effectively they are being followed are necessary steps in any dust-control project.

At the Saranac Symposium of 1935 it was intimated that five million* quartz particles per cubic foot would be a reasonable objective of cleanliness. That figure has been endorsed recently by

* Throughout this discussion it is to be understood that dust samples [illegible] by the impinger method and counts made by light field at approximately [illegible] ters magnification. If other methods are used, the results are converted into conventional impinger units.

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the Medical Committee of Secretary [illegible] Silicosis Conference. Their report goes further and in commending the practical usefulness of some arbitrary standard, the report states that: "This arbitrary standard should be based upon what is believed to be within the limits of good engineering practice and that which, from a medical viewpoint, it is judged will largely control the silicosis hazard for most industrial exposures. For this purpose it has been suggested that the maximum permissible concentration of silica in the air breathed might be expressed by the following formula. Multiply the percentage of free silica by the total particle dust count. If the result is under five million, the condition may be considered permissible. If the result in over five million, the condition may be considered too high. For example, 10% free silica with an average total dust concentration of thirty million particles per cubic foot would give 0.10 × 30 million or 3 million (good practice); 30%, with an average total dust concentration of 50 million, would equal 0.3 × 50 or 15 million (unsatisfactory). Regardless of silica content of the dust breathed, this formula should not be considered applicable to concentrations greater than 100 million particles of any dust per cubic foot of air."

The New York State Department of Labor has recognized the importance of standards of dustiness in the formulation of a new code for rock drilling. This code was published in the May 1937 issue of the Industrial Bulletin of the state. It is based upon numerous technical committee investigations, reports, and experimental studies extending as far back as 1931. The following quotation is taken from the code:

"33.1 – *Application*: – 33.1.1 – These rules shall apply to all rock drilling operations in silica-bearing rock.

"33.2 – *Definitions*: – 33.2.1 – 'Rock drilling' shall mean drilling, cutting, chipping, channeling, broaching or crushing to excavate rock, by means of machinery or hand hammers.

"33.2.2 – 'Silica bearing rock' means any rock formation, natural or synthetic, containing, as a component part, free silicon dioxide. 'Silica bearing rock' shall be divided into two classes:

"Class I – Any rock formation of substantially uniform geological character having free silicon dioxide as a component part which is known from geological studies or otherwise can be shown

by the applicant to the satisfaction of the Industrial [illegible] to run uniformly less than ten (10) per cent by weight, [illegible] formation.

"Class II – All rock formations having free silicon [illegible] component part, ten (10) per cent or more by weight, and all other formations, natural and synthetic, having a variable and unpredictable content of free silicon dioxide.

"33.2.3 – 'Injurious silica dust concentration' shall mean dust produced from drilling silica bearing rock which is in excess of the following values as determined by an approved dust count method:

"Class I – One hundred million (100,000,000) particles per cubic foot of air, as specified in Rule 33.2.2.

"Class II – Ten million (10,000,000) particles per cubic foot of air, as specified in Rule 33.2.2.

"33.2.4 – 'Approved' [illegible] the Industrial Board.

"33.3 – *Dust Control*: – All rock drilling operations [illegible] bearing rock shall be executed so that there will be no [illegible] of injurious silica dust concentration [illegible] atmosphere."

Predictions are apt to bring back ridicule upon the head of the predictor, but in the writer's opinion, the adoption of the National Silicosis Conference rule and the extension of the New York State Code to all dusty operations in most of our industrial states [illegible] to occur within the next few years. After all, both items are the result of years of experience and probably none of our states have better data at their disposal.

The practical significance of these figures to the man operating a dusty process, whether in a mine or in a mill, is about as follows: Uncontrolled dust dissemination, as from the fine grinding of dry rock, can easily exceed counts of a billion per cubic foot, enclosing the process, using water coupled with reasonable hood cleaning will usually reduce such excessive dustiness to well below 100 million. But counts of 5 million or less are difficult to attain without the application of appropriately designed exhaust systems with suitable devices for cleaning the air exhausted.

Only a minute fraction, generally far below 1%, is grinding in within the size range of physiological significance. For example, in

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drilling hand tool the Fisher trap collected [illegible] of dust per day with a full volume of about 5 cu. ft. [illegible] dust over 99%, by count is below 5 microns but less than 1%, by weight is below 5 microns.

A dust collecting plant put in operation recently for a mining company engaged in milling limestone containing zinc ore collects 1.9 pounds of dust per ton of ore milled, the total amounting to 1800 pounds of dust daily. Concentrations in the pipe lines to the collectors are about 12.5 grams per cu. m., of which 81.5% passes through a 400 mesh screen and 99% through 200.

The employer must spend money, then, to prevent the dissemination of a fraction of the product handled. Sometimes this fraction is valuable and pays for itself, but in silicosis problems the dust collected generally will not pay for the depreciation on the plant. Cost of the dust control operation must be written off, then, as prompt pleasanter and safer work-places and reducing the chance of suits for damage suits. How elaborate the plant must be will depend upon the degree of air cleanliness desired.

Exhaust Systems: The Engineering Committee of the National Silicosis Conference dealt with this subject at some length. In its technical aspects, it is discussed in a recent bulletin from the American Standards Association (1), in the bulletin by Bloomfield and DallaValle (2), and in the book by Drinker and Hatch (3).

An exhaust system for dust control consists of several parts which function as a single unit and are interdependent. They are as follows:

(a) Exhaust hoods
(b) Piping
(c) Cyclones or other primary separators
(d) Secondary separators, such as cloth filters or electrostatic precipitators
(e) Exhaust fan.

The first two items, (a) and (b), are rapidly becoming standardized and leave the engineer but little latitude. In some instances, a primary separator (c) is the only air cleaning device needed but generally a final separator is advisable. The final, or secondary unit (d) can be of many different types depending upon the job. The exhaust fan (e) likewise is also subject to considerable latitude in choice.

In several illustrations of hoods it was shown that the hood should be designed to admit only air from the source of [illegible] question. It was shown by contours that it was necessary [illegible] to obtain a velocity of 200 ft. per min. at a distance of [illegible] the face of an exhaust hood used in an immediate emergency for example. In checking up the efficiency of exhaust hoods, it is necessary [illegible] careful sampling procedures, such as the impinger. A [illegible] thoroughly practical dust detector is an endeavor. Only by [illegible] suitable lens, so that the beam can be thrown about and [illegible] spotted readily. Until such obvious sources of [illegible] estimated dust counts, as by impinger, generally will be [illegible] a waste of time to take them.

[illegible] the part of economy, to avoid [illegible] rather heavy metal such as [illegible] used in the [illegible] hoods used in sandblasting work. The expense [illegible] which can be welded, is more than offset by [illegible] resistance to abrasive wear. The design [illegible] cleanouts are all important items which have been [illegible] standardized. The credit for choosing the design of [illegible] of exhaust piping from a rule of thumb procedure to [illegible] engineering is due largely to Hatch and [illegible] tions have already been cited.

There are many jobs in which recirculation [illegible] partially cleaned, air is neither necessary nor desirable. In such instances, collecting efficiencies of 90 to 95%, of the total dust may suffice. Cyclone and similar types of primary separators remove effectively the larger particles and the [illegible] genic significance but they do not operate at efficiencies sufficient to permit the air exhaust to be recirculated to the work place.

Secondary separators, such as cloth filters, either the [illegible] screen type, should discharge air which is sufficiently clean to [illegible] the hypothetical 5 million particles per cubic foot specification. In practice, such efficiencies are not attained [illegible] the cleaning cycle is inadequate or if there are any leaks in the cloths. However, there are plenty of filter installations in which it would be perfectly safe to recirculate the cleaned air. Generally, such practice is frowned upon and considered decidedly risky but if necessary, and if frequent and careful records are kept there is no reason why recirculation is not perfectly safe. In cold climates a considerable [illegible]

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ing in fuel results from recirculation and [illegible] naturally desired by the operating man.

Electrostatic precipitation, as by the Cottrell process, has not been applied extensively to these problems due solely to the fact that the dust recovered is generally valueless. Recently, the Westinghouse Company developed a modification of the conventional high-tension precipitation process and are now applying it in general air cleaning, as for city buildings, but, at this time, the process is too new to permit of appraisal in silicosis control.

Wet collectors, such as those of Bartlett and Snow, of Cleveland, or of Schneible, of Chicago, as well as the wet "rotoclone" of American Air Filters, of Louisville, have some able and ardent advocates. There are many jobs, as in foundries, where the operator is handling mixtures of dust and steam and he is not desirous of recirculating the cleaned air. In such cases it is claimed that wet methods are better than cloth filters, while it is said that upkeep is far less expensive.

The type, capacity, and position of the exhaust fan depend again upon local conditions. It is common in small installations to place the exhaust fan just before the final separator (or upstream). It then forces the dusty air through the cleaning device. Since it handles dusty air, it is subject to considerable abrasion on the blades and housing. It is generally better practice, therefore, to have the fan follow all the cleaning devices, which are then under suction.

Dusty Operations:—In mine work dry drilling, blasting, loading of cars, and subsequent crushing and concentrating operations are all dust producers. Dry drilling is rapidly being displaced by wet drilling or by dry drilling with an exhaust hood leading to a separator of the type first made by Kadco Corporation of New York. It is a fact, but one not generally appreciated, that in mine operations wet drilling alone will not reduce dustiness enough to pass the recent code passed by New York State—the wet-drilling operations must be ventilated. Whenever possible, blasting should be done at the end of the shift, while crushing operations and subsequent dry-separation processes involving the handling of finely ground materials should be hooded and the exhaust air passed through suitable cleaners.

In factories where there are no crushing or fine grinding operations, dustiness can be caused by various processes involving the transfer of finely ground material from cars, bins, bags, or the like.

Sometimes it is due to the final packaging of finished materials as in the bagging of cement, or canning of various powders. All such processes can be kept reasonably dust free. The fact that the dust may be relatively harmless, like cement, limestone, or dolomite, is no excuse for asking men to work in unpleasant dust-laden atmospheres.

