FILE NAME: Kohler (KOH)

DATE: 1933 Feb

DOC#: KOH115

DOCUMENT DESCRIPTION: Conference Proceedings - Effects of Dusts Upon the Respiratory System

INDUSTRIAL COMMISSION
OF
WISCONSIN

Proceedings of Conference Concer

EFFECTS OF DUSTS U
THE RESPIRATOR
SYSTEM

Held at Medinah Athletic Club, Chicago
November 16–17, 1932

FEBRUARY, 1933

INDUSTRIAL COMMISSION
OF
WISCONSIN

Proceedings of Conference Concerning

EFFECTS OF DUSTS UPON THE RESPIRATORY SYSTEM

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PROCEEDINGS OF CONFERENCE CONCERNING EFFECTS OF DUSTS UPON THE RESPIRATORY SYSTEM HELD BEFORE THE INDUSTRIAL COMMISSION OF WISCONSIN AT CHICAGO, ILLINOIS, NOVEMBER 16-17, 1932

Wednesday, November 16, 1932. Morning Session

By MR. FRED M. WILCOX, *Chairman*, Industrial Commission of Wisconsin

Let the record show that there are present with me here at this conference the other members of the commission, Mr. R. G. Knutson and Mr. Voyta Wrabetz; Mr. Harry A. Nelson, Director of Workmen's Compensation for our commission; Mr. Arthur B. Doe, Attorney of Milwaukee, representing management; and Mr. L. A. Tarrell and Mr. W. J. Goldschmidt, Attorneys of Milwaukee, representing the employe group. Present also are Dr. A. W. Gray of Milwaukee, and Dr. J. B. MacLaren of Appleton, representing our committee for the development of a dust and fumes code; also our Mr. W. C. Muehlstein, Director of the Sanitation Department. I think we will register the rest of you by aid of registry slips, so that the attendance record may be both accurate and complete.

First of all I want to express for the commission its appreciation of your readiness to come here for this conference and doubly so for the readiness of insurance carriers and self-insurers in Wisconsin to make it possible for us to get together for consideration of this important subject. I think at the outset I can perhaps clear a bit of the air if I would remind you that there are only a few states that undertake to cover diseases of occupation under compensation, outstandingly Wisconsin. While California, North Dakota,

Connecticut and Massachusetts have laws under which it appears they may take care of the whole field, I am not so sure that they are doing it. There are certain other states that have laws which under a schedule enables sufferers from occupational diseases specifically listed in the law to have the benefits of compensation. That, of course, eliminates from protection any person suffering from a disease that is not listed in the law. Some states are doing nothing in this field. If they compensate any particular type of disease which they may call occupational, they do it under the accidental injury provision of their law under the theory that where there is a single contact and injury results, that the situation squares with the idea of accident and they are compensating for such disease under the theory that they are accidental injuries. We did the same thing in Wisconsin and we do it now with many types of diseases, such as typhoid fever, anthrax, ivy poisoning and the like. These are rated as diseases of particular industries when they arise out of the employment, but under the law are compensable as accidents rather than otherwise.

It is important to bear in mind that under the Wisconsin Act disease coverage is not limited to the typical occupational diseases of medical literature, but the provisions of the law are broad enough to extend to any type of disease which an industry produces or to which a particular plant operation exposes its employes. Typhoid fever is a good illustration. We can think of others. Ivy poisoning is not recognized as an occupational disease. It is a disease that you acquire because you come in contact with poison ivy. It is not present on all farms. On many farms there is none of it. But it may be on a particular farm, so it becomes a hazard to employes on that employer's premises. Just so chicken pox, scarlet fever, whooping cough and other children's diseases are diseases to which teachers are peculiarly exposed, but they are not diseases which we commonly think of as occupational in character.

It is a difficult matter to determine the extent of disability in many types of injury. Wisconsin found great advantage in an intensive study of various phases of typical injuries other than dismemberments. We went out, much as we are going today, and got information as to the ex-

tent of disability normally attaching in these specific types of injuries. Then we set up rules and figures by which we undertake to estimate disability resulting from such injuries, not dismemberments,—such as an ankylosed knee or elbow, loss of rotation, shortening of leg and that sort of injury. From those tables the commission and our industries, safety men, plant managers, personnel directors, doctors, lawyers, insurers, and the injured men themselves may get their bearings and determine what is the approximate extent of disability. This procedure is very conducive to uniformity of administration. We had the orderly help of the American Medical Association in the development of the rules by which Wisconsin measures the extent of disability of an eye and those rules have been formally adopted for guidance. Our experience in that field has been so satisfactory and has given us such assurance of better type of administration that we are ready and glad to turn to this plan of developing something in the field of pneumoconiosis, silicosis, and tuberculosis of the lungs and the respiratory tract,—diseases which are, if they are produced by an industry, compensable under the laws of Wisconsin.

Silicosis is probably recognized as a typical occupational disease. It should be included in the schedules of those states which presume to compensate for occupational diseases if they are in fact concerned with the protection of employes suffering disability from occupational hazards. Failure to do so leads unmistakably to the conclusion that schedules have for their purpose the eliminating from coverage of injuries to the lungs and to the respiratory tract. The difficulty is not in determining the extent of disability in the case of silicosis and tuberculosis. The difficult task is to determine whether or not the plant operation had anything to do with it,—whether the exposure had anything to do with the condition that the employe now presents. And if so, is it solely responsible for it, is it partially responsible for it, is it just an aggravating factor, or was this industry not responsible at all. These are serious questions and that is the field that gives the difficulty. To gain a better understanding of this whole subject we have arranged this conference.

We have some cases pending before us that form the

subject matter,—the basis for our holding of this hearing. There are men appearing here today who have no concern with incidental cases. Their investigation and their consideration of the subject is beyond an incidental case. Sometimes it is embarrassing for them to appear in behalf of litigants and we have thought to avoid any such embarrassment and to proceed on the plan of a conference for free discussion on the part of those who have come to be recognized as authority in this field. It is our desire that those who appear proceed with their discussion, without questions until they have finished, unless they may wish for the purpose of demonstrating a particular point, to call upon us to interpose a question or a query at that point. After they have finished it is our wish that the commissioners and the attorneys who are here have the privilege of asking any questions that they may see fit. We are anxious to complete the conference by tomorrow evening. The doctors and the engineers who are here will be able to stay through tomorrow. I am glad this is so, because of the importance of the subject. Unless there is some other question or some other statement that I should make at this time, I think we are ready to proceed as outlined.

First we will call upon Dr. Leroy U. Gardner, Saranac Lake, New York.

By DR. LEROY U. GARDNER, Saranac Lake, N. Y.

DR. GARDNER: Members of the Wisconsin Commission and others interested in this subject: I am sure I can speak for all the experts and particularly for Mr. Cummings, my associate, and myself, in thanking you for your confidence in us and asking us to come before you. The Chairman has suggested that possibly you may want to interrupt me from time to time if I do not make myself clear and I wish you would take the opportunity to do so. Two protocols have been submitted as a basis for this discussion; one of them is developed along more or less anatomical lines leading up to the discussion of the pathology of the disease due to the inhalation of dust and the other, apparently framed by a lawyer, I would hesitate to

use as a basis for my discussion. That could be used later in the discussion, but if you will permit me I will follow the anatomical outline to develop the subject.

ANATOMY

The first subject on this protocol is the discussion of the anatomy of the respiratory tract which must be considered under its several parts. Perhaps I can best express what I have to say on this subject with diagrams. My ability as an artist is not great, but I will attempt to illustrate the main parts of the tract.

Nasal and Mouth Cavities. Here we see the nose and this is the mouth cavity. The nasal cavity is entered through the nostrils which are guarded by a series of hairs. They are coarse and their function is to filter out large particles of dust and foreign bodies of any kind which might be drawn into the respiratory tract. Inside of the nose, if we look at it in cross section, we find that there are a series of very fine bones, like this, located on either side of a dividing partition which separates the two nasal cavities. Their surfaces are covered by a sticky mucous membrane reflected over the bones in this manner. It also covers the lateral wall of the nasal cavity. This sticky mucous membrane tends to catch and retain foreign particles that enter the nasal cavity. Mucus, secreted by the cells which cover this membrane, may be expelled later carrying away the foreign particles caught there. In addition we find that the cells covering this mucous membrane are provided with a series of microscopic hairs projecting from their border. These small hairs are called cilia. They are in active motion and the hairs on the different cells all move in unison creating a current; when you see the living cells under the microscope, the ciliary action gives the effect of wind blowing over a field of wheat, in a progressive sweeping motion. The direction of this motion in the greater part of the nasal cavity is backward so that foreign particles tend to be carried toward the throat. The nasal cavity communicates at the back of the nose with the so-called naso-pharynx which communicates with the larger opening at the back of the mouth.

The *mouth* is not provided with any special protective mechanism comparable to cilia. Here are the tonsils, which are located back in the throat, which do gather up a certain amount of foreign material, but not very much dust. They tend to take up bacteria rather than dust. Thus air may be taken into the mouth at this point through the lips and so into the pharynx, or it can be taken into the nose. The two currents of air meet in the back of the throat. We inhale foreign bodies through the mouth or through the nose and in both instances they reach the same place. The air then passes down into the windpipe or *trachea*, the tube leading from the pharynx to the bronchi. The trachea is also lined by ciliated cells, with hair-like processes. The direction of the current of the ciliary action here is upward toward the pharynx, tending to carry any foreign material which gets in the trachea upward to a point from which it can be expectorated. Large numbers of foreign bodies are removed in this manner. We all know that if we go down to the cellar to clean the coal bin, we very quickly begin to expectorate large amounts of mucus which are deeply pigmented with black dust. By this process one eliminates a tremendous amount of dust. To summarize,—the upper respiratory tract consists of a filter in the nose with its tortuous passages lined by a sticky mucous membrane provided with cilia. The cilia beat backward toward the pharynx and carry the dust to a point from which it can be expectorated. If the dust penetrates beyond this point it enters the pharynx and trachea where ciliary action carries it upward toward the mouth. This constitutes the first line of defense.

It has been argued that people who breathe through their mouths tend to get a good deal more dust into their lungs than those who breathe through their noses which is obviously true for the reasons already described. As long as the mucous membrane is normal and is not influenced by chronic infectious conditions and chronic disease due to repeated injury by dust a great deal of material will be eliminated by the normal mechanism.

The Bronchi. Let us now consider the bronchi which are simply tubes, similar in structure to the trachea. They carry the air onward into the lung. The trachea divides in

the root of the lung into two tubes one of which enters either lung and then subdivides into branches. These tubes become progressively smaller as they approach the periphery. Their inner surface is also lined by ciliated cells throughout their course until the periphery of the lung is reached. Their function is the same as that of the trachea.

Pleura. The surface of the lung is covered by a membrane which is known as the pleura. It extends over the entire outer surface of this organ with reflections inward over the fissures which separate the different lobes. In my drawing I am representing it as much thicker than it would actually be for the purpose of describing some of its details. The lung lies in a bony cavity, the thorax, which is also lined by pleura directly continuous with that over the surface of the lung. These two portions of this membrane are known respectively as the parietal or wall pleura and the visceral or lung pleura. Between them there is a potential space. Their inner surfaces are smooth and they are separated by an extremely thin layer of fluid which serves to lubricate them so that there will be no friction with the movements of respiration.

Blood Vessels. Radiating out from the root of the lung to its extreme periphery are two systems of blood vessels which I shall not illustrate in detail because of the complexity of the resulting diagram. One system is composed of the pulmonary artery and its branches. This vessel carries blood from the right side of the heart through the lung. Its various ramifications follow as closely as possible the branching of the bronchial tree. In addition to this there are branches of the pulmonary vein which start in the periphery of the lung, in the capillary blood vessels, and carry aerated blood back to the left side of the heart. These two systems of vessels communicate at the periphery through a system of capillaries. In these exceedingly small vessels the function of respiration takes place.

In order to understand the process of respiration we must consider in some detail the structure of the terminal *air spaces.* If one examines a cross section of a lung he will find that its consistence is like that of a sponge. There seems to be no particular arrangement of the spaces which make up this structure and it is only by a process of recon-

and adsorbing agents known. This phenomena has been conceived as responsible for the harmfulness in the lung since it was thought that it might absorb either body poisons or the normal protective substances present in the body fluids. More likely, however, is the possibility that whatever is responsible for the ability of silica to form active adsorbent agents is also responsible for the toxicity of the silica itself. In other words, the peculiar structure associated with many of the modifications of silica undoubtedly presents certain highly active surface groups capable of upsetting the delicate mechanism governing the normal life of cells. We hope to correlate the degree of harmfulness of any form of silica dust with the surface structure associated with that variety by using the knowledge of these structures recently obtained by physicists in certain X-ray studies.

To recapitulate, a dust is not to be considered dangerous either because it is or is not soluble in the body fluids. If it is soluble the toxicity depends upon the character of the soluble product formed. There are many known inorganic dusts both soluble and insoluble which are apparently harmless when inhaled over long periods and at high concentration. Silica is the most harmful of all industrial dusts. It is not yet known whether the dangerousness of silica is due to the formation of a soluble and toxic product or to some highly active surface phenomena associated with certain physical modifications. In general the degree of harmfulness can be correlated with the amount of free or uncombined silica present. In addition to knowing the amount of free silica present in any dust, in order to estimate its silicosis producing power, it is equally essential to know the physical state of the free silica. Silica in combination with other elements is also capable of producing a pulmonary fibrosis. It is therefore necessary to know the amount of combined silica and the material with which it is combined. It is highly probable that certain substances, when combined with silica, tend to overcome or partially neutralize the dangerousness of the silica itself. Any individual case of silicosis, or the whole problem itself, can be properly analysed only after obtaining a thorough knowledge of the chemical and physical nature of the causative dust.

There is another and equally important consideration with

regard to measuring the dangerousness of a dust which contains silica. I refer to the number of dust particles present in a given volume of air and especially to the size of these particles thus suspended. It is obvious that a high concentration of dust particles in an industrial atmosphere will be more dangerous than a low concentration. It is therefore a routine procedure to measure the concentration of dust present in dangerous industries. This is accomplished at present with a device perfected by many years of experimental work and approved by the U. S. Public Health Service. In brief, a measured volume of air is passed through an orifice and impinged upon a wet surface under a certain level of wetting fluid. The air is broken up into small bubbles which exposes a large amount of surface to the wetting medium and these bubbles then rise through the fluid and are eventually discharged again into the air. Dust particles suspended in the air are drawn into the impinger, wet by the fluid and remain suspended in it. In other words, the air is washed clean of its suspended dust. The suspending fluid is removed, measured, and diluted with clean distilled water. A measured portion of this diluted fluid is placed in a counting cell and the number of particles present in a given volume determined. By calculation the number of particles present in a cubic foot of air entering the impinger is then determined.

This device is efficient and satisfactory as far as the collection of particulate matter is concerned. I question the standard procedure adopted for counting the particles collected in the suspending fluid, however. The counting is done at a low magnification with ordinary bright field illumination and it can be shown both mathematically and experimentally that particles smaller than 1.5 to 2 microns are not visible. This limits the observation to those particles greater than this size. We must now ask ourselves what particle sizes are significant in producing silicosis.

First it has been observed from a study of silicotic lungs that dust particles larger than 10 microns in diameter seldom penetrate into the air spaces. It has therefore been universally agreed that we need not concern ouselves with particles larger than 10 microns in measuring the concentration of a dangerous dust. Moreover, it has been found

that the number of particles larger than 5 microns is relatively small in the silicotic lung and the present tendency is to consider only those particles below this figure. It can also be stated quite definitely that the really hazardous particles are probably much smaller than 5 microns.

I should like to describe an experiment which I have performed to prove this contention. Two series of rabbits were inoculated intravenously with very carefully prepared dust suspensions. In the first series of rabbits each animal received 1.3 grams of quartz dust particles whose average diameter was about 9 to 10 microns. The second group of rabbits each received 1.3 grams of quartz dust particles whose average diameter was 1 to 2 microns. At serial intervals animals in each series were killed and compared. In brief, it can be said that the larger particles provoked almost no fibrous reaction while the smaller particles produced a fibrosis so extensive as to result in the death of nearly all the inoculated rabbits after eighteen months. Dr. Gardner has described the details of the lesions produced in this experiment, but I am interested in pointing out the fact that the reaction was dependent upon particle size since both series of animals received an equal quantity of the same dust.

In order to demonstrate that the reaction was specific for silica, a third series of rabbits was inoculated intravenously with 1.3 grams of aloxite crystals whose average diameter was 1 to 2 microns. Aloxite is an electric furnace product composed of aluminum oxide and it is harder and sharper than quartz. Animals in this series developed no reaction about the dust which collected in the various tissues and even after many months the animals were found to be essentially normal. Therefore we can say definitely that silicosis is apparently highly specific, depending upon the presence of silica and not any hard, sharp or insoluble dust in fine subdivision. But even more significant is the fact that the silica dust must be extremely fine in order to produce silicosis.

This experiment is being carried much further in order to prove the contention that even finer particles are still more dangerous. It is obvious that if the smaller particle sizes are more dangerous, those are the ones with which

we should be most concerned in determining the dust concentration in industrial air. Since it appears that the finer the particle size the more potent is a silica dust, it should be our endeavor to obtain a measure of this fraction. For numerous reasons the practical limit of measurement or separation of dust particles is about ½ micron. It is therefore my belief that the methods used to measure the concentration of dust in the air should be modified to include this fraction.

At a recent meeting of those interested in this problem, Mr. Fehnel of the Metropolitan Life Insurance Company, and I demonstrated a technique by which this could be accomplished. This technique involves the use of dark field illumination and a different counting cell than that now being used. It is quite practical, and gives results which are representative of the finer particle sizes. I should like to urge its tentative adoption for several reasons, but particularly because I believe that many of the hazardous occupations are being overlooked. It is very possible that certain dusts are so distributed according to size that they contain relatively few particles over 2 microns and tremendous numbers of particles below this size. Such a distribution would constitute a great hazard to any workmen engaged in it and yet would be considered safe when measured by the present approved method. Industries in which such a dust is likely to be encountered, are the sandblasting industries, the grinding industries and those industries involving the use of micro crystalline silica such as that found in Illinois or Missouri and known as "Tripoli".

This discussion makes it apparent that it is essential to know the size distribution of dust particles as well as the total number of particles present in an industrial air. For this purpose the Owens apparatus is frequently employed. This device impinges a small volume of air on a glass slide to which the dust particles adhere. By examining this slide under the microscope and measuring large numbers of particles with a special micrometer attached to the microscope, the size distribution is determined.

This method also presents many technical difficulties, and tends to produce large errors when extremely fine dusts are encountered. The efficiency of any impinger decreases with

the size of the particles encountered and as a result very fine dusts may not all adhere to the slide. The chief difficulty encountered, however, is the problem of measuring dust below 1 micron. It is certain that the method is not even reasonably accurate in this range and even the most careful and painstaking observer will always be more impressed with the few large and easily visible particles than the hundreds of small, and almost invisible motes. For this reason, it is my feeling that though the Owens apparatus is a definite help in evaluating the size distribution, it does not provide a complete analysis of this factor. It is for this reason that I have suggested a method for separating any given silica dust into fractions of different size. This method, which has been described in the Journal of Industrial Hygiene, separates the dust into clean cut fractions of definite sizes from the largest particles encountered in the air down to ½ micron. By using this technique, the amount of each size fraction can be determined and by the use of tables which we have constructed the number of particles of each size can be obtained. From such data may be plotted a size frequency distribution curve on Hazens' log probability paper as Drinker has suggested. This curve once constructed can form the basis for any subsequent determination of the number of particles of any size present in the air of that particular hazardous occupation. It might be of interest to state that there are approximately 20 billion particles in a gram of quartz dust which is all 4 microns in diameter; approximately 250 billion particles in a gram of quartz dust 2 microns in diameter, over a trillion particles in a gram of quartz dust 1 micron in diameter and over 5 trillion particles in a gram of quartz dust ½ micron in diameter. These figures indicate that there are at least twenty times as many particles in a dust which is all ½ microns in diameter as there would be in a dust which was all 2 microns in diameter. These figures are based on actual counts, not on theoretical considerations which give essentially the same relationship.

A thorough and complete study of a commercial quartz dust used for producing silicosis in experimental animals in the Saranac Laboratory has shown that 90% of all the particles present (½ micron and greater) were below 2

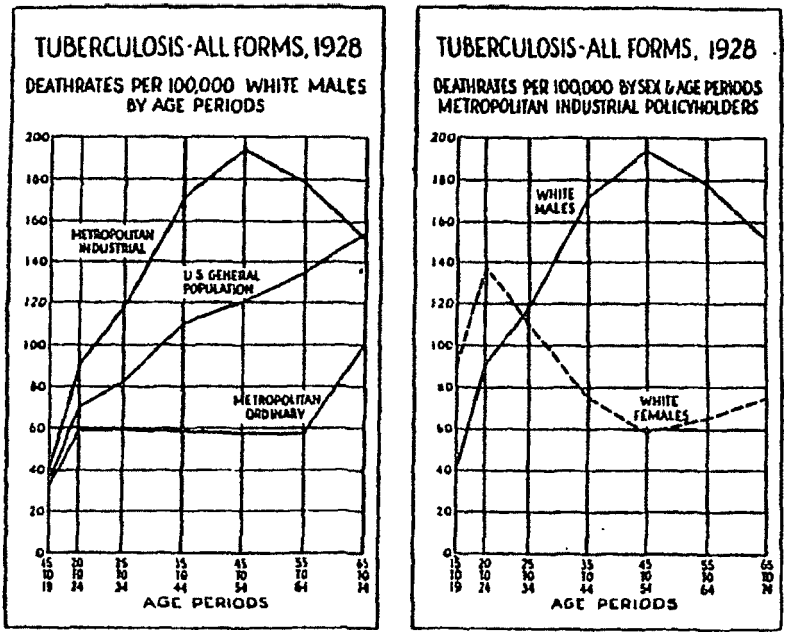
microns in size, 70% were below 1 micron, and 50% were below .6 micron. Results such as these are never obtained with the Owens sampler and it is my sincere belief that the problem of silicosis will not be solved, nor will efficient dust removing machinery be developed until consideration is given to this phase of the problem. In other words, I should like to make a plea for all those involved in the estimation of dust concentrations or the determinations of size frequencies to include in their consideration the small particles —for these are the particles which are undoubtedly responsible for the development of silicosis.

To recapitulate: We have experimental evidence that silicosis is apparently specific, being caused only by silica in several or all of its many modifications. The pathological reaction of silica increases with decreasing particle size. It is desirable to devise a technique for determining dust concentrations which will include all particles above ½ micron in size. It is equally desirable to obtain a size frequency distribution curve for all important hazardous occupations in order that the true hazard may be properly evaluated.

I should like to present some statistical evidence dealing with the known silicosis hazard existing in the lead and zinc mines of Picher, Okla. As has been repeatedly emphasized, silicotics generally die because of a pulmonary infection—usually tuberculosis. This analysis deals with the tuberculosis mortality among miners known to have had previous exposure to silica in or about the mines of Ottawa County, Oklahoma.

In order to establish a foundation on which to properly evaluate the subsequent mortality rates in silicotics let us first examine mortality rates among the ordinary classes of society.

GRAPH "A"



Graph "A" compares the tuberculosis mortality among three classifications of white people by age groups. The class with the lowest tuberculosis mortality rate is composed of those persons who are in a sufficiently favorable economic position to be able to own a $1000 (or more) ordinary life insurance policy. The second group is composed of the general population and represents the millions of policy holders under the group insurance division of the Metropolitan Life Insurance Company. The mortality rates in all these groups are apparent from an inspection of Chart A. It will be observed that the male industrial group has the highest tuberculosis mortality rate and that this rate reaches its maximum between the ages of 45 and 54. It would appear that certain factors in industry are responsible for this increased rate. Chief among such probable factors is exposure to silica dust.

Table 1

A COMPARISON OF MORTALITY RATES FROM ALL CAUSES

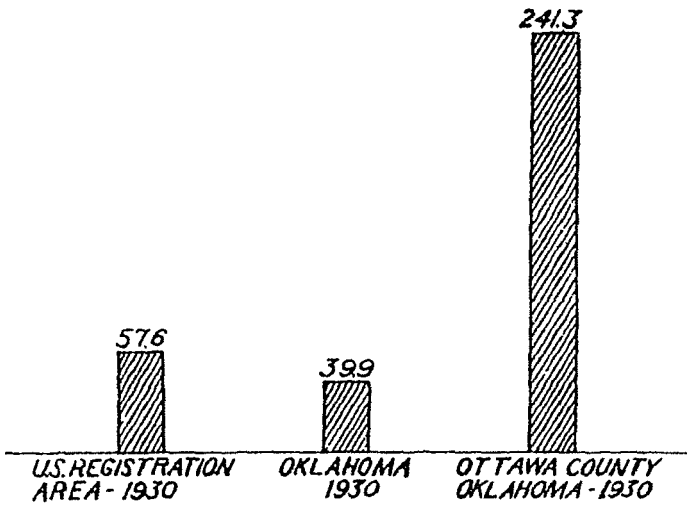
Per 100,000 WHITE POPULATION.

	Year	Total	Year	Male	Female	Ratio Male/Female
U.S.Registration Area	1930	1080.0	1927	1172.6	1021.7	1.15
Oklahoma	1930	778.2	1930	872.1	685.4	1.27
Ottawa Co. Oklahoma	1930	1742.6	1930	2056.2	1412.4	1.45

Table 1 compares the mortality rates (per 100,000 white population) from all causes, by sex, in the U. S. Registration Area, Oklahoma, and Ottawa County, Okla., for the year 1930. It will be observed that the mortality from all causes in Ottawa County (where there is a silicosis hazard) is very high and that it is particularly high among the males.

GRAPH I

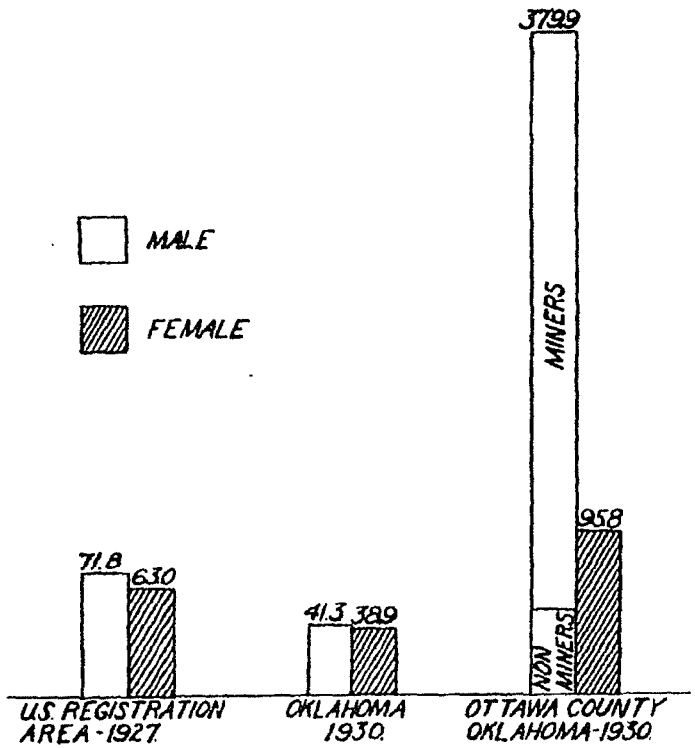
A COMPARISON OF MORTALITY RATES FROM TUBERCULOSIS (ALL FORMS) PER 100,000 WHITE POPULATION ONLY - BOTH SEXES ALL AGES - 1930.



Graph 1 compares the mortality from tuberculosis (all forms) per 100,000 white population, for both sexes and all ages during 1930, in the U. S. Reg. Area, Oklahoma, and Ottawa Co., Okla. It will be observed that the tuberculosis mortality in Ottawa Co., where silicosis is known to exist, is six times as great as that in the state of Oklahoma as a whole.

GRAPH II

A COMPARISON OF MORTALITY RATES FROM TUBERCULOSIS (ALL FORMS) PER 100,000 WHITE POPULATION ONLY - BY SEX.



Graph II compares the mortality from tuberculosis (all forms) per 100,000 white population, by sex during 1930, in Oklahoma and Ottawa County, Okla. Here it is found that the high tuberculosis mortality in Ottawa County occurs principally among the males, whose mortality rate is four times as great as that among females of the same county.

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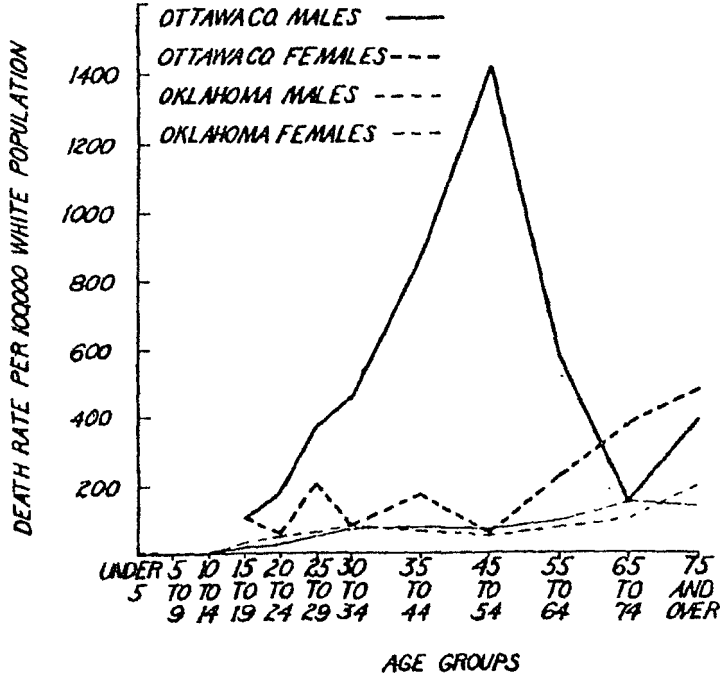
GRAPH III

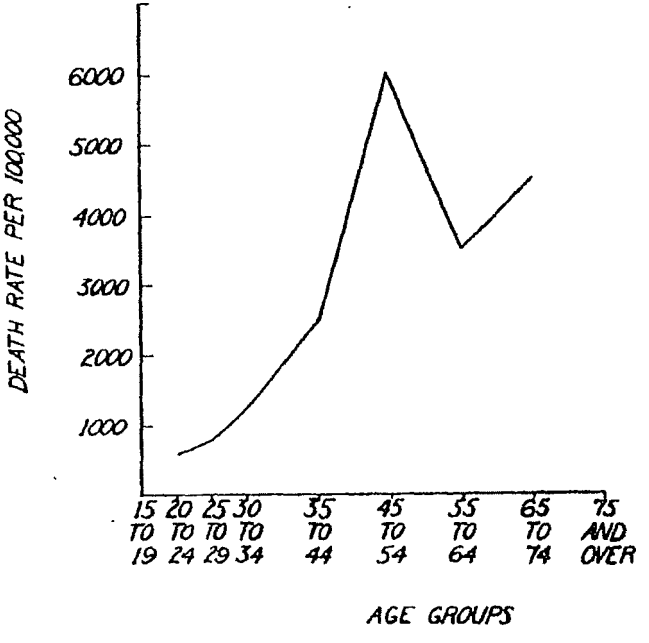
A COMPARISON OF MORTALITY RATES FROM TUBERCULOSIS ALL FORMS) PER 100,000 WHITE POPULATION ONLY - BY SEX AND BY AGE GROUPS - OKLAHOMA AND OTTAWA COUNTY, OKLAHOMA - 1930



Graph III compares the mortality from tuberculosis (all forms) per 100,000 white population, by sex and by age groups, in Oklahoma and Ottawa County, Okla. It is apparent that the male rate in this county is excessive and that this rate increases steadily to reach a maximum of over 1400 per 100,000 at ages 45 to 54.