There are many plant processes which can be changed in various ways to reduce dust production. Often it is possible to use some material which is not harmful in place of a known silicosis producer. A recent example is the introduction of non-silica parting compounds in foundries. Inasmuch as only the subsieve size dust is respirable, it is sometimes possible to eliminate the excessively fine material and use only the coarser grades. Obviously, a vast number of such process changes or substitutions are possible.

Protective Equipment.—The intermittent use of dust respirators in various jobs and the continual use of [illegible] in sand blasting and paint spraying are now [illegible] Bureau of Mines' specifications for dust respirator performance have been in force for several years. Today, there is a fairly strong trend among all of the state health agencies to permit the use of Bureau of Mines "Approved" respirators only. [illegible] drastic action than is necessary but no one will dispute the fact that the introduction of Bureau of Mines' approvals has had a very beneficial effect upon the manufacture and use of respirators.

At the present time, the Bureau of Mines' approval specifications for air-supplied masks have not appeared officially but it is understood that the schedule will be printed shortly. As is the Bureau's custom with such schedules, the informal draft of the new schedule was circulated freely among all interested so that the final draft is sure to meet with the same support which followed publication of their approvals for gas masks, hose masks, and dust respirators. It is understood that the protective equipment code of the American Standards Association will use to the fullest extent the respirator and air mask codes of the Bureau of Mines.

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Discussion of Prof. Drinker's Paper

Prof. Drinker was asked what happened to recirculating air when a filter broke. He replied that it was his practice to see that the manometers on the dust collectors were read at frequent intervals and hence a broken filter would not be overlooked for long.

He was asked whether electrical precipitators did not have to be cleaned so often that they were impractical and replied that they were designed to operate in low dust concentrations.

In reply to a question as to the relative cost of wet collectors and cloth filters, he stated that the initial cost of the two types of apparatus was very similar. The operating costs varied with the charges for water in different localities but he added that expense could be saved by recirculating water.

ADMINISTRATIVE ASPECTS OF SILICOSIS

Mr. D. L. Cleveland
Sanasa Laboratories

The problem of silicosis presenting the spectre of disability and death to a [illegible] Old [illegible] workmen's compensation [illegible] complexities to doctors, engineers, lawyers, industrialists and public health authorities. Dusted into prominence in the U. S. when fanned by the ill wind of depression after having smoldered peacefully for decades. The conflagration that resulted served to illuminate the pot of gold at the foot of the rainbow for certain of our brethren but cast deep shadows of doubt and fear across the rest of the interested multitude. The speech was brilliant and awe-inspiring while at its height but now [illegible] down to charring embers the reins shou[illegible] for their whole account worth clanging while glass are perfected for a new structure which will prove less combustible. The fire, such which the blaze flared when finally touched off can be partially attributed to the general resentment which had accumulated over many years from the failure of managements to acknowledge, record against or compensate for the disease.

No doubt the failure of managements to act constructively in the matter of silicosis was due to deplorable lack of knowledge concerning the condition itself, or even worse, to misinformation. Though these conditions still exist there fortunately [illegible] every indication that the light of understanding is penetrating even the most obstinately dusty executive atmospheres.

Now there looms the danger that a satisfactory solution of this perplexing problem may be delayed by too much administrative zeal, particularly from the hasty and poorly devised efforts of [illegible] after a brief introduction to the [illegible] panaceas to legislatures, industry, labor and [illegible].

In order that a practical procedure may be evolved for a [illegible] this matter, it is essential for the executive to [illegible] of the principal groups involved in the problem. [illegible] positions briefly.

Labor seeks protection from a proved and [illegible]

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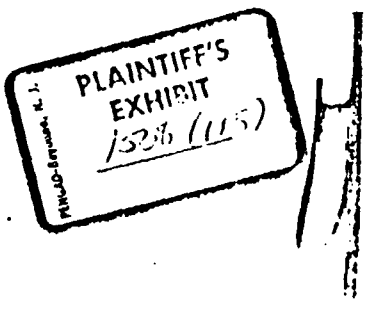
disability should be incurred in the future from harmful occupational exposure, compensation is sought on the same basis as is generally granted for industrial accidents. If a workman is silicotic but not disabled he does not wish to be discriminated against in his search for employment. Labor also finds it difficult to understand why any man who has worked during the past years in a hazardous occupation should not receive as much compensation for total disability contracted at his job as a new employee would receive for the same degree of disability incurred through accident. From the viewpoint of social justice, these issues appear fair and reasonable but the train of economic and legal procedures which they set in motion make their attainment very difficult, if not impossible. Furthermore, Labor's strong position has been undermined by several of its own foibles. In many instances workmen have loaned themselves to the racket street thus obscuring just claims in a host of false ones. Labor has been provoked into opposing physical examinations by the unreasonable attitude of certain employers and insurance carriers. Labor's demands before compensation boards, courts and legislatures have frequently been preposterous and unjust. They have not always been willing to cooperate in efforts to devise a reasonable solution of their problems but have resorted to retaliatory devices with which all are familiar.

Industry seeks always to equalize these barriers to competition represented in the form of added costs incurred in those states which provide for occupational disease benefits. In certain instances these costs are high enough to force the industry to shut down or move, thus bringing an even greater injury to Labor than the silicosis hazard. The average employer wishes from now on to assume responsibility, within his economic limitations, for just cases of occupational disease so long as he has some assurance that he can avoid unfair claims or an inception of the silicosis racket. He feels, however, that it is unfair, and in fact impossible, for him to assume full liability for all the disability or damage that has occurred when he was unaware that a hazard existed or, if aware of it, not required to create a reserve to meet it. He wishes to protect his company hereafter requiring preemployment examinations and rejecting all candidates who do not meet his specifications. Moreover, he insists on his right to make periodic examinations of his present employees so that he may control the development of disability and limit his future liability. He is usually willing and anxious to institute reasonably pre-

ventive procedures against the dust hazard when convinced that his plant or mine is involved. He always wishes to be able to assess his occupational disease risk. He has weakened his position in the past by assuming a negative attitude in the matter of recognizing, preventing or compensating for silicosis. He has been so much inclined to view the whole thing as just another racket and has resorted to legal subterfuges when a forthright policy would have been far more desirable. He has frequently discharged old employees on pension, such his insurance carrier he has rejected silicotics, thus penalizing them to better retain assets. He has been filled with fear over the situation and has remained secretive or inactive. Legislative agitation should precipitate a flood of disastrous claims. He has not provided sufficient reserves to meet this contingency, though he is doubtful if they could have been calculated. He frequently resents or deplores the age in which he lives, with its social responsibilities and is inclined to the belief (not without reason) that it is more exacting on industry and his species in particular.

The insurance carrier [illegible] all the same fundamental method of computing the liability which [illegible] endeavors to cover any given risk. It hopes to make physical examinations as well as studies of the working environment and labor records to arrive at an estimate of the risk involved [illegible] as sufficient experience has accumulated to provide a basis for estimating. In the normal course of events, the insurance carrier should not be more concerned with silicosis than with any other hazard, as most of which it calls a matter of experience and [illegible] with incalculable potential liability [illegible] quently forced to insist on physical examinations [illegible] determining the risk and rate. Insurability becomes [illegible] upon the rejection of all cases which were similar to those granted awards by courts or commissions (and these cases are [illegible] what we would now regard as practically normal) or [illegible] were made prohibitively high.

As a result the insurance carrier forced [illegible] employers and a vicious circle of events followed, the [illegible] could hardly be anticipated again in several centuries. [illegible] created by the fact that most modern [illegible]



simple insurance but a combination of insurance with accident and disease prevention service added. This latter service is frequently very valuable but its cost is simply combined with the insurance rate and the employer does not know which he is buying or the cost of either service separately. This condition produces a relatively inflexible rate structure which confuses and antagonizes the employer. In settling claims the insurance carrier usually follows the most expedient financial procedure which brings upon him the charge of being unfair to the injured workman.

PHYSICIANS wish the right to examine workmen whenever it appears desirable in their opinion to do so and with any technique which they deem most appropriate to ascertain their condition. They wish to publish their findings so that appropriate criteria may be established for the diagnosis and treatment of the conditions discovered. They believe in regulating the hazard by controlling the physical condition of the workmen. They insist that all medical questions before adjudicating bodies should be decided by medical men. They wish to make post mortem examinations of all adjudicated cases compulsory, so that clinical and roentgenological data may be [illegible] correlated with the pathological findings. Their position is [illegible] by the considerable number of unethical members in their profession who have loaned themselves to the market and by the sincere but uninformed members whose opinions or testimony on important issues is frequently at variance with the latest beliefs of the experts. The fact that medicine is not, and never can be, an exact science mitigates their usefulness in situations where an unqualified direct statement is necessary. The consequent desire of capable and fully informed doctors to escape cross examination embarrasses the fair administration of many silicosis claim cases. The highly developed ethics surrounding the profession are frequently confusing to the laymen and often misinterpreted.

ENGINEERS wish the right to examine the industrial environment of the workmen and to compare their findings with those of the physicians so that they may arrive at suitable standards for the measurement and control of silicosis hazards.

They wish to control the hazard by removing harmful dust from the breathing zone of the workmen. It is their desire to have uniform and compulsory practices and equipment for safeguarding the occupational environment of the workmen based on the standards which they have formulated from their investigations. Their greatest weakness arises out of their innate tendency to regard themselves as better doctors than the M.D.s. They have great difficulty in agreeing on proper standards among themselves and yet they insist on placing these standards in codes and laws when the techniques at present in use are not always as exact as those used by the physician. Above all else they err in the firm conviction that the silicosis problem can be solved by dust control alone and they advocate ideal conditions which tend to misinform workmen and to discourage employers because of the elaborate and expensive procedures required.

LAWYERS are difficult to include in any generalization. It would appear, however, that as time passes an ever greater number of them favor some form of statutory provision for silicosis. Compensation appears preferable to common law action for damage from silicosis in their opinion because of the nearly insurmountable difficulties involved in arriving at specific decisions to be established as precedents in handling such cases in civil courts.

When compensation laws are enacted they wish to provide certain restrictions relating to employment in the state, or specific time limitation for the filing of claims, [illegible]. Many would like to provide for a review of facts as well [illegible] fore a higher court than the compensation commission. [illegible] ness lies in the fact that the law is sometimes an ass, and [illegible] appears to suffer in their hands. They have an unexplained propensity for settling certain cases in which the issue should be determined by due process of law or for forcing a legal point when [illegible] should appear [illegible] have been [illegible].

INDUSTRIAL COMMISSIONERS [illegible] table procedures for determining the [illegible] arriving at an equitable estimation of disability arising out of the condition. They seek to administer a law with [illegible]. For this reason they would welcome definite medical opinion which could be reconciled rather than those which appear [illegible] opposed. If they can secure a fair and honest record [illegible] that they will render a fair and honest decision. [illegible] usually the sum of all the others which have been [illegible] either in the record of a claim. They are sometimes [illegible] widely swayed by political breezes and frequently [illegible]

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formed on silicosis because of their short tenure of office. They occupy a difficult role and it seems improbable that any human could fill it to the satisfaction of all others.

PUBLIC HEALTH AUTHORITIES are interested in fact finding so that they may formulate and promulgate health and safety regulations which will aid in preventing the spread of infection to others and in securing the control of dangerous occupational environments. Their efforts are devoted to inspection of hazardous industries, arranging for correct reporting of disease, enforcement of regulations embodied in codes or laws and education of all those vitally concerned with the hazard. Their weaknesses may usually be attributed to a political environment or to such inherent difficulties as they encounter in the law and which must be administered to the best of their ability notwithstanding.

RESEARCH AGENCIES offer the only reliable means of solving the [illegible] perplex and confound us in dealing with this awkward problem. They need data of every kind as well as trained technicians and elaborate facilities for their investigations. Their chief weakness springs from the confusion which is created by the contradictory findings they sometimes report (as the sericite controversy). Time eventually evaluates their work and with every addition to our basic knowledge just administration is brought nearer to realization.

* * *

Having considered each of the various groups involved in the silicosis problem, together with their conflicting attitudes or functions, it becomes apparent that no simple solution is likely to be found and that compromises are inevitable. There are, however, certain general requisites for the successful administration of this matter which may be outlined as follows:

First, reliable procedures should be utilized to disclose whether or not a given individual, occupation, process, plant or industry suffers from the disease. Second, if the disease is discovered or suspected after a competent investigation, its extent and specific characteristics should be determined by detailed study. Third, procedures intended to control the hazard should be designed to fit the requirements revealed by the specific study. Fourth, a suitable supervisory mechanism should be constructed to maintain adequate control over potential hazards and to administer equitable distribution of benefits to those injured by the disease. These four principles are equally applicable in dealing with a single silicotic, an occupation, an industry or any of the claims before a court or commission.

Administrative officers concerned with silicosis have several methods of determining the possible existence of a hazard in dusty industries. Physical examination, including stereoscopic roentgenograms, on old employees in various suspected occupations may be relied upon when they reveal definite nodular fibrosis in the lung tissue. Since it is frequently difficult to obtain satisfactory roentgenograms in isolated and small industries or to make selective examinations except under other pretexts, post mortem studies on old employees dying from any cause may be a more desirable means of securing the required information. Even in the event that these procedures can not be utilized there remains an indirect but reliable plan in the form of mortality investigations. Official records should be searched for all the data recorded on individuals who have worked in the plant under study and who have died during a ten year period. For the years from 1928 to 1935 there were about 9 deaths per year among 1000 men representing the average of all gainfully occupied males from 15 to 64 years of age. During the past eleven years there should have been about 100 deaths among 1000 men gainfully employed. These 100 deaths should be distributed among several causes about as follows:

Heart	19		
Tuberculosis	10	Tuberculosis	10% of deaths
Cancer	9	Pneumonia	8% " "
Pneumonia	8	Total Respiratory	18% " "
Nephritis	6		
Cer. Hem.	5	T[illegible] Cardio[illegible]	
Suicide	4	[illegible]	
Accidents	3	[illegible]	

The influence of color can be judged [illegible] for tuberculosis mortality among males in 1925.

Age	Negro Rate per 100,000	White Rate per 100,000	Ratio Colored to White
15-19	235	40	[illegible]
20-24	382	81	[illegible]
25-34	382	98	3.9
35-44	296	118	2.5
45-54	[illegible]	[illegible]	[illegible]
55-64	261	135	[illegible]

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The following nationalities tend to have a high tuberculosis mortality:

Mexicans	Irish
Finns	Greeks
Swedes	

If more than the average number of deaths from tuberculosis has occurred among the workmen under study over this period, it is reasonable to suspect the existence of a specific hazard. Moreover, it would be prudent to suspect such a hazard when deaths from all respiratory diseases exceed the average since tuberculosis reporting is not always as accurate as that for all respiratory infections combined.

If any danger is suspected from these preliminary investigations, the executives concerned with this problem should inaugurate a thorough survey of the operations under their supervision to determine more exactly the existence, nature and extent of the hazard. A survey of this type should include the physical and x-ray examination of all the employees, the taking of occupational records, the determination of the nature and concentration of the dust suspended in the working atmosphere, an analysis of the mortality records in the district, and where possible, post mortem examinations of old employees dying from any cause.

If no hazard is discovered by such a survey, both the employer and the employee are protected by the information secured; on the other hand, if one is detected, the original study serves as a guide to the future program of control which should be promptly initiated. A control program in a dusty industry whose hazard has been defined would include the reduction of harmful dust concentrations in the working atmosphere, the application of protective devices and procedures, the establishment of certain physical standards for new employees, the selective placement of old employees on the basis of their physical condition, and the adoption of an educational program to inform all those vitally concerned with the effects of a dust hazard.

Reliable methods for determining dust concentrations are available, and engineers equipped to utilize these procedures can [illegible] the sources and extent of any contamination and devise means of either reducing the hazard to within safe limits or applying adequate protective devices. A constant check of the dust concentration in

the plant should be maintained, and records should be kept [illegible] the condition of the working atmosphere, the nature of the protective equipment or procedures in use, and the total amount of time each employee is required to spend [illegible] occupation.

Pre-employment examinations including an occupational history and excellent stereoscopic chest roentgenograms should be given each new employee. In general these examinations will serve to classify men into three main groups composed of those who [illegible] are apparently normal, those who have an occupational fibrosis without any other pulmonary abnormality, and those who have tuberculosis or other pulmonary abnormalities either with or without occupational fibrosis. In considering the desirability of employing men whose chests are apparently normal, it should be emphasized that young men up to the age of thirty or thereabouts are not altogether perfect risks for employment in a hazardous dust since they are in an age period during which they are most likely to contract tuberculosis. In the speaker's opinion, men who show evidence of simple occupational fibrosis or silicosis and who are past the age of thirty, as most of them will be, are fair risks for employment, since ordinarily uncomplicated silicosis is not regarded as disabling, and since these men are past the age period during which they are most likely to develop tuberculosis. Men in this group may [illegible] progression of their fibrosis after continued employment but they will not tend to develop disability if they have been rigidly examined to exclude those showing any x-ray evidence of tuberculosis or other pulmonary injury.

If this class of persons is [illegible] most unfortunate social [illegible] capable of a lifetime of [illegible] become embittered and the prey of [illegible] tors who further complicate the situation by [illegible] and neurotics of valuable workmen. These individuals should not be eligible for compensation, but if they are made to [illegible] then legislation should also be provided to [illegible] their right to compensation for an occupational [illegible] they may have the privilege of working at the task [illegible] are best suited. Providing this group [illegible] infection, and is kept under medical supervision [illegible] their experience should be favorable. As to the [illegible] an injustice is done both to the [illegible]

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ployee in placing at work in a hazardous dust any person who has tuberculosis or other significant pulmonary abnormality, whether or not occupational fibrosis or silicosis is present.

In the presence of a known dust hazard old employees should be placed, so far as possible, in those occupations most compatible with their health and welfare on the basis of the physical examinations which they have undergone. Men with normal chests, who are past the age of thirty, or those with silicosis may be employed at any occupation where the dust concentration is within the limits dictated by reasonable control; while young men under thirty should not be exposed in those operations which produce the most harmful dust and should be re-examined fairly frequently. Those cases of inactive tuberculosis, either with or without silicosis, should be carefully evaluated and every effort made to select for them occupations such that their health will not be impaired or their ability to earn a living wage interfered with. Men in this group should also be re-examined at frequent intervals. Cases of active tuberculosis should not be employed in a dangerous dust either for their own safety or that of their fellow workmen, and should be encouraged to enter a sanatorium or to place themselves under the care of a competent physician.

The occupational disease problem is similar to that presented by industrial accidents when the first compensation acts were passed. Disabling conditions created or aggravated by exposure to harmful dust can be prevented or controlled. The success of any plan designed to accomplish these purposes will depend upon maintaining a broad educational program similar to that adopted in the "Safety First" movement. These policies are not only humanitarian, but they are practical and have been placed in operation in mining districts employing thousands of men. May I sincerely recommend them for your consideration?

Discussion of Mr. Cummings' Paper

Dr. Riddell congratulated Mr. Cummings on his presentation and stated that if the members of the symposium had heard no other paper they would have received a point of view on silicosis which was absolutely invaluable.

Mr. Cummings was asked whether the water blast, which he had described for mines, would dissolve poisonous gases. He replied that it would and that one could enter the working places with safety within a few minutes after blasting. He stated that the water curtain was about 60% efficient in removing dust from the atmosphere.