GRAPH IV

THE TUBERCULOSIS MORTALITY RATE PER 100,000 WHITE MALE LEAD AND ZINC MINERS BY AGE GROUPS - OTTAWA COUNTY, OKLAHOMA, 1930



Graph IV depicts the mortality from tuberculosis (all forms) per 100,000 white male miners in Ottawa Co., Okla., during 1930. The mortality rate shown in this graph is probably the highest ever recorded for a group of civilized white males. The rate rises steadily to reach its maximum of over 6000 per 100,000 at ages 45 to 54, coincident with 25 to 35 years exposure to silica dust.

Table II

Mortality Statistics for Ottawa County, Okla., 1930 - White Population 35468

A total of 629 true deaths*
145 of these deaths occurred among miners - or 23% of all deaths in the county.

Of the 145 Miners
65 Died of TB----44.8%
16 Died of Respiratory Diseases other than TB----11.0%
81 Died of Respiratory Diseases----55.8%

Of the 145 Miners
22 Died of Accidents, Suicides, Burns or Gun Shot Wounds----14.9%

Of the 123 Remaining Deaths Among Miners
81 Died of Respiratory Diseases----65.8%

The Chief Causes of Death, other than TB or Accidents, Among the Miners of Ottawa Co., Okla., in 1930 were as follows:

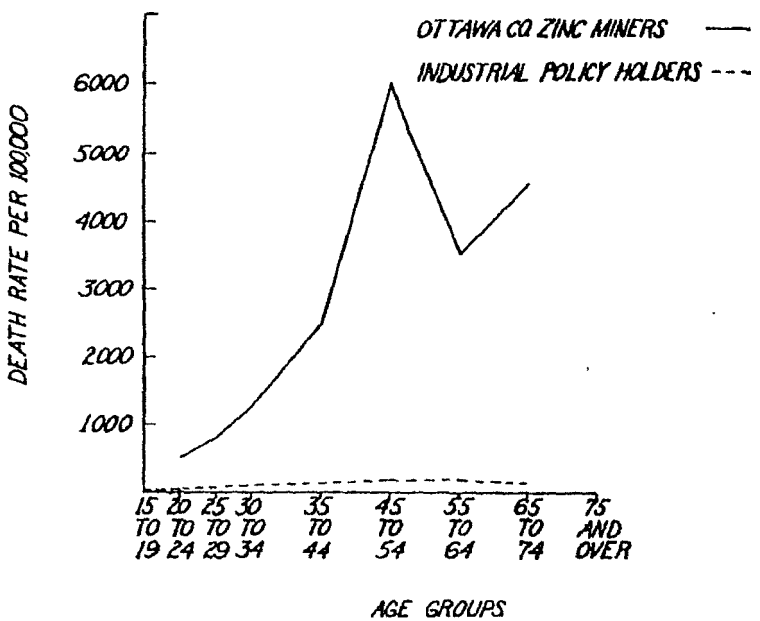
Heart Disease	6.2	% of all deaths among miners.
Senility	4.1	" " " " " "
Nephritis	3.4	" " " " " "
Apoplexy	2.8	" " " " " "
Cancer	2.1	" " " " " "
Parlysis	1.4	" " " " " "
Uremic Coma	1.4	" " " " " "
All Other Causes	7.6	" " " " " "

* 71 still births not included

Table II gives additional data relative to the principal causes of death among white miners in Ottawa County, Oklahoma, for 1930. It should be observed that 56% of all deaths were due to respiratory diseases.

GRAPH V

A COMPARISON OF MORTALITY RATES FROM TUBERCULOSIS (ALL FORMS) PER 100,000 WHITE MALE INDUSTRIAL POLICY HOLDERS OF THE METROPOLITAN LIFE INSURANCE CO AND WHITE MALE LEAD AND ZINC MINERS OF OTTAWA CO, OKLA - BY AGE GROUPS - 1930



Graph V compares the mortality rates from tuberculosis (all forms) per 100,000 white male miners of Ottawa County, Okla., and white male industrial policy holders of the Metropolitan Life Insurance Co., by age groups for 1930. This graph strikingly illustrates the additional hazard imposed by silica on industrial workers.

Table 111

A COMPARISON OF MORTALITY RATES FROM ALL CAUSES AND FROM TUBERCULOSIS AMONG WHITE FEMALES.

	U.S.Reg.Area 1927	Oklahoma 1930	Ottawa County 1930
Death Rate per 100,000 All Causes	1021.7	685.4	1412.4
Death Rate per 100,000 Tuberculosis	63.0	38.9	95.8
Number of Deaths from Tuberculosis in 1000 Deaths from All Causes	61.7	56.8	67.8

Table III compares the mortality from all causes and from tuberculosis per 100,000 white females in the U. S. Reg. Area, Oklahoma, and Ottawa County, Oklahoma. This table illustrates the fact that the tuberculosis mortality among the females, living in a community where silicosis and tuberculosis are outstanding among the males, remains in fairly good proportion to the mortality from all causes as observed in the U. S. Registration Area.

To recapitulate: It can be shown statistically that silicotics suffer from an enormous tuberculosis mortality. This excessive mortality can be attributed to no other cause than the inhalation of silica dust.

A question was asked relative to the incidence of syphilis in the Picher area and its effect on the development of silicosis. This was a problem in which Dr. Meriwether was particularly interested and he was kind enough to show me a large number of cases illustrating his contention that silicosis did develop much more rapidly in the man who had a positive Wasserman than in the man who had a negative Wasserman. Apparently from the data Dr. Meriwether has accumulated, this contention is true. Moreover, he found that in those men who had a positive Wasserman and were

given treatment for their syphilis, the silicotic process also seemed to improve during treatment. As the Wasserman changed from positive to negative, the X-ray films of these men seemed to improve. I think we may draw a fairly accurate generalization that the presence of any infection in a silicotic is likely to result in a more rapidly progressive pathological process than would occur if the infection could be eliminated.

I found quite a large number of cases among the Picher silicotics that had received a diagnosis of suspected tuberculosis on the basis of their roentgenograms. These men had a large amount of sputum but tubercle bacilli were not found after repeated examinations. On further examination of the sputum a number of these men were found to have other bacteria which seemed to be responsible for their pulmonary infections. The main group of organisms responsible for such infections was similar to the organism which produces syphilis. These men were given syphilitic treatment even though they did not have syphilis and some showed marked improvement. Dr. Meriwether continued to carry on this work in Picher and reported that of some 20 men treated all had returned to work after having been disabled for a long time. Whether or not this condition obtains in other communities I cannot say, but I think it is true that a man with silicosis is predisposed, not only to tuberculosis, but to other infections as well. Silicosis constitutes a hazard in causing an increased susceptibility to tuberculosis, but it also increases the susceptibility to other infections. Some of these men with infections other than tuberculosis, may offer more hope for effective therapy than do tuberculous silicotics.

MR. WRIGHT: What sort of mines?

MR. CUMMINGS: Lead and zinc mines in Oklahoma.

MR. WRIGHT: Any lead poisoning mixed up with it?

MR. CUMMINGS: No. They are not really exposed to lead vapors and consequently there is no real lead hazard. They mine this ore out of hard silica rock—called chert.

MR. WRIGHT: Is there a great deal of silica present in this rock?

MR. CUMMINGS: Yes. Over 90% silica. It is not quartz but a flint or chert—a very dangerous dust.

MR. TARRELL: Are they Finnish laborers?

MR. CUMMINGS: No. They are not Finns. They are native American whites. They come from Kentucky and Tennessee and are true early American settlers.

MR. KUECHLE: Have you gotten anywhere so far with your experiments that Dr. Gardner referred to on neutralizing the poisonous effects of silica in the lungs?

MR. CUMMINGS: I believe that silica does dissolve after entering the body, then a soluble silicate is formed and it is present about the particles that are collected in the lymphoid areas. There are certain chemicals which will precipitate soluble silicates from their solutions. Such substances as iron salts, calcium salts and a great many other substances will cause this precipitation. If it would be possible to bring such substances into intimate contact with the dust collected in the silicotic nodules it might also be possible to precipitate this soluble silicate and thereby neutralize its toxic influence, if the soluble silicate is responsible for this toxicity. There is a fairly good illustration that this may be true. Asbestos is a magnesium silicate in which iron usually replaces part of the magnesium in the molecule. When asbestos is inhaled into the lung the individual fibres tend to dissolve or hydralyze. The fibres swell and some soluble silica is formed. Iron is released by this hydrolysis and it recombines with the soluble silica to form an insoluble substance. Now it so happens that in asbestosis a definite fibrosis develops but there does not seem to be a marked increase in the susceptibility to tuberculosis in spite of this well established fibrosis, or asbestosis. It may be possible that the iron which constitutes a part of the asbestos fibre is responsible for overcoming the increased susceptibility to tuberculosis common to most cases of pneumoconiosis.

MR. DOE: Dr. Gardner, I gathered from your discussion that there is a great deal of difference between the reaction of silica and other dusts with regard to the character of formation, that the scar tissue formation is characteristic

of silica and not characteristic of the other dusts you examined?

DR. GARDNER: That is true.

MR. DOE: Can that difference be detected on X-ray and if so, how?

DR. GARDNER: I don't believe the character can be detected by X-ray, no. The distribution of the reaction is the thing that we have to rely upon in the X-ray. Possibly the density of the reaction, but even that would be likely to fall down. It is only when you have the tissue under the microscope that you can be sure. Of course, you can on gross examination of the lung determine density to a certain extent by palpation, but the characteristic thing about the silica reaction is its microscopic appearance.

MR. DOE: What would you say as to the characteristic difference in distribution for instance, if you were interpreting an X-ray and did not know what the chemical composition of the dust was and were trying to determine whether the manifestations on the plate were silicotic or otherwise, what would you look for to make that differentiation with regard to the distribution of the pathology?

DR. GARDNER: As far as we know today—I am not a roentgenologist and cannot qualify as an expert, but as far as we know today there are only two types of dust that will produce extensive pulmonary changes visible by the X-ray; one is pure silica in one form or the other. That produces a generalized fibrosis of the lung characterized by increase in the linear markings and characterized by the formation of nodular shadows. Such changes tend to be pretty uniformly distributed throughout both lungs with the exception of small areas over the diaphragm which are generally left uninvolved. Asbestos dust will also produce a generalized fibrosis of the lung, but it is not accompanied by the formation of discrete nodular shadows. It is a diffuse fibrosis and widespread. We also know that slate workers working in a silicate get a good deal of diffuse fibrosis. I believe Dr. Russell is prepared to discuss slate workers. These slate workers also get diffuse fibrosis, but not generally as marked as that which occurs in the asbestos group.

It may be in some instances. The reaction to the other types of dust is insignificant in our experience as compared with that produced by pure silica or by the silicate asbestos and perhaps the silicate slate. When we examine the lung tissue of the asbestos worker we find that the amount of reaction is apparently much greater than would appear from the X-ray plate. Just why this should be is puzzling, but we have not had opportunity to study as many asbestos workers as we should before we can answer this question.

MR. DOE: Then the discrete character of the distribution and the absence of nodules, that is, the absence of the generalized distribution and the absence of nodules would tend to rule out silicosis?

DR. GARDNER: Yes.

MR. DOE: In your opinion?

DR. GARDNER: In my opinion, but this must again be qualified because we may deal with mixtures of dust. Dr. Russell in his work in Barre, failed to find nodular fibrosis in the lungs of many of the granite cutters exposed to a dust containing as much as 30% free silica combined with several silicates. It is my belief that probably the silicates and other non-siliceous substances modified the action of the silica so that the nodular lesions which would be characteristic of pure silica failed to develop. We know the clear cut and definite picture of silica alone. That picture is characteristic and on that one I don't think we should fall down. When we deal with silica complicated by other substances or when we are confronted with silicates alone we are not in position to be so definite.

MR. DOE: When you speak of aluminum oxide, do you use that term in the same sense that Dr. Clark does, that is, when the percentage of silica is so small, 1.76, that for all practical purposes in this inquiry it can be disregarded?

DR. GARDNER: I believe that would be a fair view, yes.

DR. CLARK: Combined silica.

MR. DOE: I meant as you described it, Dr. Clark, would that apply in your opinion Dr. Gardner, to other dusts in

combination, if the percentage of silica was below, say two per cent, that it could be regarded as a non-siliceous dust?

DR. GARDNER: I think it might be. I can conceive of conditions where other substances might favor the reaction of silica itself. I don't know of any such one, but it seems conceivable there might be.

MR. DOE: In the work you and Mr. Cummings have done what possible combinations that tend to minimize the effect of silica,—he mentioned iron,—are there others?

DR. GARDNER: Theoretically iron, calcium and aluminum might combine with this silica dissolved in the body fluids and prevent its reaction with the body tissues. We have selected all these as possibilities and hope to experiment with them.

MR. DOE: With regard to these particles of less than two microns, they wouldn't reappear in the lungs of the experimental animals, have you an opinion as to whether that has any significance in dealing with humans? Would there be an anatomical difference which might result in particles less than two microns localizing in the lungs of human beings and not in rabbits?

DR. GARDNER: I think if we were successful in obtaining a sufficient quantity of particles less than two microns in diameter that probably many of them would remain in the lung, but I do not know. We have not been able with our apparatus and money available to us to produce such a quantity and we have not been able to investigate the subject. We had to turn to the intravenous method of experimentation because of the limited amount of material available.

MR. DOE: What is the mechanics of those tiny particles in the rabbits getting to the liver?

DR. GARDNER: From the ear vein they are carried through the right side of the heart into the pulmonary artery; they go directly through the capillaries of the lung and are not filtered out there as we had hoped that they might be. They are carried back through the pulmonary vein into the left side of the heart and from the left side of the heart pumped out to the aorta and the general circulation. The

liver is a very good filtering organ, and so is the spleen. They tend to circulate about until they happen to lodge in one of these filtering organs.