Asked whether the humidity created a health problem he replied that there might be harmful effects, but that as a general rule the men had not objected as long as the temperature had remained moderately low. He called attention to the fact that in many mines the humidity was high even when dust preventive measures were not in use. Deep mines and those which were burning were usually hot. When the temperature rose to 90° F. or more, high humidity rendered the atmosphere too uncomfortable for work. Mines of this type could be worked by cooling the incoming air.

Mr. Johnson of the American Mutual Liability Insurance Co. said that the hazards discussed by most of the speakers had been confined to those that were generally recognized as causes of silicosis. Experimental studies had been described which dealt with mixed dusts but no one had mentioned the [illegible] in places where such mixtures [illegible] mixed dusts were usually encountered and [illegible] discussion of the hazard in this industry. He [illegible] that the opinion was prevalent among lay groups that employment in a foundry necessarily implied development of silicosis. He asked specifically whether anyone could draw any comparison between the general hazard in the foundry and that in specific occupations, like sand blasting, which were recognized as hazardous.

The chairman replied that it was now coming to be recognized that the foundry did not create as general a hazard as that created by an industry like mining. In his opinion the dust hazard in the foundry had been greatly over emphasized. He called on Dr. [illegible]

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Director of the Tuberculosis Division of the New York State Department of Health, to express his views.

Dr. Siegal replied that about a year ago the Division of Industrial Hygiene and Health Department had undertaken a survey of the foundry industry in the State of New York. It was estimated that about 12,000 men were employed in 200 plants, representing chiefly iron and a few non-ferrous foundries. The survey included 50 to 60 foundries of all sizes, from those employing 4 or 5 men to those with over 300 employees. An engineering survey of the dust concentrations and general house-keeping conditions was made; a complete occupational history and a flat (chest) film of every employee was taken. The films had been interpreted by himself, by another physician in his department, by Dr. H. L. Sampson, and by two other recognized roentgenologists. Where there had been differences of opinion that of the majority had been accepted. In the main the conclusions of Dr. Sampson had been substantiated.

Approximately 5000 men were examined. The tabulation of the results had not been completed but they had failed to find evidence of the hazard which they had anticipated. Neither tuberculosis nor nodular silicosis were unduly prevalent. They had been surprised to find silicosis where they had not expected it, and in places where they might have expected to find silicosis, they had often seen little or none. They had been forced to conclude that silicosis in the New York State foundries, at least, was far from being the problem it had been reputed to be.

The chairman mentioned that he had been present when many of Dr. Siegal's films were reviewed and had been impressed with the paucity of cases of silicosis with or without evidence of coexistant tuberculosis. He was struck by the fact that the evidences of nodulation that were discovered occurred in men of advanced age who had had exceptionally long periods of employment. He then asked whether Mr. Johnson would care to comment on the foundry situation in Massachusetts.

Mr. Johnson replied that he hoped that the number of claims from the foundry group was not indicative of the severity of the hazard in this industry. There had been a half a dozen foci from which the number of claims indicated a legal rather than a health hazard. Such claims arose from loss of employment because men had been discharged for real or suspected evidence of silicosis. Disability had not entered the picture. He cited a motormen's record in a certain foundry where of 41 who were discharged, 40 were subsequently shown to have no disease.

He had been at a loss to correlate the apparent dust hazard in certain foundries with medical findings in the employees. He recalled one pipe foundry which appeared to be as dusty as any of those described by Prof. Drinker. A complete medical examination had not been made on the men but an examination of the records of a very efficient employees benefit association had shown no excess of pulmonary disease over a period of 25 years. Subsequent examination of the dust had disclosed no free silica, only clay, in the atmosphere.

His company had had about 5000 flat films made of workers in the foundry industry. These had revealed an occasional case of conglomerate fibrosis, probably due to associated infection, but a few of simple discrete nodulation. There were also a few instances of uncomplicated tuberculosis. The incidence of pneumonia [illegible] seemed to be no greater than normal. This he could [illegible]

The chairman commented that he had seen many of the films of the foundry workers taken by the Massachusetts State Department of Health and had again been impressed by the paucity of cases with advanced nodulation. He agreed that they were usually of the conglomerate type whose infectious etiology Mr. Johnson had mentioned.

Dr. Riddell stated that in his experience the incidence of silicosis varied widely from foundry to foundry. [illegible] in general the foundry industry was [illegible] as far as silicosis was concerned. [illegible] from which he expected to [illegible] the next 10 or 15 years. He believed that [illegible] programs now inaugurated and the substitution of non-silica parting compounds for silica would [illegible]

The chairman expressed surprise that Mr. Johnson had failed to discover evidence of an abnormal incidence of pneumonia in foundry workers because others had reported quite contrary findings.

Mr. Cummings stated that the foundry experience presented a confusing picture. A dust hazard was encountered in [illegible] steel and brass foundries, but in other foundries [illegible] little evidence of silicosis in the men even though the plant was

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be dusty and the processes might seem dangerous. It was his belief that there was a high incidence of pneumonia and that the infection was to possible for the unfavorable opinion which was prevalent. He felt that the high incidence of pneumonia was not attributable to dust in the atmosphere of foundries but to wet clothing in temperature. He recalled that granite cutters, who worked in the open and were exposed to considerable quantities of harmful dust, developed silicosis with associated tuberculosis but they did not suffer unduly from pneumonia. Soft coal miners, who were exposed to coal dust underground did not develop silicosis nor have a high mortality from tuberculosis because there was so little silica in the dust they inhaled, but they did suffer from an excess of pneumonia. Workers in steel plants, who were not exposed to dusts but who experienced frequent and marked changes in temperature, were also subject to pneumonia but not to tuberculosis. He confirmed these statements by statistics quoted from Jessimine Whitney's monograph, "Death Rates by Occupations" published by the National Tuberculosis Association.

In his opinion the foundries had been paying many unjust claims for silicosis. He thought that the industry had not been given the sympathetic attention which it might otherwise have expected because it was common knowledge that foundrymen suffered from some form of respiratory disease. Foundries could and should protect their employees from marked changes in temperature. If this were done their high pneumonia rates would fall and the concern over all respiratory diseases would soon be forgotten. He believed that much of the disability and many of the deaths among foundrymen now attributed to silicosis and tuberculosis were in fact due to pneumonia.

Mr. Cummings was asked whether records of absenteeism were not a valuable adjunct in evaluating an industrial hazard. He replied in the affirmative but regretted that formerly not many industries had kept such records.

Mr. Stratton of the Travelers Insurance Company raised the question of the disposition of men unemployable by virtue of the standards of health that had been proposed. He felt that only about 10% of employees could qualify for work under such rigid specifications. Mr. Cummings replied that there was always debate on this point. It was his belief that if more than 5% of applicants

were rejected in preemployment examinations the purpose of medical control would be jeopardized. Employers should be encouraged to accept some medical risks. He was convinced that it was a good policy for the employer to hire some men [illegible] both these individuals and their [illegible] employer was not [illegible] to the employer was small for such men were [illegible] under medical supervision to detect [illegible] stood an excellent chance of working for many years. A person with silicosis was apt to be one who had acquired special skill by virtue of long employment and hence, if he had no infection, he would be more valuable to the employer than an untrained man. He gave an instance which could befall a silicotic was to be no, but his pal [illegible] blacklisted he could not find work anywhere and [illegible] that the social problem thus created would have much wider implications than that of the disability in a few individuals. Compensation claims would seldom be filed if workmen were sure that they would not lose their jobs.

Mr. Cummings was asked why he would [illegible] young men in industries which had reduced dust concentrations to levels which were considered safe. He replied that [illegible] man was more prone to develop tuberculosis there might be a suspicion that the infection had been activated by the [illegible] of its concentration. While most persons would accept [illegible] quantities probable had little or no influence on the [illegible] no one actually knew how much silica dust was required to [illegible] pulmonary tuberculosis which might otherwise [illegible]

Mr. Stratton inquired as to [illegible] by the use of a siliceous parting [illegible] that some of the older compounds were [illegible] but that foremen had always been loath to give [illegible] satisfactory material. Since reliable non-siliceous compounds [illegible] available, foundry operators and insurance carriers [illegible] upon their use.

Dr. Sappington pointed out that one feature of the [illegible] had not been mentioned, namely the [illegible] persons qualified by training and experience to [illegible] groups of prevention that had been described. It was [illegible] that much of the work now being done in the [illegible]

PLAINTIFF'S EXHIBIT 1573 (21)

hands of persons who had had little opportunity to acquire accurate information. It was his belief that not for another generation could one hope for an adequately trained personnel.

The chairman remarked that all members of the symposium had a responsibility to see that information was distributed by every available means. The elements of industrial hygiene should be taught in all medical schools since most of the graduates were to practice in an industrial population. The specialists who were to fill positions in industrial plants and on the regulatory state and city boards would have to be trained in post-graduate courses offered by the universities.

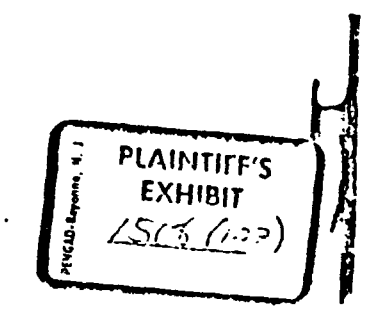
LEGISLATIVE CONTROL AND COMPENSATION

Mr. T. C. Waters,
Occupational Disease Commissioner, Pittsburgh, Pa.

During the present week those of us who are interested in silicosis have been privileged to participate in the most elaborate round of the medical and engineering phases of that subject that we have heretofore enjoyed. All of us are indebted to Dr. Gardner and the officials of the Saranac Laboratory for the opportunity to attend this symposium. We have met with and have been instructed by those who know most about the medical and engineering aspects of silicosis, and I believe that we now have a better understanding and appreciation of the intricacies of the problem that it presents in modern industry.