MR. CUMMINGS: It should be brought out that if they are inhaled the mechanism would be different than if they came into the lung by way of the blood stream. In the blood they are inclined to be rushed on through just as the blood corpuscles, because there is nothing to cause them to come out, whereas, if they are inhaled they would lodge inside the air space and as a consequence I believe they would be picked up by phagocytes and carried to the lymph nodes.

MR. DOE: Dr. Gardner, in the examination made at post mortem of silicotic individuals, I think you said sometimes silicotic nodules were found in the liver?

DR. GARDNER: Yes.

MR. DOE: In human beings?

DR. GARDNER: Yes.

MR. DOE: Presumably, the only silica to which that individual was exposed was by inhalation?

DR. GARDNER: Yes.

MR. DOE: How would those particles get to the liver?

DR. GARDNER: We conceive that in this case there has been an overflow of dust cells from the tracheobronchial lymph nodes at the root of the lung. This overflow has carried these particles perhaps inside phagocytic cells to the venous circulation, by which they are delivered to the right side of the heart; the right side of the heart pumped them through the lung. There they may not be filtered out by the pulmonary capillaries any more effectively than were the particles which were injected into the ear vein of the rabbit. They would be carried back to the left side of the heart into the aorta and finally be caught in the more efficient filter of the liver.

MR. DOE: That may be one of the processes that normally goes on may it not?

DR. GARDNER: I would believe so.

MR. DOE: In ridding silicotic lungs of dust?

DR. GARDNER: I would believe so. But it is comparatively rare to find silicotic nodules in the liver and spleen of human beings. It may be that these particular human beings who show such lesions are abnormal in some anatomical respects and it may be, this is something we have never checked up on, that these nodules in the liver and spleen are composed of extremely fine particles and it may be that this particular individual was inhaling an excess number of fine particles, instead of the more usual ones two to eight microns in diameter.

MR. DOE: Do you believe that it would be a possibility for men working as machinists, that is, on drill presses or borers and machines of that kind, to create sufficient dust to institute a hazard for siderosis; that is, where there is no sand or no artificial abrasive in the process, just the use of the metal itself?

DR. GARDNER: I rather doubt it. I do not believe we know whether there is such a thing as siderosis. We talk about the condition. We know that certain hematite miners have red lungs. We do not know whether iron in any form alone is sufficient to produce a reaction. I have seen the lungs of one boiler maker who had a long exposure, apparently to iron dust. We were not able to get a good occupational history. This man had a peculiar type of fibrosis in his lungs without tuberculosis. He had a large amount of iron in his lungs. It is impossible by any method that we know of to determine whether that iron was from an external source or whether it came from within the body, because iron is one of the components of body tissue. Possibly siderosis may be due to contaminating silica more or less modified by the excessive amounts of iron in the dust.

MR. DOE: Is there any scientific basis for assuming now that the inhalation of iron and steel particles from processes such as polishers, drill presses and the like where there is no combination of silica, that that predisposes to tuberculosis?

DR. GARDNER: None that I know of. I would be inclined to believe that we would not find any.

MR. DOE: Doctor, when you have the reaction which you described as being characteristic of asbestos and characteristic of other dusts not containing a high percentage of free silica, what is the fact as to whether or not the incidence of tuberculosis among those individuals is higher or lower than the miners in general?

DR. GARDNER: I have not had any opportunity to observe such groups myself. Our experimental work tended to show that we could stir up a non-virulent tuberculous infection temporarily but it would not progress to any extent. There would be a temporary reactivation of the latent focus and then the lesion would heal with formation of considerable scar tissue. While there have been autopsy reports of human beings dying with a combination of tuberculosis and asbestosis, so far as living individuals working in the asbestos industry are concerned, observation generally tends to show that the incidence of tuberculosis is not excessive.

MR. DOE: Is there any information available that you are familiar with that the incidence of tuberculosis is higher in any industry or any dust exposure in the country at large, where the silica content, we will say, is less than 2%? I mean to include in that silica, rayon, wool, tobacco, iron, and in combination with silica, and millers and packers and flour mills. All sorts of people.

DR. GARDNER: There are the statistics of Dr. Hoffman reported in the Public Health Service reports, but I think these should be examined quite critically and possibly should not always be accepted at their face value. There are other factors that come into play besides the one of dust inhalation which might be responsible for the increase in tuberculosis in these trades. I do not know of any figures sustaining the point of view which you mentioned.

MR. DOE: Supposing that the exposure in a case containing, we will say, less than 1%, just a trace, a normal amount of silica, was very protracted—some cases like those which Dr. Clark told us about for thirty to forty years. Would that make any difference in your last conclusion?

DR. GARDNER: Our experience with carborundum dust might be considered a substantiation of such a view. We were at a loss to explain it in any other way. We also found that marble dust, which contains only a fraction of one per cent of silica, exhibited a slight tendency to cause the tuberculous foci to become progressive for a short while, but then they again retrogressed and healed. It is possible that if the exposure to silica in minute amounts were continued for a sufficiently long time and that in the meantime the tuberculous process could remain potentially active, that one could reactivate a tuberculous focus.

MR. DOE: In case of non-siliceous dust, Doctor, is there any distinction to be taken between causing tuberculosis in the first instance or rather more accurately predisposing to the disease and reactivating an old process?

DR. GARDNER: I do not know. I don't think I could answer the question.

MR. DOE: Well, on the basis of the knowledge presently available, you have already testified that there is no scientific basis for testifying that there is more prevalence among those employed in silica than among the population at large. Would your answer be the same as to those persons who had had tuberculosis and had gone into those occupations?

DR. GARDNER: I would more or less agree with Dr. Clark that any kind of dust would be a bad thing for a person with a potentially active tuberculous focus in his body. I would hate to see him for his own good go into dusty industry. However, I do not know enough about it to do more than theorize. I would not want to risk working in dust if I myself had such a focus.

MR. DOE: My point is, will anything in the present state of our knowledge establish the fact that there is any risk?

DR. GARDNER: No, I don't think we have any definite information.

MR. DOE: Is there any way of telling, Dr. Gardner, when you see a case of tuberculosis and there is exposure

to a non-siliceous dust, whether or not that particular case of tuberculosis became superimposed on the pneumoconiosis or upon the reaction which might be described by any other name than silicosis, or whether the latter merely is co-incidental with tuberculosis?

DR. GARDNER: That is a very difficult question to decide. We can only surmise that when the tuberculosis is progressing in a more or less normal way, starting from an apical focus and gradually extending downward, but more rapidly than usual that then perhaps the dust may have been instrumental. However, we have no accurate basis of information.

Thursday, November 17, 1932. Morning Session

MR. DOE: Dr. Gardner, yesterday in Dr. Clark's account of his observations in Worcester, he said that in writing these monographs he had intended to limit the discussion to the results of his studies in connection with the reactivation of old tuberculosis by artificial abrasive dust; in your work have you come upon anything which questions the correctness of Dr. Clark's conclusions?

DR. GARDNER: When I discussed our experiments designed to reactivate latent tuberculosis infections, I mentioned the fact that in guinea pigs we were able with carborundum dust to produce reactivation in a certain proportion of animals. This result we have been at a loss to explain on the basis of our present knowledge. The only assumption which we could make was the fact, as Mr. Cummings suggested, that there might be in the particular carborundum that we used, a certain amount of free silica and that possibly this free silica was responsible for the reactivating effect. The carborundum that we used in our experiments came from a Niagara Falls plant and not from the same source as that of Dr. Clark's. Both are commercial products, said to be composed largely of carbide of silicon, but there might very well be differences in them. We did not make a chemical analysis of the ma-

terial which we used; we accepted the manufacturer's analysis.

MR. DOE: From these experiments on guinea pigs would it be your judgment that there is any justification for assuming that human beings might react differently; that is to say, that Dr. Clark's experiments, if the analysis were the same, might be correct as to humans and also as to guinea pigs and there would be no conflict between those two results?

DR. GARDNER: This is possible, of course. We have always attempted, when we obtained experimental results, to check them as closely as possible by what was being found in the field of human pathology. In the case of carborundum we have no data on human beings except what Dr. Clark reports.

MR. DOE: Have you had at the sanatorium at Saranac Lake, cases of grinders using an artificial abrasive, either carborundum or aluminum oxide?

DR. GARDNER: We have never had grinders. We have had several employes of the Carborundum Company at Niagara Falls, who have shown an extensive tuberculosis and these men have given a history of rather long contact with the dust, but they have not been grinders. Many of them have been office men. The office men, however, were in a position to inhale a good deal of dust.

MR. DOE: Is there any means of telling, in a case such as you have last described, as to the duration of the tuberculosis with reference to the dust exposure, can you give in a given case whether the tuberculosis is recent or old and if so, how do you tell?

DR. GARDNER: The cases as they came to us were rather far advanced pulmonary tuberculosis and it was not possible in any of them to date the onset. I say this with reservation, because I did not see them clinically. I saw their X-ray films and heard them discussed but I could not give you accurate information.

MR. DOE: Dr. Gardner, what or who do you consider are the leading authorities on the subject of silicosis in Germany?

DR. GARDNER: Boehme is probably the best observer in Germany. Ickert has also done extremely good work; I think these two are the outstanding ones.

MR. DOE: Who would you say are the outstanding English workers at the present time?

DR. GARDNER: Kettle in the experimental field and Middleton in the general field of public health.

MR. DOE: Now, in the United States, with the exception of the gentlemen who have been here, are there any people in this country that you regard as authoritative on silicosis problems?

DR. GARDNER: I would include the men who have been invited to speak before this group. Dr. Sayers of the Bureau of Mines, Dr. Lanza of the Metropolitan Life Insurance Co., and Dr. Pancoast of the University of Pennsylvania, Dr. Meriwether of the Bureau of Mines, and Dr. Drinker of Harvard University. I would also include Dr. Britton and Dr. Head who have recently become interested in the subject.

MR. DOE: Dr. Gardner, suppose that a case of a man who had been employed as a grinder, using artificial abrasives, not including carborundum, were to come to you for study to determine whether or not his tuberculosis from which he was suffering was occupational, what would you do to try to get at the correct answer to that question, how would you go about it if you were the physician whose decision would furnish the answer to whether he was a compensable case or not?

DR. GARDNER: Of course, one would need an accurate occupational history with a survey of the dust concentration in the atmosphere in which he worked, a knowledge of the dust itself, its chemical composition, its petrographic composition; one would want to know when his tuberculosis first became manifest, whether he had had contact with tuberculosis previous to his entering the dusty industry; finally, one should have a careful clinical and radiographic study of the man himself. In other words, I would try to find out everything I could about the individual and his history.

MR. DOE: Do you have an opinion, Dr. Gardner, as to the percentage of adults that contract tuberculosis by contact?

DR. GARDNER: Opinion is changing on this subject. It used to be commonly believed among the group at Saranac Lake that the majority of the cases of adult tuberculosis were due to a recrudescence of a childhood infection which later became lighted up by physical or mental strain. When I came to Saranac Lake in 1917 the camp was more or less divided in its opinion on the subject. Dr. Baldwin thought practically all such cases were reactivated childhood lesions; Dr. Lawrason Brown on the other hand thought that many, perhaps 75%, were re-infections from the outside occurring in adult life. Today I think the two authorities are willing to admit that there is an even chance for either to occur. One cannot tell accurately and absolutely where the new infection comes from; whether from within or from without. The opinion seems to be more or less prevalent throughout the world that occupational tuberculosis with which we are dealing, is in many instances a new infection from without. But even after careful examination at post mortem one can only guess whether one or the other mechanism of infection is involved and some times one can't even guess. From the experimentalists' standpoint I have been influenced by the observation that a previous infection tends to immunize against subsequent infections but recently we have had experience which makes me believe that the immunity conferred by one injection of tubercle bacilli is not a steady and constant affair, but that it may fluctuate. We now believe that there may be periods in the life of immunized animals when resistance is even lower than that in a normal animal. At such times the individual may be even more susceptible to infection from without than if he had never been injected. This is still more or less speculative and we have no absolute proof.

MR. DOE: In examining a hypothetical individual such as I have stated, suppose that evidence was produced of contact with an active case, how would you evaluate the significance of that in coming to a conclusion as to whether the tuberculosis was industrial or not?

DR. GARDNER: If the individual had a well developed silicosis, if he gave a history of having had contact with silica in doses of sufficient concentration to produce a characteristic picture of silicosis and if in addition to that he had a generalized more or less acute tuberculosis, I would certainly think that the individual probably was infected from without, rather than having lighted up an old preexisting infection. That is on the basis of our experience with experimental animals.

MR. DOE: I mean to state a case, Doctor, in which the dust inhaled is aluminum oxide, so that you have a case where a man who has been employed in such an occupation and has had actual contact with an active tuberculosis case, obviously outside of his employment, what significance would you give to that contact?

DR. GARDNER: If I knew nothing about the man previous to his entry to the industry?

MR. DOE: Yes.

DR. GARDNER: I don't believe I would be able to decide, that is, if he were still alive. Of course, you do not give me the benefit of an autopsy. I should, of course, seek the help of some good clinician for I myself am not a clinician, but I don't think from the information that you would allow me I could even guess.

MR. DOE: What is your personal opinion about what you might call the percentage of cases of adults that are infected from one another, would you say that was low or high?

DR. GARDNER: There is a good deal of evidence against adult infection among normal individuals who do not have the additional factor of inhaled dust in their lungs. There are plenty of figures which can be produced to show there have been some individuals living intimately with one another that do not infect each other. If you had the factor of aluminum oxide in one member of the group, or pair, from my personal experience I would rather be inclined to believe it would not affect the situation very much, however, this is only a guess.

MR. DOE: Have you an opinion, Dr. Gardner, as to the number of million particles of concentration of aluminum oxide dust that could for all practical purposes be considered safe for workmen over a long period of time?

DR. GARDNER: I would rather leave that question to my colleague, Mr. Cummings.

MR. CUMMINGS: The study made at Barre, Vermont, by the U. S. Public Health Service showed that the men exposed to granite dust, in a concentration below ten million particles per cubic foot of air, seemed to have a much better mortality experience than those in which the dust concentration was higher. For this reason they separated the group exposed to less than ten million particles from the others under investigation because the sickness and mortality rates in this group justified such a distinction. These investigators felt that five million particles of granite determined by the method used by the Public Health Service represented a fairly safe concentration of dust. Evidence obtained in the Saranac Laboratory would indicate that granite is more potent in producing a fibrosis or in reactivating a tuberculosis, than aluminum oxide. Consequently, I would feel that five million particles of aluminum oxide in a cubic foot of air (determined by the same method) could be considered a safe concentration for men to work in over a long time.

MR. DOE: When you say "long time" what do you mean?

MR. CUMMINGS: Thirty-five to forty years, sir.

MR. DOE: When you say "safe", do you mean from a disability through pneumonoconiosis or to tuberculosis, or both?

MR. CUMMINGS: To a pulmonary fibrosis. I think one has to have that fairly well developed before the increased susceptibility to tuberculosis becomes very marked.

MR. DOE: Would you say that for aluminum oxide containing less than two per cent silica that that figure of five million could be substantially increased without changing the result?