I do not believe it is amiss to emphasize the importance of the work that Dr. Gardner and his associates have been carrying on here at Saranac Lake. Through these efforts the medical profession generally has come to a greater knowledge of the diseases, peculiar upon workers exposed to silica hazards, the types of disability that such workers may suffer, and the development of complicating infections. We have been impressed with the fate of those employees who may be its victims and are looking forward to the day when industrial hygienists may find some solution to effect as full and final control and cure. That of itself offers [illegible] search and study but [illegible] answered.

This Symposium has afforded to us [illegible] our thoughts and express our views totally independent of our professional or business affiliations that we may have. We are not here to speak for any class we are concerned with the various aspects of the silicosis problem and may express what we will in our individual capacity, not as the advocate or spokesman of any special group or interest. Only in that capacity are we able to [illegible] cess for the purposes that we seek to accomplish. It is the characteristic that has signalized the success of the [illegible] have met, either here or elsewhere. I have in mind [illegible]



there is less disabling silicosis in industry than was popularly believed, so that the industrial hysteria existing a few years ago has been gradually dissipated.

In considering any program to provide compensation for silicosis injuries, there are various subjects that merit particular attention. At this symposium primary emphasis has been placed upon the medical and engineering phases of the problem, and the following subjects relating thereto are particularly pertinent in the discussion of legislative provisions dealing with compensation and control:

1. Compensation for Partial and Total Disability.
2. Potential Liability of an Employer for Silicosis Injuries Existing at the Effective Date of the Law.
3. Pre-employment and Periodic Post-Employment Physical Examinations of Employees; Autopsies in Death Cases.
4. Creation of a Medical Board.
5. Prevention and Control.

As evidence of the importance of these subjects, there have been instances where silicosis compensation acts were passed, failing to give consideration to the particular problem presented thereby, resulting in the shut-down of industries in such states, cancellation of compensation insurance risks, creating unemployment problems that have been most serious. As an example, I would refer you to the industrial condition existing in New York State pursuant to the passage of the General Occupational Disease amendment to the Workmen's Compensation Act, effective April 8th, 1935. This amendment added to the schedule of compensable diseases the following:

"Item 28. Any and all occupational diseases."

By its general terms this included the disease of silicosis, without giving consideration to the subjects above referred to. There followed the cancellation of insurance policies in those industries wherein silica hazards existed; manufacturers turned to the State Fund, and the rates for silicosis coverage advanced unreasonably. Many industries were forced to shut down and migrate to other States, while countless men were thrown out of employment. The situation became so serious that in the spring of 1936 a Commission was appointed, with representatives of both employers and em-

ployees, for the purpose of drafting a [illegible] Law, making special provisions for silicosis compensation. The result was the proposal and enactment of a Bill, Chapter [illegible] of 1936, effective June 6, 1936. The [illegible] relating to the subjects above referred to, and, according to information, has been administered satisfactorily, [illegible]

Each of these subjects, due to its importance, will be [illegible] individually.

1. *Compensation for Partial and Total Disability.* [illegible] of the disease of silicosis is unique and unlike [illegible] tional diseases or traumatic injuries that may happen to [illegible] Many silicotics are presently employed in trade, not knowing of the fact of their affliction, demonstrating no noticeable or demonstrable disability in the particular occupations in which they are engaged. Other diseases present immediate manifestations, symptoms and disabilities, but the development of silicosis in the lungs of an employee suffering therefrom is unaware of its existence until the infection renders him disabled. Several of the States that have recently enacted silicosis laws specifically deny compensation for partial disability (New York, Ohio, Pennsylvania, Michigan). The medical authority for such provisions, I quote the following statement contained in Medical Series Bulletin No. [illegible] Hygiene Foundation of America, Inc., upon whose [illegible] mittee have served several of the distinguished doctors who addressed us during the present meeting:

> "Simply that is, uncomplicated [illegible] industries in the United States [illegible] vere disability."

and the following:

> "It should be stressed that a demonstrated silicosis does not necessarily mean disability, and therefore a complete diagnosis should state whether disease is present and [illegible] for work and, if possible, how much. Determining this is still a most difficult problem."

Considered in their practical aspects, the [illegible] that award compensation for partial disability [illegible]

The Pennsylvania law was passed [illegible] June 6th. On June [illegible]

PLAINTIFF'S EXHIBIT 1528 (104)

plexing problem to the agency administering such laws, and to those industries wherein the hazards of silica exist. The intricacies of the medical aspects of silicosis are generally unknown to the average physician throughout the country. All employees engaged in dusty trades presently show some fibrosis of the lungs, not in fact disabling. Today it is exceedingly difficult to find physicians, conveniently available to industrial plants removed from our larger cities, qualified and experienced in the diagnosis of silicosis. This makes possible the improper diagnosis of silicosis which may be prejudicial to both the employee as well as the employer. If the employee is not in fact disabled and is told that he is suffering from the disease he naturally worries, desires to give up the job wherein he is exposed to the hazard, begins to brood over his condition, and soon he and his family become public charges upon their community. There have been many instances with this unfortunate result and its seriousness must be recognized. From the standpoint of the employer, improper diagnosis may result in the legal persecution, by common law suits or unjustified compensation claims, of those industries wherein the silica hazard may be present. Litigation is costly, not only in dollars and cents, but in its general effect upon the entire employer employee relationship and may tend to destroy public confidence in an industry well managed, properly equipped and operated with full consideration for the protection of the health of its employees.

Many irresponsible statements have been made in the public press by uninformed persons about the subject of disability from silicosis. Fortunately, a great deal of this information is being rapidly disproved, but the inclusion of partial disability benefits in compensation laws serves as the medium by which unjust and improper awards may be made and often tends to serve no particular benefit to the claimant.

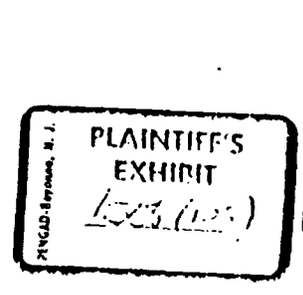
The basic theory of the Workmen's Compensation Acts of our several States is to provide compensation only for actual disability. In the absence of some competent and approved method of evaluating partial disability, recognized and accepted by the medical profession, it is submitted that legislative provisions denying compensation for partial disability are desirable. Perhaps by continued study and research, the medical profession may find some method to value partial disability and at that time the laws can be amended providing such compensation.

With respect to compensation for total disability, [illegible] is far simpler. The medical profession is agreed that when silicosis is accompanied by active pulmonary tuberculosis, the employee should be removed from the hazard of [illegible] protection and for that of his fellow workmen. [illegible] cases where the employee's disability is caused by silicosis [illegible] ied by active pulmonary tuberculosis, he should receive the full benefits of the law. Silicosis accompanied by other types of pulmonary infection may similarly render the employee [illegible] work [illegible] it is reasonable and proper that full compensation should be awarded. [illegible] this problem that [illegible] difficult from its legislative [illegible] that is to write into the law some proper definition of the [illegible] infections that will not permit other diseases [illegible] silicosis to be the basis of compensation awards. I [illegible] to you for medical consideration and believe that some proper answer to this difficulty may be found.

Before leaving the subject of disability, I desire to express the hope that [illegible] the medical profession may find the [illegible] means of effecting cures for silicosis or at least arresting [illegible] that [illegible] disabling. In the past we have accepted [illegible] the thought that the disease is incurable and leads [illegible] death. I am confident that with continued research some means [illegible] be developed to change our fatalistic thought and that we may [illegible] see the rehabilitation of those suffering from this disease.

2. *Potential Liability of an Employee for Silicosis Exposed Existing at the Effective Date of the Law.* [illegible] materials, employees have been [illegible] and their present Roentgen examination [illegible] some dust in their lungs. The medical [illegible] each of us has some present evidence of fibrosis [illegible] cumulation of injurious substances over a period of [illegible] States enacting compensation laws, the potential liability [illegible] juries sustained by employees during the previous [illegible] ployment becomes actual, even though [illegible] effective date of the act is not compensable. The [illegible] loosely called by the misnomer, "accrued liability," [illegible] been a matter of grave concern to employers and [illegible] carriers in contemplating the enactment of compensation [illegible]

PLAINTIFF'S EXHIBIT
[illegible]
PENGAD-Bayonne, N. J.



In my judgment this concern is well founded and present a most difficult problem. On their part, employers feel that it is unjust to impose upon them present liability for injuries for which they were not responsible prior to the effective date of the act. In a very real sense, their contractual relationship with their employees is changed by law. However, employees are concerned solely with the fact that they have contracted the disease and have sustained disability therefrom. They are not concerned with legal and medical distinctions that attempt to appraise the injuries sustained by them before the act becomes effective. Labor leaders are unanimous in their insistence that no part of this liability should be assumed by the employee. This statement is not made critically, because I have full sympathy with this point of view. However, in stating the problem you will immediately recognize its controversial nature, and all of the States upon enactment of legislation, have made some provision to solve this difficulty in a manner that attempts to be equitable to the interests that are affected.

There have been devised three methods relating to this subject to which I will make a brief reference, evidenced by the acts now effective in the States of New York, Illinois and Pennsylvania. By the New York method, there is imposed a limitation of liability upon the employer in the amount of $500 for disability of an employee occurring during the first month the act becomes effective, with progressive increases at the rate of $500 per month thereafter, until a maximum benefit of $3,000 is reached. This recognizes the necessity for a limitation of liability to be imposed upon the employer for injuries existing at the time the act becomes effective and adopts a plan of progressive liability so that the cost thereof may be absorbed by the employer over a period of years. It is to be noted that full liability created by the law is imposed solely upon the employer and that the particular industry wherein the silica hazard exists must discharge this liability. This method is followed in the present act now effective in the State of Ohio, and was continued in the silicosis compensation law passed by the recent session of the Rhode Island Legislature, although vetoed by the Governor of that State after passage. Similarly it was followed in the law before the 1937 session of the Maryland Legislature, which failed of enactment.

By the present law now effective in the State of Illinois, provision is made that where an employee had contracted silicosis prior

to the effective date of the act, but was not disabled thereby, he was permitted, within sixty days after such effective date, to file with the Industrial Commission a request for permission to receive full compensation benefits under the law. Thereupon with the approval of the Industrial Commission, such employees were permitted to continue in their employment, waiving full benefits, and thereafter were entitled to receive only fifty per cent of the compensation which, but for such waiver, would have been payable by the employer. Upon the enactment of this law, certain industries, wherein silica was a hazard caused their employees to be examined to determine the extent of injury that had previously occurred to their lungs. I was also advised that certain insurance companies refused to insure silicosis risks under this law, pending the completion of such examinations of employees. This resulted in the refusal of many employers to continue certain of their employees in employment because of the potential liability exposure by their continuance. I am not familiar with its practical operation. I believe that this method gives rise to invariable difficulties of administration.

At the 1937 session of the Pennsylvania Legislature, provisional [illegible] was enacted, including provision for compensation for silicosis injuries. We are aware that [illegible] Pennsylvania has one of the most severe problems [illegible] dust diseases, due to the coal industry. The Pennsylvania [illegible] method of dealing with the subject, provides that within one year the act becomes effective, the employer pays accrued and current compensation for injuries that become disabling during that year and the Commonwealth reimburses the employer. During each succeeding year the [illegible] rate of one-tenth, while the [illegible] decreased one-tenth. At the end of ten years [illegible] imposed upon the employer for [illegible] An appropriation in the amount of $[illegible] to discharge the liability of the Commonwealth. It is [illegible] appropriation is inadequate. There is no [illegible] to determine the extent of this liability, and [illegible] administration of this law the State may well [illegible] provision.

In the light of the present limited experience [illegible] tion of laws that have been heretofore enacted [illegible]

PLAINTIFF'S EXHIBIT 1045 (1-)
RYKO-Bayonne, N. J.

we now know the best method of dealing with this problem of accrued liability. If our present society deems it proper to provide full compensation for injuries sustained by employees before the effective date of the act, the burden of such compensation should not be imposed solely upon the employer. On the other hand, it seems unfortunate that the employee should be required to participate in the expense of his disability. While I recognize certain merit in the Pennsylvania plan above described, I question the wisdom of burdening the taxpayers of the State with an expense that is peculiar to certain of its industries.

3. *Pre-employment and Periodic Post-employment Physical Examinations of Employees; Autopsies in Death Cases.* The medical profession recognizes and recommends pre-employment and periodic medical examinations for the prevention and control of silicosis. As authority for this statement, I quote the following extract from Medical Series Bulletin No. 1 of the Air Hygiene Foundation of America, Inc., above referred to:

> "Medical control should include a practical program of pre-employment and periodic examinations of all workers exposed to dust containing silica."

Examinations are indispensable to eliminate tuberculars from silica hazards, and to protect the employee suffering from the disease, as well as fellow employees who may be exposed to infection. Therefore, any attempted prohibition of physical examinations, either prior to employment or periodically thereafter is a backward step in any legislative program providing for the control and prevention of silicosis. Certain of the present laws deal with this subject. Section 65 of the New York law declares the policy of the Legislature of that State to be:

> "Prevention of silicosis and other dust diseases. 1. It is hereby declared to be the policy of the legislature of this state, in enacting this article, to prohibit through every lawful means available, any requirement as a prerequisite to employment which compels an applicant for employment in any occupation coming within the purview of this article to undergo a medical examination."

It is to be noted, however, that under the New York law, there is no statutory prohibition of pre-employment or periodic physical

examinations. In the present law recently passed in the State of Pennsylvania, provision is made that when an employer fails to accept the Workmen's Compensation Act, and later subject his employees to physical examination and within the period of one year thereafter such employees are discharged, the employees may elect:

(1) To recover compensation for occupational disease injuries under the present occupational disease act or

(2) To maintain an action at common law against the employer for occupational disease injuries so sustained, in which proceeding the common law defenses of the employer are denied.

This provision is clearly designed to prohibit the discharge of employees after physical examinations, operating as a penalty upon employers who have not elected to be bound by the provisions of the Workmen's Compensation Act, including its provision for compensating occupational diseases.

In most of the silicosis acts now effective there is a provision requiring claimants for compensation to subject themselves to physical and Roentgen examinations, either by physicians selected by the employer or designated by medical boards created by the terms of these acts. Continued refusal on the part of the claimant to so subject himself to these examinations operates as a bar to compensation. I believe that such provisions are indispensable to the proper administration of silicosis laws and to enable the administrative agency to determine the actual condition of the claimant.

Similarly, provision is made [illegible] event of disputed claims compen[illegible] of the decedent who refuse to permit [illegible] made. The importance of [illegible] sion and apparently affords the most [illegible] tive diagnosis of the disease. In those States [illegible] the board is given power to order autopsies [illegible] this is necessary to accurately and scientifically determine the cause of death. The rights of the claimant are protected by [illegible] his physician to attend and observe the autopsy and to [illegible] decedent's condition. The examiner's certificate [illegible] and becomes a part of the record of the claim. In [illegible]

PLAINTIFF'S EXHIBIT 15[illegible] (177)

is afforded to the administrative agency indisputable evidence upon which to base its decision.

Any comprehensive plan for the medical control of silicosis should include pre-employment and periodic post-employment physical examinations of employees. We must recognize the justified criticism of labor agencies with respect to the use of such physical examinations that may have been heretofore made by unconscionable employers. The practice of manufacturers who would attempt to use physical examinations of employees for the avoidance of their just liability cannot be condoned, but I am firmly convinced that we will never be successful in solving this industrial problem without making these examinations an essential part of the program of manufacturers in those industries wherein silica hazards exist.

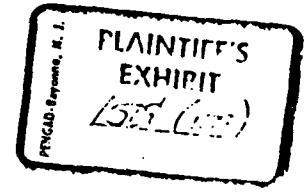
4. *Creation of a Medical Board.* This subject is similarly one of the most controversial that is presented by any proposed act for the compensation of silicosis injuries. In my opinion, the creation of medical boards is of vital necessity for the proper administration of any occupational disease law. The very fact that society recognizes the new [illegible] type of compensation legislation of itself admits the necessity of establishing an agency for the diagnosis of the disease and the appraisal of disability arising therefrom. These are the most important questions presented by the claim and a board of qualified physicians is the most competent agency for their determination. By the creation of medical boards, the controverted medical opinions would be decided by an agency, competent, impartial and best qualified to determine the actual condition of the claimant. In those state laws that provide for the appointment of medical boards or physicians, the powers granted to such boards are limited to be advisory to the Workmen's Compensation Board, Referee, or other agency administering the law. This has materially assisted in efficient administration.

Full power should be granted to the medical board for the examination and re-examination of claimants, with the right to conduct autopsies in death cases where the board determines such procedure to be essential to the determination of the cause of death. All medical questions arising in connection with claims should be referred to this board for investigation and report, and further proceedings with respect to the claim should be stayed until the Board has conducted its investigation and filed its report. The successful administration of [illegible] procedure [illegible] dependent upon this procedure and in the event the [illegible] medical board may be impeached by other agencies, the [illegible] of the parties affected thereby will be [illegible]

From the standpoint of the administrative agency, it is not unlikely that controversy will arise upon the medical questions presented by the claim. Parties appearing as [illegible] claimant or employer will present their respective views. It is assumed that these opinions will be thoroughly professional in their nature and represent the honest and mature judgement of the witnesses. Consider for a moment the dilemma of the agency charged with the determination of the merit of this claim. We return to the [illegible] existing in the old method of jury trials where such new claims require that the parties to the litigation are charged with the necessity of educating the Workmen's Compensation Board or Referee in the medicine of silicosis. The medical board, composed of competent, qualified physicians representing the State, disinterested as a party to the proceedings, is better able to determine these questions than any other agency that may be suggested.

Before leaving the subjects above discussed, I believe it would be of interest to state briefly the attitude of representatives of employers and employees with respect thereto. I do this, not in a spirit of controversy, but simply to emphasize their importance. Employers, harassed by incompetent medical diagnosis, have advocated the denial of compensation for partial disability. On their behalf, I believe it may be fairly stated that today they recognize their moral obligation to provide compensation for [illegible] the disease of silicosis is accompanied [illegible] losis. As to accrued liability, they [illegible] tion upon them of the cost of procedure [illegible] for which they were not previously legally responsible. Upon competent medical advice, they have, in many instances, encouraged the [illegible] of pre-employment and periodic post-employment physical examinations. Generally speaking, they have opposed [illegible] powers to medical boards, having been reluctant to accept the final determination of such agencies as to such claims.

On their part, employees have been uniformly [illegible] for compensation for partial disability. As previously [illegible] have opposed their assumption of any part of the cost [illegible]



tion for injuries sustained prior to the effective date of the act, contending that there should be no limitation of the liability of their employers. Believing that individual employees have been persecuted by the use of physical examinations, they have opposed pre-employment or post-employment physical examinations of employees conducted by physicians selected by their employers or by physicians selected by anyone other than their representative unions. They have consistently opposed provisions for the creation of medical boards or the granting to such boards of powers for the determination of their injuries. I believe that the position of employees is strikingly expressed by the following incident that occurred within the past month.

I was not employed in any manner in connection with the silicosis bill pending at the recent session of the Rhode Island Legislature and in fact, knew nothing of its details until it was passed by the Legislature and vetoed by the Governor. My attention was called to a newspaper article in the Providence Journal under date of May 5th relating to the reasons of the Governor for his action in vetoing the bill, the article stating that he had vetoed the bill at the request of Secretary Perkins, the State branch of the American Federation of Labor, and other labor organizations. Upon procuring a copy of the law as passed, I found that it was modeled upon the law now effective in the State of New York, adopting the same method of compensating for accrued liability, with a limitation of $3,000. It also denied compensation for partial disability and created a medical advisory board, whose determination of the medical issues in controversy was made conclusive. On May 29th, I addressed the following inquiry to Secretary Perkins:

"Miss Frances Perkins,
Secretary of Labor,
Washington, D. C.