MR. CUMMINGS: I believe it could be, sir, but I wouldn't be sure. We haven't definite evidence, either clinical or experimental, to substantiate that fact.

MR. DOE: Let's take a hypothetical case—suppose that the concentration were ten million; could you estimate the period of time that such an individual could work without impairing the risk of a tuberculosis complication?

MR. CUMMINGS: I might answer your question indirectly. A great many men employed by the Norton Company, (whose main product is aluminum oxide and not carborundum) have undoubtedly been working in a concentration of ten million particles or more for years; and as you know, the films which Dr. Clark presented failed to indicate a marked pulmonary fibrosis that could be regarded as significant. Neither does tuberculosis appear to be a great problem with the Norton Co. Consequently I feel that ten million particles of Alundum per cubic foot of air could be tolerated by a normal individual over a period of a great many years—more than twenty years —without serious damage. Our evidence with regard to the possibility of that man acquiring a new infection or reactivating an old one doesn't permit us to give any accurate information as to what might happen to him in the presence of that infection.

MR. DOE: When you speak of five and ten million particles, Mr. Cummings, do you mean by that under ten microns in size by method of computation that the Bureau of Mines has used?

MR. CUMMINGS: Yes.

MR. DOE: From the pictures which Dr. Clark exhibited yesterday would you say that those men with the chest findings that appeared were working in a safe concentration from the standpoint of either tuberculosis complication or a disability from dust inhalation?

MR. CUMMINGS: I don't believe that we have evidence enough to make a statement with regard to the safety of any concentration of dust in which tuberculous individuals may work. I think that so far as we know, the con-

centration of dust they have been working in is safe if we consider only its ability to produce a pulmonary fibrosis.

MR. DOE: What I meant was from the films themselves, in the absence of fibrosis, is there any scientific basis for saying that those particular individuals are in any particular danger of tuberculosis infection so far as their employment is concerned and their chest findings?

MR. CUMMINGS: It is true that the greater the extent of the pulmonary fibrosis which develops after exposure to silica the greater the susceptibility, and the greater the mortality from tuberculosis will be. Therefore, in the absence of a pulmonary fibrosis in these men, one would feel that no increased susceptibility to tuberculosis existed. However, there is one exception which we have repeatedly pointed out. Experiments with carborundum dust would indicate that even though it fails to produce a pulmonary fibrosis there did appear to be an increased susceptibility to tuberculosis in the animals that had inhaled this dust over long periods.

MR. DOE: Then every one of Dr. Clark's employes, if it did so happen he never had been exposed to carborundum dust and the chest picture was substantially as shown here yesterday, you would say that individual was not more susceptible to tuberculosis than he would have been had he had the exposure to aluminum oxide?

MR. CUMMINGS: I would say that positively, but it wouldn't be substantiated by complete experimental evidence at the present time.

MR. DOE: Is there any experimental evidence to the contrary?

MR. CUMMINGS: There is not.

MR. DOE: That is all.

MR. TARRELL: In your answer to these questions of Mr. Doe you have assumed a hypothetical case of exposure only to aluminum oxide dust?

MR. CUMMINGS: Yes, sir.

again as I see these questions come up, it is a matter of getting important necessary data concerning every individual industry and every individual person concerned. There are many dusts undoubtedly that do not harm at all and we can't regulate the amount of particles of that dust which should affect a person with 1/10 or 1/1000 as much dust of another sort which might do serious harm to an individual in breathing it. It seems to me it comes down to the question of types of dust which are known to do harm and an effort should be made to find out what sort of harm those dusts do. That may be an impractical sort of thing, but it is certainly something that wants attention.

DR. CLARK: I am going to talk at length on the subject a little later and show some films.

DR. BELLIS: Are we to interpret the expression "harm to the lung" as merely the production of fibrosis or are we to interpret it as an irritation which would favor the onset of infection? We certainly must agree that the inhalation of dust is not a hygienic procedure and that infection can ride into the lung on particles of dust, so that dust inhalation is certainly harmful, but if we are to have this discussion of silicosis and formation of fibrosis as being the only harmful effect of dust, then we have to consider the other dusts as not harmful. It seems to me that where we see cases of exposure to emery dust that develop tuberculosis and where post mortem examination does show marked evidence of irritation from dust that we cannot say that inhalation of emery dust is not harmful.

November 16, 1932. Afternoon Session

DR. CLARK: Dr. Gardner has given you an excellent outline of the physiology and pathology of the lung. In doing so he has mentioned that there is apparently a different action or reaction of the body tissues, especially of the phagocytic cells to different types of dust. He has pointed out to you that the reaction of the cell to silica dust differs from the reaction of that same cell to carborundum dust.

I am going to talk to you for a few minutes on the work which I have been doing for the last twenty years in the Norton Company in following up the clinical effect of the inhalation of aluminum oxide and carbide of silicon dust on the lungs of the workers. This manufactory takes these two substances in their crude form and develops them into grinding wheels so that the workers are exposed to the dust from these two materials in rather large quantities. In the past they were exposed to what I should now consider excessive amounts of dust, because as you know the development of dust removing apparatus has been comparatively slow, and for that reason for many years a number of these men who have been employed many years have been exposed to very large quantities of dust in their early experience. This has been reduced as time has gone on, until now the dust hazard is very materially less than it has been at any time in the history of the company. We must remember in considering these cases that they have been exposed to very massive inhalation of aluminum oxide and carbide of silicon dust. They have also been exposed to a similar quantity of clay dust, because clay is mixed with the artificial abrasive in the manufacture of the wheel, and they have also been exposed to the dust of what is called the fired bond of the wheel. The bond is the clay after the wheel has passed through the kiln and has been burned and comes out hard in the form which you see the ordinary grinding wheel. It is the glue which holds the particles of the grinding wheel together. I imagine that the majority of the men here are interested in the effect of the inhalation of the dust of the grinding wheels and also of the dust of the artificial abrasive used in polishing. I would have you bear in mind there are three things you must consider—the effect of the artificial abrasive dust which is thrown off in the process of grinding, the effect of the very small quantity of bond which is present in the wheel and which is broken off and becomes dust, and the effect on the lung of the dust of the material which is being ground. In the majority of cases this material is steel.

In the construction of the grinding wheel we have a series of grains of various shapes which are held together by the bond. I have thickened up the spaces to show

the bond which glues these grains together, and if you put a grinding wheel under high powered glass, you will see it looks something like a surface of a fine sponge. The separate grains will stand out glued together by this bond. What are these two substances? The average composition of aluminum oxide, and this is an average composition covered by samples taken over eight years in our laboratories and kept for manufacturing records, is combined silicates 1.71%, iron oxide 46%, titanium oxide 3.27% and aluminum oxide 94.56%. The great bulk is aluminum oxide; there is a very small proportion, 1.71%, of the combined silicates. Silica is not present as free silica, but is probably confined to the complex silica slag between the crystalline alumina. This supposition is confirmed by petrographic examination. Iron is reported as ferric oxide, an inherent custom, although it is present almost certainly as ferrous oxide by virtue of the strongly reduced conditions under which abrasive is made. Titanium is also properly in a lower state of opacity than TIO_2.

The other substance we will consider is carbide of silicon. Carbide of silicon is theoretically a silicon compound of 100% purity. On account of the fact that the sand, coke, and sawdust used in the manufacture are themselves less than chemically pure, the product contains about 98% silicon compound and about 2% associated impurities. Now the effect of the inhalation of these dusts I have studied clinically for a period of about twenty years. I have published the results in three studies. The first one is "Dust Hazard in the Abrasive Industry", in which I examined a group of men who had been employed in the company for over ten years. These men were examined by physical examination and X-rays were taken of their chests. A little later I made a second study in which I reviewed the study made three years before, re-examining the chests of these men by X-ray and again having physical examination made of the chest. Each year we have continued to make physical examinations of the chest. However, we have not taken that original group and re-x-rayed them because pathological change was apparently occurring in such a slight degree that I felt it was safe to wait three or four years between X-ray examinations. The re-

examination of the chest by X-ray will probably take place this winter on this original group. Since that period we have examined two other groups who have been exposed to ten years inhalation of artificial abrasive dust. Our experience in physical examining these men is negative. We find that they are apparently perfectly healthy men. We have men who have been manufacturing in this business for many years, some of them who have been there forty years, and they all appear to be in normal condition so far as the chest goes, considering the period of time which they have been working and the age of the men and type of work they have been doing. So far as the chest goes we cannot find on physical examination any great abnormalities. It is only on the X-ray that we can find that the men have been working in a dusty atmosphere. In order to show you what the effect of these less harmful dusts are upon the chest, I have brought some X-ray films. Before I show these, I thought it would be of interest to you to know how wide spread the use of aluminum oxide and carbide of silicon wheels is in industry at the present time, because I think this will bring to us the importance of these dusts.

I have this letter from our Market Research Department: "Grinding wheels and abrasive grain began to be widely used in 1904, and their use rapidly increased up to 1929. These substances, therefore, have been in use in large quantities for about twenty-eight years. The following figures give a rough estimate of the tons or pounds of manufactured abrasives consumed in the United States in 1929, 1930, and 1931. In the year 1929 there were 53,000 tons of wheels or 106,000,000 pounds of wheels consumed; in 1930—22,000 tons or 44,000,000 pounds; in 1931—33,000 tons or 66,000,000 pounds. Of polishing grain including the coated product, in 1929—23,037 tons; in 1930—11,750 tons; in 1931—9,750 tons. The total wheels and grain used in coated products in 1929 was 76,037 tons; in 1930—33,750 tons, and 1931—42,750 tons."

I have made up a selected group of X-rays endeavoring to select men of various numbers of years of service and of various ages. We don't know how much dust exposure these men had before they came to work for us. It is very

difficult when a man has been working for you from seventeen to twenty years for him to remember anything about work he did before he came to your company. It is difficult to find out whether these men have been exposed to dust prior to their entering our employ and some of them undoubtedly worked in places where there was a great deal of dust, where, for instance, there may have been sand blasting, and, therefore, we may not have a pure picture of the result of aluminum oxide and carbide of silicon dust inhalation. You will see that these chest pictures show perhaps a little more marking than the normal man of that age would have, but there are none of those patches of fibrous tissue that Dr. Gardner has spoken of.

Here is a picture of the chest of a worker thirty-seven years old with sixteen years exposure. There are some little spots along in here showing that there is some blocking of the lymphatics, probably due to deposits of artificial abrasive. I would very much like some who are experts on X-ray to express opinions on any of these chest pictures. These men are working at the present time without any apparent trouble. The next picture is of a man having sixteen years exposure, age 39. This man has been exposed to artificial abrasive dust and also to a clay dust. Now I show you a picture of one year more, 18 years exposure. This man has been exposed to the dust due to truing of wheels. That work is extremely dusty. He has been exposed to pretty heavy dosage of dust for that period of time. You will notice again that while there is a very distinct increase in linear markings, it is a pretty fair looking lung.

Now we get up to twenty years. This man has been exposed to heavy dosage of the pure abrasive dust. Very heavy dosage. And you will find that there is a tendency to a clouding throughout the chest here. There are some little speckles around throughout, but there are no areas of consolidation such as you will see when Dr. Russell will show you some pictures of the fibrosis that goes with silicosis. This is a case of aluminum oxide exposure. Our next patient is 63 years old, and here is a point I think we should bear in mind. When we get a man 63 years old he shows a certain amount of fibrosis in his chest

whether he works in dust or whether he does not, at least that has been my experience. I have had pictures taken for conditions other than dust inhalation. Most of the men around 63 show some fibrosis of the chest whether they have been working in dust or not. I would like to have Dr. Gardner discuss that, if he will.

Here is a picture of a man exposed for 24 years. He is 38 years of age. He began work very young, probably working with us from the time he began. He has been exposed to large dosages of artificial abrasive dust. The dust was composed of aluminum oxide in large quantities with a small quantity of carbide of silicon dust.

Here we have the picture of a man 56 years old, whose work was the grinding of wheels down to the proper size. This man has thirty years of service. Here again we see very much the same picture, little linear fibrosis through the lungs, but no fibrotic areas. This man looks the picture of health. I saw him about two days before I left, and he is a very happy, strong man who is doing regular work and so far as I know hasn't lost any time at all except from an attack of rheumatism that he had. That is how I happened to see him.

Here is a man 55 years old working in the same department, with 37 years exposure. Perhaps a trifle more pronounced lung markings, but not very much more than those I have showed you. I do not notice that there are any fibrous areas.

Last of all I show you the film of a man of longest service, 42 years exposure to abrasive dust, and mixed with that a little clay. He is in the Shaving Department which is extremely dusty. He was 72 years of age when this picture was taken. He has since died of cancer of the prostate gland, not from any lung condition.

I have tried to show you different stages of the effect of artificial abrasive dust inhalation over a period of years. Now I am going to show you two cases of men of more advanced pneumoconiosis with a shorter period of exposure. Here is a little Italian who weighs 128 pounds, five feet high. He was exposed only thirteen years. His film shows a little bit more fibrosis. A little thickening suggestion in there, and he has some thickening of the pleura

between the upper and middle lobes of the right lung, showing silicosis.

MR. KUECHLE: Do you mean silicosis?

DR. CLARK: No. I mean pneumoconiosis. We use that term so frequently. Drs. Pancoast and Pendergrass call everything silicosis. They do not use the word "pneumoconiosis" very much.

MR. TARRELL: What was that statement?

DR. CLARK: Dr. Pancoast and Dr. Pendergrass are very apt to use the word silicosis. They very often do.

MR. TARRELL: You said they used it entirely.

DR. CLARK: Here is another man forty years old who, I think, shows some suggestion of reaction in his lung. That is, we gave him perhaps second stage on that. He is 40 years old, perfectly healthy, weighs 209 pounds. In fact, he has gained five pounds in the last three years.

Those last two films are the worst that I was able to find in my series of chest pictures. They both of them are rather short exposure to dust. I am unable to determine whether that short exposure means that they were working in dust before they came to work in the Norton Company, or whether that was all due to aluminum oxide. I have no way of knowing. It is almost impossible to follow up the exact work record, so I think we will have to take the blame for it in our particular industry.

Now as to the physical condition of these men—I have here their original records; I don't want to take up your time, unless you wish to ask some specific questions about them. Every one of the men has had a physical examination each year. That physical examination is a complete general physical examination, and in addition to the complete examination he has a special lung examination which is made to determine what has happened during the past year. We want to know whether he has had any cough, expectoration of blood, difficulty in breathing, and *that* we consider important. We ask him very carefully about whether he is short of breath, and we hop the man on one foot fifty times to see if he has any shortness of breath.