Dear Madame Secretary,

I have been very much interested in the silicosis bill that was pending before the Rhode Island Legislature, and have been advised that after passage it was vetoed by the Governor. The enclosed clipping from the Providence Bulletin reports that the Governor vetoed the bill at your request and at the request of certain labor organizations of the State. I would appreciate your advice as to whether or

not this is correct and your reasons for advising the Governor. I have been generally interested in seeing the disease of silicosis made compensable under the compensation laws and would greatly appreciate any advice that you can give me.

Very truly yours,
[illegible]

and received the following reply:

"Mr. Theodore C. Waters, Chairman,
Maryland Commission for the Study of
Occupational Diseases,
1941 Baltimore Trust Building,
Baltimore, Maryland.

Dear Mr. Waters:

The Secretary has referred your letter of May 29th to this Division for answer.

The Department of Labor did point out to the Governor and the Labor Commission of Rhode Island the dangers inherent in the silicosis bill pending in that State. We did not feel that the bill protected adequately the interests of labor and the general public. In the first place, it placed an extremely low limit on compensation liability and on medical benefits. It eliminated any compensation benefits for partial disability despite the experience of the States that total disability or death from silicosis is almost always preceded by relatively long periods of [illegible] reduced earnings on the part of the [illegible] administration in the [illegible] autopsies compulsory, and for the [illegible] set up made it particularly difficult for the [illegible] or his dependents to secure even the low benefits provided by the act.

Should you have further questions I am sure Mr. [illegible] mer will be glad to discuss them with you when he returns from Europe the latter part of this month.

Very truly yours,
[illegible]
Acting Director"

PLAINTIFF'S EXHIBIT 6523 (129)
PENGAD-Bayonne, N. J.

5. *Prevention and Control.* The prevention and control of injuries from silicosis is and should be the goal of all parties interested in industrial hygiene. Employees are more concerned about the maintenance of their health than in compensation for injuries to that health, while to employers the cost of the prevention of silicosis injuries will prove to be but a small portion of the cost of compensation for such injuries.

I believe it is to be regretted that more emphasis has not been placed upon state legislation relating to the prevention and control of silicosis. Statutes making provision for the establishment of departments of industrial hygiene to which are assigned programs of prevention and control are subject to the criticism that the duties assigned to such departments are somewhat indefinite and incomprehensive. Many of the States have passed compensation acts but have not established the necessary agencies required to promote an effective program of prevention. The provisions of the New York law are perhaps the most comprehensive and effective in this respect. Section 65 of this law provides as follows:

> "2. The industrial commissioner and the industrial board are hereby required to add to the industrial code, as provided in sections twenty-eight and twenty-nine of the Labor Law, effective rules and regulations governing the installation, maintenance and effective operation in all industries and operations wherein silica dust or other harmful dust hazard is present, of approved devices designed to eliminate such harmful dusts and to promulgate such other regulations as will effectively control the incidence of silicosis and similar diseases."

Pursuant to this provision, the Industrial Board of New York has undertaken the promulgation of rules and regulations designed to accomplish the intended purposes of this act.

Rules and regulations promulgated by departments of industrial hygiene should take into consideration the particular needs of the industries of the State, but once adopted, their enforcement may effectively assist in the control of the hazard existing in the State's industries. It must be borne in mind that regardless of the particular statutory provisions that may be adopted, the success of the program for prevention must rest upon the administration of the law.

Adequate personnel must be developed to provide [illegible] other officers trained in industrial hygiene for the purpose of [illegible] ing working areas and assisting manufacturers in the development [illegible] of machinery and equipment [illegible] of employees. This could be accomplished within the existing State Departments of Health and Labor, and with the cooperation and advice of such State agencies, many of the hazards now existing in our industrial processes could be controlled or eliminated.

Summary: We must bear in mind that most of the [illegible] laws now effective are of relatively recent enactment. [illegible] are perfect and in their administration, giving effect to the [illegible] element, certain inequities will inevitably arise. Defects [illegible] administration will be corrected by further amendments and [illegible] mately they will be fitted to the needs existing in the particular [illegible] where they are effective.

One of the principal difficulties presented is the lack of authoritative information by those charged with their administration. During the past several years I have met with and consulted compensation officials and have found that they are intensely interested in the proper, just and efficient discharge of their duties. [illegible] or investigation presents to them a new appreciation of the importance of their function and with continued experience they will be better enabled to perform the duties with which they are charged.

The medical, engineering and legislative aspects of the problem still present many questions, the answers to which are not known by even the best informed. To answer these questions further research is necessary for the development of [illegible] control. The United States Public Health [illegible] state departments and other public and [illegible] voting their energies to the development of [illegible] sorely needed to eradicate this disease, and I believe that we may now look forward with confidence to a time in the near future [illegible] full medical and engineering control will have reduced to a [illegible] the hazards of silicosis in industry.

PLAINTIFF'S EXHIBIT
1537 (130)
PENGAD-Pennsa, N. J.

Discussion of Mr. Waters' Paper

Mr. Waters was asked by Dr. Kammer if Labor might be expected to oppose preemployment and periodic examinations. He replied that to his knowledge no such program was contemplated. He said he had discussed the subject with an official of the A. F. of L. who inferred that Labor seemed reconciled to the general policy of physical examinations. The particular official said that he recognized that such examinations were indispensable but that Labor wanted them made by physicians designated by their local unions. The men, he said, had no confidence in the Medical Boards that have been created, and felt that employers and insurance carriers would receive greater consideration than they. To meet this objection Mr. Waters thought that future laws might make some provision for nomination of one member of the Medical Board by the labor group of the state concerned. The two other members could be selected by the State and by the employers respectively. It was his belief that the informed leaders of the labor movement generally recognized the need of physical examination in industries with a silica hazard but that they did not have much confidence in the Medical Boards as now constituted.

Dr. Kammer said that his question referred more particularly to preemployment examinations made by physicians in the community or in the employ of the company. Mr. Waters said that perhaps someone else could answer this question better than he. Mr. Cummings recalled that the A. F. of L. in Massachusetts had ordered an assessment of $50.00 on any member who submitted to physical examination. The C. I. O. had not yet had opportunity to express its attitude. He reported that Michigan had now an Occupational Disease Law which did not provide for a Medical Board. Before the present law was passed an agreed bill was under consideration which did provide for both preemployment examinations in hazardous industries and for a Medical Board. This bill failed to pass but in anticipation that it might, the C. I. O. had set up a Medical Board of its own to examine all of its members. It was inferred that if the findings of its Board had been at variance with those of the State Board there would have been a legal contest on the evidence. It was his opinion that too many of the pending acts were hastily devised and often contained unreasonable provisions. If in-

formed leaders of both labor and industry could agree on proper bills he felt that the administration of such acts would be reasonable.

He believed that the City of Milwaukee had handled the situation admirably. A private physician who was thoroughly trained and had adequate facilities managed a clinic where preemployment examinations were made for various local industries. At a nominal charge he issued certificates of physical fitness for employment in trades of all kinds. The Wisconsin State Industrial Commission, under the able direction of Mr. Voyta Wrabetz, had assumed a very fair attitude; it had held that uncomplicated silicosis was not disabling in a majority of instances. Men with silicosis plus [illegible] were considered totally disabled and given compensation [illegible] treatment had inspired confidence in the labor group and they had responded in a sane and reasonable manner.

Mr. Waters was asked whether he had given thought to the question of who should constitute the proper authority to make preemployment examinations in case Labor objected to having [illegible] by physicians in the employ of industry. He replied that it would be more appropriate for the chairman to answer this question. Dr. Gardner said that he had thought much about the question. It was obviously the duty of some group of medical men to make such examinations and certify as to the fitness for employment in various industrial environments. Most private practitioners [illegible] so little about industrial conditions that they were not equipped to decide whether an applicant's physical condition would be [illegible] ably influenced by working at a particular job. [illegible] employ of the state might have to a [illegible] done in South Africa if [illegible] isfactory manner. For the state [illegible] be a great misfortune and would most [illegible] lems than now existed. It was his hope that industry in general would follow the lead of many concerns now engaged in the practice and employ well trained conscientious physicians who [illegible] deal fairly with both labor and employers. In the [illegible] industries which could not afford to employ their own physicians he felt that the Milwaukee plan would be the ideal solution.

Mr. Cummings said that he hoped the day would [illegible] when industry would have to forsake its right to make [illegible] employment examinations. He felt that the operating [illegible]

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and the hazards of each industry were so specialized that no board or group of state boards could know enough of these individual hazards to certify intelligently as to the fitness of all individuals for various employment. He believed that industry might have to modify some of its present procedures after consultation with representatives of labor. But he believed that this was the only proper means for conducting preemployment examinations and earnestly hoped that the system might evolve along these lines.

Mr. Waters then recited examples of the unfortunate experience of a certain industry, for which he was counsel, in attempting to employ the services of local general practitioners. These men had not had sufficient experience to diagnose silicosis that might have developed nor to make proper preemployment examinations, particularly when the interpretation of roentgenograms was concerned. In one state such a consultant had advised rejection of 75% of the applicants he had examined. Review of their examination records and films by Dr. Pendergrass resulted in rejection of only one man in 100.

Mr. Cummings called attention to the fact that the Saranac Laboratory had inaugurated a program of medical and engineering control in a steel plant. The problem here was not one of silicosis but of promotion of general health. This plant had been closed by a strike but among their other grievances, the organizers had never mentioned the preemployment or periodic examinations that had been made. He intimated that had these examinations been objectionable to Labor, they would undoubtedly have been included in the bill of complaints.

Dr. Kammer, the physician in charge of the clinic at this plant, was asked if he would express his opinion of the attitude of the employees. He replied that he believed that any dissatisfaction on the part of Labor had arisen because of the unfair treatment that they had received from many employers in the past. He believed that his own employers had the confidence of their men and that the latter were pleased with the medical services that they received; that, given fair treatment, the great majority of laboring men were easy to deal with and that their reaction to such health measures need not be feared. As an example of the manner in which his clinic operated, he said that the employee got a much more detailed report of his condition than the management; that he was permitted to discuss his case with the clinic physicians and was then referred to [illegible] local physician for treatment if such was indicated. The [illegible] ment was not interested in these details; it wished to know [illegible] whether the man was fit for his [illegible] if not whether he [illegible] made fit or whether he should [illegible] to some other work commensurate with his strength. It had been gratifying [illegible] that many employees had voluntarily come to the clinic [illegible] examinations before they had been called.

Mr. Waters was asked for his opinion on the operation [illegible] Occupational Disease Law in North Carolina. He replied that he had little personal knowledge but from conversation with Mr. [illegible] set he had the impression that there had been little complaint [illegible] to the administration of this law. In December there had been only 4 or 5 silicosis claims, a smaller number than had been filed [illegible] the passage of the Act. The same seemed to be true in other states; in West Virginia the law had provided compensation for [illegible] since 1934 and yet up to 6 months ago only 5 claims had been brought and no awards had been granted. This seemed [illegible] prising in view of the notorious situation that had existed in Gauley Bridge previous to the passage of the law. Kentucky had [illegible] one silicosis claim.

Dr. Pierson, at the request of Mr. Waters, stated that California had had a general coverage occupational disease [illegible] time (1918). He could not recall when the first award [illegible] was made but since the Gauley Bridge affair there had been a very large number of claims, many of them unjustified. California [illegible] provided only for compensation for [illegible] that it had been administered fairly [illegible] been just in its adjudication of [illegible]

He felt that one source of difficulty [illegible] differentiate the cause of disability in [illegible] might have had silicosis but was disabled from some other [illegible] not caused by or related to the silicosis. He had [illegible] ence to cardiac conditions which physicians had been [illegible] tribute to the disease in the lung.

Mr. Waters said that under the Pennsylvania Act, [illegible] not yet been signed by the Governor, compensation [illegible] ability was denied. Total disability for silicosis was [illegible] proof that the claimant had silicosis accompanied by either [illegible]

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loss or streptococcic infection. He said that he had never known of the inclusion of the latter type of complication and asked if there were any medical authority for it.

Dr. Gardner replied that he knew of none and could only imagine the inclusion of this infection had been prompted by some individual case that had happened to impress the committee at the moment.

Mr. Waters then asked whether other types of infection not ordinarily related to silicosis would be held to consideration under this specific provision.

Dr. Gardner replied that he would not think so. The only condition that might cause confusion would be pneumonia. He recalled that in one of the previous sessions this subject had been discussed with the conclusion that lobar pneumonia was probably not caused by silicosis although its course might be modified by it.

Dr. Sappington commented upon the Illinois Act, passed in October 1936, which provided that employees with some evidence of pneumoconiosis might continue employment if they waived their rights to future benefits under the Law. Since its passage, application for about 800 waivers had been filed. He pointed out that compensation was not the proper solution to the silicosis problem. It was merely a palliative and prevention of future disability was the proper objective.

* * *

In closing the Symposium Dr. Gardner commented that silicosis had been discussed from many angles. An attempt had been made to present not only what was definitely known about the condition and its causation but many of the hypotheses that were being entertained by different investigators had been presented and evaluated in the light of clinical and experimental observation. He trusted that the wealth of detail had not been confusing and that the members of the group had been able to differentiate between fact and supposition. He then thanked those who had taken part in the discussions and were responsible for the success of the meetings.

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*Prepared by Miriam Thurber, Chief Librarian of the

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pass on through it and are deposited in the more normal lung beyond. When the intrapulmonary reaction extends to the surface of the lung, the pleura becomes inflammed and a local fibrosis ensues. The mechanism of its involvement is not altogether clear as asbestosis bodies are generally scarce in the pleura itself.

Animal experiments have thus far failed to reproduce the widespread fibrosis seen in the human cases that have come to autopsy. Whether this is due to insufficient exposures or whether it is because the fatal human cases have been complicated by an element of healed infection still awaits demonstration. The speaker suspects that the latter may be the case for many infections, including tuberculosis, show a decided tendency to heal with the production of extensive scar formation in animals with asbestosis.

The method by which asbestos exerts its effect has not been clearly defined. There are some who believe that asbestosis is merely a silicosis which manifests itself in a different anatomic form. They hypothecate that the magnesium is leached out of the silicate molecule leaving silica atoms to act upon the tissues. If this were true one would expect asbestos to exert its effect upon connective tissue in any part of the body as happens when free silica is injected, but such is not the case. Sayers and Miller failed to produce fibrosis by intraperitoneal injection of chrysotile asbestos and have classified this substance as "inert". Similar results have been obtained in the Saranac laboratory both in the peritoneal cavity and other tissues of the guinea pig and in the extra pulmonary organs of other species. In the light of present experience asbestos seems to be a dust capable of producing reaction only in the lungs. Possibly the irritation in this organ is largely mechanical although this remains to be demonstrated.

An unusual feature of this disease is the formation of the asbestosis body. This results from the development of an iron containing coating upon the surface of inhaled asbestos fibres. It appears after about 2 months contact with the pulmonary tissues. Injection of asbestos into other tissues usually fails to produce such bodies although a very occasional one has been discovered in the peritoneal and subcutaneous tissues. As the coating thickens it fractures into segments whose edges become smooth and rounded. The structures are bright chrome yellow and may be 20, 70 and even 100 or more microns in length, and 2 to 4 microns in width. They resist digestion with trypsin and strong alkalies but not strong sulphuric acid, they apparently persist in the tissues for prolonged periods. Dr. R. G. Mills reports a case in which death occurred 32 years after a short exposure to asbestos dust. Sections of the lungs, which the speaker has studied, still show what appear to be the remnants of largely digested asbestosis bodies in addition to large numbers of naked fibres. This finding in an isolated case suggests that in time the coating on the fibres may be redissolved. Many observers have reached the conclusion that the coating substance is laid down by the body to protect itself against irritation. In support of their view is the fact that numbers of bodies digested out of the tissues with antiformin and injected into animals failed to cause fibrosis or other reaction. But if this explanation is correct one would expect similar deposits on foreign bodies of other types, but such is not the case. It seems much more probable that as Cummings has suggested, the coating material is dissolved out of the fibre and redeposited on its surface. The iron may be derived from the fibre or from the body fluids as proved by injecting iron free asbestos.

The significance of asbestosis bodies in the sputum or in pulmonary tissues depends upon the occurrence of associated fibrosis. If only a very few are present they may result from accidental inhalation and hence do not mean disease. The speaker has found occasional ones in the lungs of at least a half dozen cases where there was no industrial exposure to asbestos dust.

RELATIONSHIP BETWEEN PNEUMOCONIOSIS AND TUBERCULOSIS

This section on infection relates particularly to tuberculosis for while it is generally granted that all types of pulmonary infection are more common in the silicotic than the normal lung, the evidence in support of this view is not too clearly defined. It is coming to be recognized, for example, that lobar pneumonia is not unusually common or unusually fatal in persons with silicosis. The same may not be true of the bronchopneumonias, which need much more investigation. But there is plenty of evidence to prove a specific etiologic relationship between silicosis and tuberculous infection.

Experimental Methods—have substantiated a host of clinical and statistical investigations which prove that two conditions are interrelated. Price of Toronto demonstrated that the addition of

[illegible]
controlled and con[illegible]

The subject that has been a[illegible]
"Legislative Control and Compensation for Sili[illegible]
order that I may not be misunderstood in the remarks that I
make, I desire to identify myself and state the interest with which
I have been heretofore affiliated. My original interest in silicosis
began as general counsel for a corporation engaged in the mining
and manufacture of silica for [illegible] purposes. That company
had been subjected to a number of common law suits, all of which
were based upon alleged disabilities from silicosis on the part of
their employees. From that experience, I came to the conclusion that
common law procedure with jury trials was not a satisfactory method
to adjust this type of industrial dispute. Since then I have been em-
ployed by various other corporations producing or using silica, in
the defense of common law suits and compensation claims, or en-
gaged in their behalf in the drafting of legislation providing com-
pensation for these injuries. In a non-professional way I served,
during the past year, upon the Maryland Commission for the Study
of Occupational Diseases, which prepared and submitted to the
Governor a proposed law, designed to control and compensate occu-
pational disease injuries. I was also a member of the Legal Com-
mittee of the National Silicosis Conference appointed by the Secre-
tary of Labor. From these experiences I have reached certain con-
clusions with respect to compensation legislation and, in the present
instance, am at liberty to express my views totally independent of
any connection which I may have had heretofore. Several of the
subjects concerned in that legislation are controversial, and I have
no doubt that many of you may disagree with some of the opinions
or conclusions that I may express during the course of my remarks.

[illegible]
[illegible]*ferent* from previ[illegible]
Acts, and there have been many [illegible]
administration develops defects. Generally [illegible]
this method of providing compensation has elevated [illegible]
humanity over common law procedure and modern industry
copting it as an essential part of our compensation procedure.

I am unable to speak to you as one trained in [illegible]
science of legislation. My experience in such matters has been
ed although for the past five years I have been interested [illegible]
ally in legislation having as its purpose the compensation [illegible]
prevention of occupational diseases. The most perplexing pr
e that presented by the disease of silicosis, which requires
provisions separate and distinct from other types of occup
disease injuries that employees may suffer during the course
employment. This has created controversies among the vari
terests that may be affected. Of late all employers and em
alike there have been presented radical, reasonable and conse
views and intelligent effort has been directed to find some w
solution to the intricate questions that the problem presen
recent years considerable publicity has been given to silicosis
alleged effects upon employees suffering therefrom. Even
we have come to know more about the disease and how [illegible]

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