We take his weight, make a complete physical examination of the chest, including inspection to see whether he seems to be breathing lightly or deeply, precussion, auscultation to note breath, voice, and any rales present, as well as examining the condition of the heart and blood vessels. We have found in all of these cases practically everything is negative. Occasionally a man will report a cold; they have no dyspnoea; one man had apparent failing weight. They all seem to be in pretty good condition in this particular group. To sum up, I have published in the Journal of Industrial Hygiene three papers giving the result of our work. The conclusions of my first investigation are as follows: In factories which provide proven methods of dust removal, continuous inhalation of artificial abrasive dust over many years does not produce the symptoms or present in X-ray findings of pneumoconiosis. I think I should be inclined to change that now. This was my first paper. I think they do show some signs possibly of beginning pneumoconiosis, due to dust inhalation. I don't think they show any signs of disease of such an extent as to have any effect on the workman's ability to carry on his work. The number of cases of tuberculosis occurring in the artificial abrasive industry do not greatly exceed the number of cases in the community. Workers who habitually use grinding wheels will not develop pneumoconiosis if they use artificial abrasives rather than sandstone wheels. I considered sandstone wheels from the point of view of silica content, and from the point of view of the very much larger amount of dust. The second paper was simply a follow-up of the work done in the first paper, and I arrived at these conclusions. Continued inhalation of artificial abrasive dust composed of aluminum oxide and carbide of silicon will not produce disabling silicosis in the working period. The X-ray failed to reveal any case of third stage silicosis, and I used Dr. Pancoast's description of third stage silicosis. It is possible for men with arrested process in the lung to work in artificial abrasive dust for a considerable period without relighting the process. That is based on the fact that we found quite a number of cases in which the X-ray showed tuberculosis at the apex of the lung. I followed those cases with the greatest care. We

have examined them repeatedly. We have followed them at their work; we have not changed the work, and yet none of those cases have at the present time broken down with active tuberculosis. Our cases of tuberculosis have all been cases we didn't expect would show tuberculosis. Many were cases that came from parts of the factory where there was no dust hazard.

In regard to this question of tuberculosis, I made another study along this line in an effort to find out whether the inhalation of the artificial abrasive dust would or would not increase the amount of tuberculosis that there was among men who are exposed to it. I took the factory as a whole and I took the dust departments and analyzed them, and I found without question that there was more tuberculosis in the departments where there was artificial abrasive dust than there was proportionately in the factory as a whole. I am not sure whether that is due to the artificial abrasive dust or whether it is due to the fact that the men who work in these dusty departments are very apt on the whole to have lower living conditions and are more exposed to tuberculosis outside than are the men who work in other parts of the factory. For instance, in the departments where they make grinding machines, they require a very high type of mechanic. These workers live under very much better conditions than the men working in the dusty departments. It is a fact that there is a little more tuberculosis occurring over long periods of years among the workers who work in the dusty departments than among the workers who work in the non-dusty departments. That leads me to the conclusion that active pulmonary tuberculosis is a dangerous condition to introduce into any dusty place. We ought to prohibit men from working in a department where there is dust of any kind if they have active tuberculosis. Whether it makes a difference when they have healed tuberculosis I am not sure. We have enough cases here of healed tuberculosis, to make me feel there is less danger than one would anticipate. I can't put it any stronger than that. They may all break down the day after tomorrow; I don't know.

Just for a practical point, why do we keep these men with healed tuberculosis working in dusty atmospheres? I

think that can be well explained by a case we had a number of years ago. This man worked in one of our dusty departments and he broke down with active tuberculosis, a very definite tuberculosis, hemorrhage and so forth. We sent him to a sanatarium. He came back and the question was what to do with him. He was extremely skillful in his work and he said, "Doctor, I have got to work at that job. It is the only job I can do well; I have to work so that I can support my wife and children. If I try to do something else, I can't. I am willing to take the chance. I have been at the sanitarium and I know the first symptoms of a break down and I will stop then." So I gave him the chance and he worked for many, many years in that department without any difficulty or reactivation of that tuberculosis. Those are the situations we run into. It is a pretty serious thing to take a man's job away from him, unless you are absolutely sure it is going to hurt him or others in his department. For that reason I have preferred in these cases of apparently arrested tuberculosis to tell the man there was some danger, but to let him continue at his original occupation, follow him along, examine him frequently and keep him on the job for which he is best fitted.

I would like very much indeed to know more about the effect of the inhalation of artificial abrasive dust on the lungs of large numbers of people. I have a comparatively small group that I am working with. It is a group under excellent control. I am pretty sure of the facts here, but I haven't got that broad point of view which one gets in studying such a problem as silicosis pure and simple, where the literature is teeming with plenty of material. We need very much to know how much tuberculosis and how much pneumoconiosis there is among the people using artificial grinding wheels throughout this country. Probably that will gradually develop, but it is a subject that should be studied, watched and given a great deal of thought, particularly at the present time.

One further thing and then I will be through. I have here the mortality record. The morbidity record from tuberculosis which is most interesting is contained in this study of the dust hazard of the abrasive industry, and if

anyone wishes a copy of that, I will send it to them. The mortality record, however, has not been published. This mortality record goes back to 1920, twelve years ago. It contains the cause of death of every man who has died in the Norton Company during that period of time, and the cause of death is that registered in the City Hall of Worcester, so that we feel as far as we can tell it is correct. We didn't make the diagnosis. The diagnosis was made by hospitals and by the family physicians in every case. Now, let's see how that looks when we come to the question of different diseases. If we take the heart and vascular system which means heart disease, hemorrhages in the brain and so forth, I find that I have three and one-third pages. I am not going to give actual numbers, but I am trying to give you an impression. If I take malignant disease I find I have one and two-third pages; if I take tuberculosis I find I have one page, so that our death rate from tuberculosis is less than our death rate from cancer at the present time and way under our death rate from heart and vascular disease. I also find in looking over these deaths that a very large number of them never worked in contact with dust at all, but I will not go into those figures because they are of no particular interest. What I am trying to present to you is a clinical picture of the effect of dust on the workers of a factory where artificial abrasives are made, where there has been in the past very heavy exposure to dust, and where even at the present time the exposure to dust is probably higher than in well-equipped grinding rooms.

I should be very glad to answer any questions.

MR. DOE: Dr. Clark, in the studies that you have made have you had any opportunity to make any classification as between those exposed to carborundum and those exposed to aluminum oxide?

DR. CLARK: I have been unable to, because the men are shifted from one department to another. There are a number of transfers depending upon the activity of the various departments. The making of a carbide of silicon wheel is exactly the same as the making of an aluminum oxide wheel, so that if they happen to be busy in one

department and quiet in another, men are shifted from one department to another. The same applies in making the abrasives, so that the men are transferred from one department to another and it is impossible to separate them.

MR. DOE: Have you any opinion as to whether the tuberculosis incidence is higher where carborundum is used than where aluminum oxide is used?

DR. CLARK: I have no means of knowing that at all.

MR. DOE: You have no opinion?

DR. CLARK: No opinion, except what I know from outside; not from my own experience.

MR. DOE: Have you information, Dr. Clark, about what the dust counts are that these men are working in?

DR. CLARK: No, I will probably have some the day after tomorrow. We have been on the point of making dust counts. In 1929 we were all set to take dust counts. We had our plans made for the expert to come down to see what our dust count was. Then the depression came and practically half the machines became idle. The dust problem became a small proposition. We wanted a count under larger dosage. We have just taken counts around some machines to determine how dusty they were, but I feel quite sure in certain departments in the remote past there has been a very high dust count. I should say twenty years ago as high as fifty or sixty million. I think now, due to carefully planned dust control, it is very low.

MR. DOE: Can you give us any information about the incidence of pneumonia?

DR. CLARK: I can only give you the mortality of pneumonia. I can't give you the incidence of it. I should like very much, indeed, to keep a morbidity chart on all our respiratory diseases, but unfortunately the labor connected with doing that is considerable, and we are so cut down at the present time in endeavoring to keep our expense down that it has not yet been carried out. That is one of the studies I have been personally interested in making. Our mortality record from pneumonia is about the

same in the number of names that I see here as it is for malignant disease, one and two-thirds pages of names.

MR. DOE: Can you give us any information between the death rate from pneumonia and the death rate of the city of Worcester generally?

DR. CLARK: No, I can't for pneumonia. I can give you the incidence of tuberculosis in Worcester and I can give you the incidence of tuberculosis in our industry. The incidence of tuberculosis in the City of Worcester on a basis of 10,000, that is the basis we have gone on if we have that same population, is an average of twelve for the city of Worcester and thirteen for the Norton Company. On the other hand, we rather balanced that by the fact that at the Norton Company we know every case of tuberculosis we have, because it is pretty hard for a person with tuberculosis to escape if he is working for the company. In the city of Worcester all cases are not reported and there are a number of children who developed tuberculosis. Our average age is 36 at the Norton Company, so that we have older people than are included in the city of Worcester. So I think it probably balances pretty close.

MR. DOE: With reference to your data on deaths from heart and vascular conditions have you any opinion, Dr. Clark, as to whether any of those deaths have been secondary to the pneumoconiosis factor?

DR. CLARK: I think not. That would be my impression. I am just looking through, before I answer that question, to find out how many of these men have been exposed to dust. There are ten out of fifty-seven.

MR. DOE: Ten of the heart cases have been employed in the dusty departments?

DR. CLARK: Yes, out of 57.

MR. DOE: What is the proportion of employes in the dusty departments as compared to the total number of employes?

DR. CLARK: Roughly, about four. That is, there are about four times as many non-dusty as there are dusty.

MR. DOE: You say, Doctor, that in your annual re-examination of these men you make particular inquiry as to shortness of breath?

DR. CLARK: Yes.

MR. DOE: In your studies have any of these men that you have examined complained of shortness of breath?

DR. CLARK: No.

MR. DOE: You say that you also make particular inquiry as to colds and coughs, expectoration and so forth; what are the results as to that?

DR. CLARK: There are very few. There does not seem to be any abnormal amount.

MR. DOE: What do you find with regard to any other disease such as pleurisy or asthma or any of the other diseases of that tract?

DR. CLARK: I do not find it.

MR. DOE: So that you would say that the men you have been studying are from physical examination, apart from the X-ray, entirely negative?

DR. CLARK: Yes, that is as far as the lungs go. They may have hernias.

MR. DOE: Yes, that is what I mean. Does that apply to these men that have worked as high as forty years?

DR. CLARK: Yes.

MR. DOE: To all of them?

DR. CLARK: Yes.

MR. TARRELL: Dr. Clark, in these X-rays that you have exposed what type of pneumoconiosis do you see there?

DR. CLARK: I don't know; I don't pretend to be an expert on reading X-rays; I am trying to learn all the time about that.

MR. TARRELL: How many laborers do you have at the Norton Company plant?

DR. CLARK: Usually 2,500, now about half the number. At the present time there is no turnover.

MR. TARRELL: You make physical examinations of every employe?

DR. CLARK: Yes, and as frequently as necessary after that. Those exposed to dust are examined every year.

MR. TARRELL: What percentage of those given physical examinations are discharged or refused employment?

DR. CLARK: I should say none now. I think it was a fraction of one per cent in the past.

MR. TARRELL: A fraction of one per cent were refused employment?

DR. CLARK: Yes.

MR. TARRELL: What percentage of your employes were discharged or transferred to other employments after physical examinations?

DR. CLARK: Comparatively small number on first examination. We do most of our transfers after a man has been there for a certain period of time. When he shows some defect, he may develop a hernia or a murmur in the heart, and that makes it advisable to transfer him. My experience has been that most men apply for jobs to which they are physically fitted and the cases which we have been obliged to turn away have been a few cases of contagious disease, either active tuberculosis or some venereal disease, or more particularly men with only one eye. In our industry we don't think it is fair to have any man work in a department where there are flying abrasives if he has only one eye; there is always a danger. If he can do anything else we think it is very much better for him to do it. We will occasionally hire such a man for work in a non-dusty department.

MR. TARRELL: Do you know what percentage of employes working in exposure to abrasive dust are found to have defective lungs?

DR. CLARK: Well, we won't allow any man to work in artificial abrasive dust exposure if he has got any disease in his lungs that is active.

MR. TARRELL: You permit him to work if he has no noticeable disease of the respiratory tract?

DR. CLARK: Yes.

MR. TARRELL: If on examination you find a man is suffering from dyspnoea, what disposition do you make of his case?

DR. CLARK: That would have to be a case for study. We would want to find out what caused the dyspnoea, whether due to his heart or whether due to lungs, and why.

MR. TARRELL: With reference to Mr. Doe's question about the frequency of influenza—in an article that was written by you and published in the Journal of Industrial Hygiene, December 1931, you made mention of some cases in which you made the statement that "of those who developed active pulmonary tuberculosis in the departments where abrasive dust was prevalent, three developed symptoms in less than one year, six in less than four years, one in five years, and one in fifteen and one in twenty and one in twenty-one years. Four of these developed the disease immediately following an attack of influenza and after very short exposure to dust." That is a correct statement of what you found?

DR. CLARK: Yes, that is right.

MR. TARRELL: And those working in clay dust, one developed the disease after twelve years of service?

DR. CLARK: Yes. The clay dust contains nine per cent free silica.

MR. TARRELL: You made the further statement among those working in the abrasive dusts, in proportion to the number of workers involved there were approximately twice as many cases of pulmonary tuberculosis as among those in departments where no abrasive dust occurred?

DR. CLARK: Yes, before I made the analysis, which I think is a fair analysis, which reduces it to one and one-

half if you cut those cases of influenza out. I do believe as I said before that the inhalation of dust, I don't care whether it is artificial abrasive dust or whether a dust of high silica content, or perhaps even tobacco dust or cotton dust, I think any of those dusts are to a certain degree, to a certain number of people provocative of lighting up a quiescent tuberculosis.

MR. TARRELL: In other words, you think they cause an irritation?

DR. CLARK: They cause an irritation, but I also feel that the cases that we have had should be considered. We have at the present time a certain number of men who are working in dusty departments with X-ray tuberculosis. The X-ray shows that the man has tuberculosis which hasn't broken down, and I don't feel that it is fair to discharge such cases on the chance that they may break down. If they do it is up to us to take care of them.

MR. TARRELL: Where you have an irritated respiratory tract and an irritated lung it is pure speculation to say how soon it may break down?

DR. CLARK: Yes.

MR. TARRELL: In this same article, in your conclusion you said that about the only conclusion which can be drawn from the present study is that it is "inadvisable for persons who have had pulmonary tuberculosis to work in a department in which large amounts of artificial abrasive or any other dust are present."

DR. CLARK: That is my—

MR. TARRELL: Do you find any reason to change your conclusion in that respect?

DR. CLARK: No. I don't.

MR. WRIGHT: Heavy work or light work?

DR. CLARK: Pretty heavy work. They have to lift heavy wheels; sometimes one hundred and fifty pounds. It depends entirely upon what part of the factory they are working in.

DR. BELLIS: *What protection is given your recommendation against inhalation of dust?*

DR. CLARK: The use of hoods on all machines and very powerful suction which draws the dust away from the operator at the point at which dust is formed. It is an interesting thing about very fine dust in artificial abrasive wheels. When the wheel is traveling at great speed, which they do, there is a tendency for the dust to cling to the wheel. Large pieces of it throw off, but the fine dust seems to cling to the wheel, and this with the larger of the fine particles are very adequately taken care of by suction apparatus and the hood which protects two-thirds of the wheel.

MR. DOE: Do you feel within your work you have discovered any scientific evidence that aluminum oxide abrasives pre-dispose to tuberculosis?

DR. CLARK: No.

MR. DOE: Well, on that hypothesis, what scientific basis is there for saying that any dust predisposes to tuberculosis?

DR. CLARK: I don't think any doctor would advise a patient who had tuberculosis to work in a dusty atmosphere. I think that is just the general medical feeling. If we know a person has tuberculosis we try to put him in a place where the air is clean. In the case of determining the type of work for a person who has had tuberculosis, it is then a question whether he is on the safe side or danger side. I feel it would depend on the individual case. There are some cases in which I feel I would be perfectly willing to put a man back to work who had had tuberculosis, and I have done so in dusty departments where I felt I could control the man enough so if he did break down I could get him up again.

MR. DOE: I don't mean to assume a case, doctor, where a man either was in a state of having active tuberculosis or of having had tuberculosis. I mean a person who at the time he starts to work is absolutely negative for tuberculosis at that time. What scientific basis is there for saying that any dust predisposes that individual to tuberculosis?

DR. CLARK: I don't think there is any, except that there is undoubtedly some irritation in the bronchus and there is a possibility that if he is exposed to a heavy dose of tubercle bacilli he will get tuberculosis, but I would prefer to have that discussed by Dr. Gardner. What I was trying to discuss in this paper was whether tuberculosis was activated by dust. My final conclusion was that the people who had tuberculosis had better not work in a dusty atmosphere. That had nothing to do with a person who has no tuberculosis.

MR. DOE: Was your answer to Mr. Tarrell a few minutes ago to that same effect—that persons who had had tuberculosis any dusty atmosphere was contraindicated?

DR. CLARK: Yes.

MR. DOE: You didn't mean to express an opinion in regard to those who did not have tuberculosis?

DR. CLARK: No.

MR. DOE: In your cases where you have found that there were certain developments of tuberculosis in the dusty departments, were those persons who had not been previously detected as being healed cases?

DR. CLARK: Yes. We do not take an X-ray of all our employes. That is one of the developments I hope to get to. I hope to have an X-ray on every man working in a dusty department. We are on the way, but it hasn't been done. We have to take on a great deal of work in the Norton Company medical department which has nothing whatever to do with the question of dust inhalation in the lungs. We are confined to a certain staff and we have to cover a large amount of preventative work, diagnose many cases, and treat all minor diseases as well, so that the amount of time we can spend on any particular problem is not the same as in a university where one has unlimited time and funds.

MR. DOE: In those cases that became active who were employed in dust, you are unable to say whether they were tuberculous before entering the employment?

DR. CLARK: We can only say they did not show signs of active tuberculosis to the stethoscope.

MR. DOE: You perhaps hadn't reached any conclusion on the question of whether a person who showed no past tuberculosis would be predisposed by inhalation of aluminum oxide and carborundum?

DR. CLARK: No.

MR. DOE: You have no opinion on that?

DR. CLARK: I have no scientific opinion on that at all.

MR. NELSON: Do your figures include men who have left the employ of the company and been gone for some time?

DR. CLARK: So far as we know it does for a certain number. If a man leaves the company for some time and develops any serious condition, it almost always gets back to the company some way. Through other men or the man himself, or through the insurance company.

MR. NELSON: Do you make a systematic check up?

DR. CLARK: No. Not on the men who have left the employ of the company. We don't know where they are.

MR. DOE: I understand that in no single case where you discovered the tuberculosis by X-ray has there been a breakdown afterwards?

DR. CLARK: I have had none so far.

DR. KUHN: Do your employes in your company have physical examinations pertaining to the chest? Do they have X-ray examinations prior to employment?

DR. CLARK: No. They only have X-rays of the chest if they have been working in a dusty department for ten years.

DR. KUHN: How do you find it possible to diagnose fibroid tuberculosis after they have been in the employment for say a period of three months or six months and you X-ray the chest and find this condition if you have no prior check up as they come in from an X-ray examination?

DR. CLARK: I don't quite get what you want.

DR. KUHN: The point I am trying to make is, if you haven't X-ray examination prior to employment and then you have later an X-ray examination after they are employed three months—

DR. CLARK: But we don't examine them by X-ray until they have been employed for ten years.

A VOICE: If they have lung pathology, you do X-ray them?

DR. CLARK: Yes. If a man shows any lung pathology that we can find, and they are coming in all the time, any man who has a cough which lasts for more than a week, we give him a very careful examination and sputum examination and if there is any question of definite disease, we send him for X-ray.

DR. GARDNER: I shall attempt to summarize and amplify my remarks of this morning in a series of lantern slides. I shall demonstrate the characteristics of the reaction to different types of dust in the normal and in the tuberculous animal.

(There followed a series of slides in which various features were pointed out. Since the stenographic report of this demonstration is pointless without illustrations, I shall tabulate and summarize the features which were brought out by these slides.)

1. An early silicotic nodule—consisting of a zone of dust-filled phagocytes about a lymphoid nodule in the periphery of the lung.

2. Longitudinal section of an artery in the lung with lymphatic trunks running through its wall. Encroaching upon the channel of the lymph vessel but outside its membrane of lining cells are masses of lymphoid tissue. These masses contain small collections of dust cells.

3. Further development of the silicotic nodules in the areas shown in slide 2. Note that the channel of the lmyph vessel is greatly narrowed by the expanding nodule. This is one of the causes of lymph stasis.

4. Extensive silicosis in a tracheobronchial lymph node obliterating most of the lymph channels—a further cause

of interference with the normal flow of lymph. When the reactions illustrated in slides 3 and 4 have occurred the dust can no longer be removed and it accumulates everywhere in the walls of the pulmonary air spaces.

5. Diffuse and nodular fibrosis in pulmonary air space walls—producing so much distortion that the external characteristics of the organ are obliterated. No interchange of oxygen and carbon dioxide could take place through walls thickened in this manner.

6. Nodular silicosis in rabbit's lung produced by inhalation of pure crystalline silica. Nodular lesions are characteristic of the disease in both human beings and animals. The reproduction of such reactions demonstrates the adequacy of the experimental method for studying reactions to any dust. The nodules show degenerative changes at their centres due to the toxic action of the silica and not to a lack of nutrition. This is demonstrated by the presence of a blood vessel traversing the degenerated portion. The margins of the nodule, formed by cellular fibrous tissue, are sharply defined.

7. Cross section of a pair of lungs from a guinea pig exposed to crystalline silica for 14 months presents nodular lesions throughout both lungs with particularly large numbers beneath the pleura. The tracheobronchial lymph nodes are enlarged and replaced by reaction to the silica.

8. An X-ray film of the animal whose lungs were shown in slide 7. Note the similarity to similar films of human beings with silicosis.

9. Another X-ray film of a guinea pig exposed for 1 year and 10 months. The density and number of the nodular shadows is greater.

10. Lung of a rabbit exposed to inhalation of crystalline silica for 13 months. Minute nodules resembling tubercles scattered throughout the lung.

11. Higher magnification of the individual nodules shown in slide 10. Demonstrates the formation of silicotic nodules in lymphoid tissues associated with blood vessels and bronchi. Note that the nodules have sharply defined borders and that very few dust cells are seen in the air spaces of the lung outside the nodules.

12. Lung of a rabbit exposed for 13 months as in the case of the animal exhibited in slide 10 but then removed from the dust room and allowed to live in a normal atmosphere for another 8 months. Its lung shows nodules larger than those seen in slide 10; in other words without further exposure to dust the tissue reaction has progressed.

13. Higher magnification of two of the nodules illustrated in slide 12. Note that the margins of the nodules are no longer sharply defined but that they are surrounded by a large number of dust cells. Some of them may represent additional cells which have migrated to the focus after the dust exposure was discontinued. But sections of the first animal showed that most of the inhaled dust had already been collected within the nodules. Therefore it is assumed that most of the cells about the borders of the nodules are attracted by silica particles which other migrating cells have carried out of the nodule and deposited on the periphery.

Demonstration of such progression of the disease has also been possible by the use of serial roentgenograms not only in the experimental animal but in human beings. The progressive nature of silicosis is one of its most discouraging aspects. It is our hope that research will disclose some method either of eliminating or of fixing and rendering harmless the silica which has been inhaled.

14. Three graded samples of very fine silica particles sizes 12 to 9 microns, 8 to 6 microns, and 3 to 1 microns in diameter, respectively. These separations were made by a method which Mr. Cummings has devised depending upon variation of the settling rate of various sized particles in alcohol.

15. Reaction in lung to 1.3 grams of the largest particles (9 to 12 microns) which were injected intravenously in divided doses over a period of one month. Animal killed after completing last injection. Note that the particles remain in the walls of the air spaces where they have been surrounded by giant phagocytes. Section stained specifically to bring out the fibrous elements of connective tissue. The giant phagocytes are surrounded by only a few thin fibres.

16. Another animal treated similarly to that shown in slide 15 but not killed until one year after completing the injection of the large particles. Note that there has been little change in the character of the reaction. The nodule of phagocytes is only slightly larger and the number and thickness of the connective tissue fibres is only slightly increased over those seen in the previous slide.

By comparing slides 15 and 16 we conclude that particles as large as 9 to 12 microns in diameter are relatively inert. They do not produce the progressive type of reaction seen with smaller silica particles. They do not excite the specific effect of silica but are combated with a cellular response similar to that excited by any insoluble foreign body.

17. A silicotic nodule produced by inhalation of very fine particles—magnification the same as that used in figures 15 and 16—to show how much larger the area of reaction and how much greater the amount of black stained fibrous tissue. The fine particles produce the typical silica effect.

18. Lymph node draining the liver from an animal injected intravenously with particles 6 to 8 microns in diameter. These particles are so small that most of them are carried through the capillaries of the lung to the left heart and from thence to the liver. From this organ its lymph vessels transport them to a lymph node near the gall bladder. Concentration in this location results in reaction. The node is some 20 times its normal size. It contains a great number of cells filled with dust and resembles the reaction to the tubercle bacillus. But there is no tuberculosis in this case; the reaction is due to silica particles which can be demonstrated by a special form of illumination known as polarized light.

19. Another portion of the same lymph node shown in slide 18. Here one sees an early silicotic nodule which is composed of cellular connective tissue which is beginning to show the specific hyaline degeneration in its central portion. Polarized light reveals that the very finest silica particles are in the nodular part of the lymph node; in the area of diffuse reaction seen in slide 18 the particles are larger.

The body has separated the particles varying from 6 to 8 microns in diameter into fractions. The smaller ones being more active excite the nodular reaction characteristic of advanced silicosis; the larger ones the diffuse response of the early stages of the picture.

• 20, 21, 22, 23. Sections of livers of animals injected by vein with 1.3 gram of the very finest silica particles, 1 to 3 microns in diameter.

These particles also passed through the lung and were carried to the liver and thence to the lymph node which drains this organ. There, because of their very small size, they rapidly excited the formation of scar tissue which interferred with lymphatic drainage from the liver. The later injections of the dust could not be removed from the liver but stayed in this location. There they excited a reaction which is essentially the same as silicosis of the lung. It started as a nodule of dust cells in the connective tissues (illustrated). This nodule was gradually transformed into typical hyaline fibrous tissue, a typical silicotic nodule (illustrated). The silica gradually killed off the liver cells and most of them were replaced by scar tissue until there were only small islands of such cells (illustrated) or group of the liver bile ducts (illustrated) to be seen. Any extensive formation of scar tissue in the liver is called a cirrhosis. This experimental lesion would be called a silicotic cirrhosis. The whole organ is involved so that in gross it resembles a white nodular mass of scar with little resemblance to normal liver tissue.

By killing rabbits at successive intervals after discontinuing the injections of dust it was shown that the reaction progressed from month to month, but in each case it was made up of a series of silicotic nodules and a diffuse formation of silicotic fibrous tissue.

The experiments illustrated in slides 15 to 23 offer suggestive evidence to confirm the solubility hypothesis. They show that the rate of reaction to silica dust varies directly with the size of the particle. The smaller the particle the larger is its surface area and the greater its capacity to excite reaction. Such relationships are characteristic of chemical rather than mechanical irritation.

These observations suggest the importance of determin-

ing whether the extremely fine and therefore most potent particles are not only inhaled but are retained in the lungs. Analyses of the ash of silicotic lungs show that the majority of particles vary from 1 to 3 microns in diameter. Whether our methods are adequate to detect and measure the smaller ones, I do not know nor have we any evidence to show whether the cilia may eliminate particles less than 1 micron in diameter more rapidly than those of the 1 to 3 micron size group. If it should be proven that the extremely active particles under a micron in diameter are retained within the lung our apparatus for dust counting must be modified. Mr. Bloomfield and Mr. Cummings will have more to say on this subject.

24. Lt. Lung of a normal guinea pig infected by inhalation of our attenuated tubercle bacillus. The reaction corresponds to the childhood type of tuberculous infection in human beings. It consists of a localized tubercle or nodule beneath the pleura and more extensive tuberculosis in the tracheobronchial lymph node. The latter is produced by drainage of bacilli from the lung tubercle through the lymphatic vessels. In normal guinea pigs such tubercles tend to heal of their own accord and ultimately disappear.

24. Rt. Lung of a guinea pig infected with the same attenuated tubercle bacillus after one year's exposure to silica dust. The area of disease in the lung and in the tracheobronchial lymph node is now much more extensive. It is a new type of disease, an acute silico-tuberculosis. It does not heal and disappear but progresses and ultimately kills the animal. (In this case within two months after the infection).

25. Inhalation of silica dust begun after an infection with the attenuated tubercle bacillus has begun to heal. The silica reactivated the healing tubercles and caused them to become progressive again. The infection has spread extensively throughout the lung and numerous cavities have formed where the original tubercles were located. The amount of disease in the tracheobronchial lymph nodes is excessive and has spread beyond their limits into the connective tissue about them (the mediastinum).

26. Chronic silico-tuberculosis in a guinea pig produced by the inhalation of silica dust during the period of develop-

ment of tuberculous infection with the attenuated tubercle bacillus.

The desease is widespread, and slowly progressive. I use the compound term silico-tuberculosis to indicate the combined lesion but not in the sense the South Africans employ silico-tuberculosis or tuberculo-silicosis to emphasize priority of one or the other irritant.

27. Exposure to asbestos dust, 8 hours daily for 840 days. Instead of a nodular reaction this dust excites the formation of scar tissue in the form of elongated collars which surround the terminal bronchioles. Migratory phagocytes and the lymphatic system play little part in the localization of this dust probably because of the fibrous character of its particles. The fibres come to rest along the walls of the smaller bronchioles and are carried directly into the substance of the nearby walls. Practically none of the dust reaches the tracheobronchial nodes and the section shows no significant reaction.

28 and 29. A higher magnification of terminal bronchioles in early asbestosis. Note the cellular character of the fibrous tissue with none of the hyaline material characteristic of silicosis. Contraction of the collar like cylinder of fibrous tissue would collapse the bronchiole and prevent air from entering the air spaces which it supplies. Such collapse is followed by a diffuse fibrosis of the area involved.

I have recently acquired a specimen from the lung of the case of asbestosis reported by Dr. W. B. Soper in the American Review of Tuberculosis (1930). This man was exposed to asbestos dust for 13 years and died 4 years later. The lung exhibited a very extensive and more or less diffuse fibrosis which in most places bore no relationship to the bronchioles. In fact the normal anatomical structures were hardly recognizable. Material from such far advanced cases is hardly suitable for analysis. Judging from the evolution of the process observed in guinea pigs it has been assumed that much of the diffuse fibrosis may be associated with the collapse of air spaces following compression of the bronchioles by the primary reaction about their walls.

30. A group of asbestosis bodies—These peculiar structures are golden yellow in color with swollen ends and irregular nodules along their sides. They probably develop,

Mr. Cummings believes, as the result of solution of silica from the fibres and a redeposition of this substance on the same fibre. They also contain iron which is responsible for their color.

31. Spleen of rabbit injected intravenously with 1.3 grams of aluminum oxide particles, 1 to 3 microns in diameter, and killed three months after the last injection. The particles are collected in large phagocytes which have to rest in the normal tissue and provoked no reaction.

32. Liver of a rabbit injected with aluminum oxide particles as in slide 31. This animal was not killed for two years after the injection. Again one sees the same large phagocytes packed with dust particles reposing in lymph spaces of the normal connective tissue of the organ. There is no fibrosis even after 2 years. Except for the miscroscopic collections of dust this liver is a normal organ.

33, 34, 35. Livers of animals injected with the same amount of the same sized quartz particles. Phagocytes filled with this dust localize in the same locations but the cells are grouped together and there are more of them. They form a nodule, it becomes fibrous and undergoes the characteristic hyaline change. The liver cells are killed and the whole organ is replaced by fibrous tissue. (Compare slides 20 to 23).

Mr. Cummings performed this experiment to show that silica exerted a specific poisonous action on the tissue. He selected aluminum oxide as a control material because it is an extremely hard substance which fractures into particles similar to those of quartz. The outcome indicates the correctness of his supposition. These negative findings with aluminum oxide are of particular interest in view of the discussion of this subject during the earlier parts of this meeting.

36. Cross section of the lungs and tracheobronchial lymph nodes of a guinea pig exposed to the inhalation of carborundum dust for 4 years. A great deal of dust has settled in the air spaces of this lung, particularly those beneath the pleura. In the tracheobronchial lymph nodes the amount of dust is relatively small when compared to that seen in an animal inhaling quartz.

37. A higher magnification of the subpleural air spaces from the same animal shown in slide 36. There are large masses of phagocytes packed with dust particles which nearly fill the interior of many of the air spaces. The adjacent walls of many of them are thickened but there is no massive or nodular fibrosis. Undoubtedly there may be loss of function in such places but it will be noted that many other air spaces in the immediate vicinity have apparently normal walls which would allow free interchange of oxygen and carbon dioxide between their interior and the blood capillaries. Very little dust is carried to the tracheobronchial lymph nodes and in consequence they show only the slightest amount of reaction. No appreciable abnormalities in the lymphatic vessels are discoverable. A relatively small amount of dust is carried into the lymphatic vessels and there is some evidence of reaction in the loose tissue about them. This may easily account for the thickening of the blood vessels and bronchi seen in Dr. Clark's X-ray films of silicon carbide workers. The diffuse haze which he mentioned is probably due to the obstruction with the rays by the slightly thickened air space walls and possibly by the great amount of dust within the air spaces.

Carborundum dust in combination with tuberculous infection has offered difficulties which are not easy to explain in the light of our present theories. This substance is the carbide of silicon and is reported to contain no free silica, the substance which theoretically activates a tuberculous infection. The manufacturers analyses have not been checked in our laboratory because of the lack of proper apparatus. Mention has already been made of the possibility that the weakly alkaline fluids of the body may attack this substance and ultimately break it down.

In the experiments already referred to where a partially healed tuberculous infection was subsequently reactivated by exposures to dust we found that next to quartz dust, carborundum was the most potent irritant yet employed. More infections were made progressive with this dust than with granite. Whether the carbide of silicon has the same effect in human beings has not been determined for lack of post-mortem material. A large number of autopsy studies

on persons dying after long exposures to this dust are urgently required.

In reciting our experience with this dust mention was accidentally omitted of several other experiments in each of which carborundum was found to be almost as potent as quartz in its effect upon tuberculous infection.

38. Section of the lung of a soft coal miner,—note that there are deposits of this dust along the pleura and along the bronchi and blood vessels. These are the positions in which the superficial and deep sets of lymphatics respectively lie. Apparently the phagocytes ingesting coal make their way into the lymphatics but many of them fail to reach their destination in the lymphoid tissues of the lungs and the tracheobronchial lymph nodes. Instead they appear to leave the lymph vessel and are deposited in the loose connective tissue through which these fine vessels course. There they cause pigmentation and perhaps a little overgrowth of the connective tissues, but pure coal dust excites no true fibrosis. In an X-ray film the trunk shadows would be accentuated as in the case of carborundum dust.

39. A cross section of a blood vessel from another coal miner's lung, surrounded by a ring of densely pigmented true scar tissue. This scar exhibits modified characteristics of changes seen in silicosis. Silica particles cannot be detected but if the carbon is removed by incineration of the section in a muffle furnace great numbers of doubly refractile particles which I think are silica are visualized. We are now having some petrographic determinations made to satisfy ourselves as to the nature of these particles.

The hard coal miner is apt to work in rock containing high percentages of silica. If this substance is in excess the individual develops a *nodular* silicosis accompanied by an intense pigmentation of the nodules by coal dust. If the reverse is true and only a small amount of silica is mixed with a large amount of coal the reaction is characteristically *linear*, along the course of the lymphatics but enough silica may be present to produce some hyaline change in the connective tissue fibres. It is only by chemical and microscopic analysis that we can determine how much silica is involved in any case of anthracosis. It would appear that pure coal

with no admixture of silica produces no fibrous changes in the lung.

In giving the testimony no mention was made of "miner's asthma". This is a disabling condition seen in coal miners probably due to a pathological change known as emphysema, a dilatation of the terminal air spaces. Frequently the lungs show collections of air spaces which are dilated to such a size that they admit the head of a pin or even a pea. These spaces have exceedingly thin, smooth, shiny walls. Frequently a deposit of coal dust occurs on one side of such a space. Why coal and not other dusts produces this change is unknown.

The inhalation of soft coal dust is said to protect against the development of tuberculosis and statistical evidence would seem to favor a lower incidence of this infection among certain groups of soft coal miners. However, this is not universal. Wainwright and Nichols reported some experiments in which they thought they had demonstrated a protective action of coal dust against tuberculosis, but they have never been verified. When guinea pigs are infected with the same attenuated tubercle bacillus already mentioned and then exposed to coal dust there is no influence on the tuberculosis. It heals neither faster nor more slowly than in the undusted control animals. Moreover coal has no influence in checking the progress of infection with fully virulent infections. We are not yet convinced that this substance might not prevent progress of an infection which is not too severe. It is hoped that we may do further work with coal dust and an infection of moderate virulence.

I think this covers as briefly as I know how, the type of work in which we have been engaged. It illustrates to you that there are definite differences in the reaction to different types of dust. It illustrates that silica dust is unique in injuring the body tissues; that it produces a progressive type of disease and that it renders the body more susceptible to the tubercle bacilli. Someone asked this morning why tubercle bacilli would grow more readily in the silicotic lung than in the normal lung. This is the subject which is, of course, tremendously interesting. We do not know today why it happens but I suspect that it does because the silica poisons the body cells, the soil, in such a way as to favor

the growth of tubercle bacilli. What we hope to do is to neutralize this poisoning effect of silica and thereby prevent the development of susceptibility to the tubercle bacillus. If we could do this we would have accomplished a great deal, because probably 75% of silicotics will die of tuberculous infection. If they escape infection or if they are no more susceptible than normal individuals, they would presumably carry on for a period of ten, fifteen or more years. They would not be perfect individuals, but they might be able to support themselves and their families for a much longer time than they do at the present. If we can neutralize this toxic effect of silica, we will be accomplishing something and this is the goal to which Mr. Cummings and I have set ourselves.

If there are questions, I shall be glad to try to answer them.

MR. CUMMINGS: Dr. Gardner has discussed the normal pulmonary anatomy and the pathological reaction of the lung to the various inhaled dusts, particularly silica. I think it would probably be worth while to amplify the chemical and physical properties which certain dusts possess in order to evaluate their ability to provoke pathological changes in the lungs of man.

It was proposed in the protocol to discuss dusts of both organic and inorganic composition. Organic dusts, such as cotton, undoubtedly do give rise to certain pathological conditions in the lung or bronchi. But we have not investigated these dusts and I therefore beg your permission to pass them over for the present. The inorganic dusts, particularly ore dusts, are the ones with which we are vitally concerned at the present time. These dusts I should prefer to classify first into readily soluble, and insoluble dusts. By readily soluble dusts, I mean dusts which will dissolve fairly readily in the lung tissue or juices. The degree of harmfulness of these soluble dusts is in direct proportion to the toxicity of the soluble products formed. For example, we know that lime stone (or marble) which is a soluble dust, does dissolve in the lung juices, and soluble calcium salts, which are more or less similar to substances that are already present in the lung, are produced by this solution and consequently there is no evidence of toxicity on the part

of this material. On the other hand, there are the toxic soluble dusts, some of which will dissolve only slightly but which will nevertheless produce definitely toxic substances. Freshly generated zinc oxide, for instance, is quite toxic. Lead dust is toxic and we can attribute its toxicity to its solubility.

Some years ago, Collis, and numerous other English investigators, reported that silica dust could be regarded as dangerous because it was insoluble and that in general the degree of insolubility of any dust would also determine the ability of that dust to produce pathological changes. Dr. Gardner and I do not adhere to this theory. We believe that because a dust is insoluble or inert it does not follow that it is capable of producing a reaction in the lung. Dr. Gardner has mentioned a few dusts that we believe do not produce marked reactions. Aluminum oxide in the form of corundum, which is certainly as insoluble as any of the dusts, fails to produce a significant reaction. Silica cannot, therefore, be regarded as harmful simply because it is insoluble. I would like to emphasize that there are, in addition to the dusts that Dr. Gardner has mentioned, certain harmful and harmless inorganic and more or less insoluble dusts; for instance, carbon in the form of soot or smoke, or in the form of graphite or diamond, is insoluble and also relatively harmless in producing the typical fibrous reaction that we associate with silicosis. Coal, which is a combination of carbon and certain organic substances, is also relatively harmless—particularly soft coal. Dr. Gardner has emphasized that, at the present time at least, we associate the harmful influence that coal may exert on the lung with the silica which is probably inhaled along with it. This is particularly true of hard coal miners who find it necessary to dig shafts through hard rock. Carborundum dust is not particularly dangerous though it is insoluble. Carborundum is an unusual dust from this standpoint. It is silicon carbide and contains practically no silica. It is possible that it is slightly soluble in the alkaline fluids of the body but it apparently fails to produce reaction per se. Even though the inhalation of carborundum dust fails to promote a marked reaction itself, there may still be enough of this particular soluble substance formed to ex-

plain the experimental evidence that we have for its ability to reactivate latent tuberculosis. Cement dust is another material which contains some soluble and some insoluble elements and which nevertheless is not particularly harmful. On the other hand we have silica which so far as we know is relatively insoluble and yet is the most harmful of all dusts. We cannot therefore associate the degree of harmfulness of any dust with its solubility or insolubility, but we must regard silica as a specifically dangerous compound. We must also consider the various silicates which represent combinations of silica with other elements and also the mixtures of silica and silicates or mixtures of silicates and other substances.

I propose to discuss silica and these silicates and mixtures with respect to their physical and chemical properties. At the outset we must recognize the different forms which silica may assume in a given dust. It may occur as free silica, combined silica or mixtures of both. When an analysis is made of a certain dust which is regarded as dangerous, we estimate the total amount of silica present and in addition we seek to obtain a figure which represents the amount of silica which is not combined with other elements. This is the fraction spoken of as free silica and is universally regarded as a direct measure of the silicosis-producing power of any dust. There are various methods for determining this fraction. One is the petrographic method, which is an approximation arrived at by simply examining fine dusts under a special microscope and estimating the relative numbers of each type of crystal that is recognized. There is also a chemical method for determining free silica of which I shall say more later.

There are a great many physical modifications of silica which differ from the typical quartz dusts which are generally used for experimental work or that are more often referred to in connection with silicosis. It would be well to mention these various physical modifications since it is my belief that their reaction in the lung may be quite different. Some are undoubtedly more harmful than others and it may even be possible that certain varieties of silica are relatively harmless. It is my belief that a thorough study of the reactions produced by each of these modifications will

provide the key to a scientific understanding of the true mechanism of the development of silicosis.

There are in general three principal physical forms of silica occurring in nature. This classification is based on physical structure and indicates the differences in the molecular arrangement of crystals, the differentiation of crystalline and non-crystalline or amorphous forms, and also the degree of hydration of the water-containing varieties. This classification is as follows:

Principal Phases	*Micro Forms*	*Hydrated Varieties*
Quartz	Chalcedonic silica	Opaline Silica
Tridymite	or micro fibrous	Chalcedonic Silica
Cristobalite	Micro Amorphous	
Vitreous Silica		

Quartz is the stable crystalline form of silica from ordinary atmospheric temperature to 870° C.; tridymite is stable from 870° C. to 1470° C.; cristobalite is a stable crystalline form of silica from 1470° C. to 1710° C. However, all of these forms may exist at ordinary temperatures. All are hard, sharp, definitely crystalline forms of silica and constitute the greatest bulk of the silica found on the earth's surface. Chalcedonic silica is a micro fibrous form having measurable size in only one dimension. Carnelian, agates, flints, cherts, are the best known representatives of this form of silica and are also widely distributed over the earth's surface. The micro amorphous varieties of silica are particularly interesting since they occur principally in deposits which represent the remains of certain sea plants called diatoms. This modification of silica is more readily soluble than the crystalline varieties mentioned above. Similarly, the forms known as opaline silica represent silica in combination with more or less water and this group of substances is best represented by opal itself.

It is apparent that silica may assume a very extensive variety of physical forms and that each of these modifications may produce a very characteristic reaction is quite conceivable. It is therefore essential to understand the exact physical nature of any silica-containing dust in evaluating its ability to produce silicosis.

The term silica then does not define the properties or

harmfulness of a dust for the reaction produced by it may depend upon the physical state of the silica quite as much as upon the concentration, for example. The two methods for determining this quantity of free silica are, as I have mentioned, the petrographic and chemical method. The chemical method is as yet totally inadequate for the determination of all forms of free silica since it was designed to determine only the free silica existing as quartz, or one of the quartz-like materials. If the free silica in a certain dust should be in the amorphous form, that silica would not be determined as such in the analysis, and a large error would be made. The method, therefore, needs revision or at least its inadequacies need better recognition.

In addition to the free silica, dust may contain silica combined with other elements as it is in asbestos, slate, or other silicates, and here again it presents still a different structure. Where it is combined with other elements many of these combinations may be more readily soluble than is quartz, but in spite of this fact the silicate has yet to be found which is as dangerous as quartz. This either constitutes strong evidence against the theory of a soluble poison originating from silica by solution, or it is excellent proof of the contention that certain other substances, when intimately associated with silica tend to overcome its dangerous properties. The pathological changes resulting from the inhalation of a few of the silicates are now known. Dr. Gardner and I have studied the reaction produced by asbestos in experimental animals and we are convinced that asbestos is slowly soluble since the inhaled fibers give rise to a peculiar structure known as the asbestosis body. A pulmonary fibrosis does result in asbestos workers but here again the reaction is quite different from a true silicosis and emphasizes the necessity of knowing the properties of the dust inhaled. It is hoped that the study of several silicates will enable us to distinguish whether the reaction is due to the peculiar surface phenomena presented by these combinations of silica or whether the reaction is always due to soluble products from the silica—masked in the case of silicates and mixtures by the presence of another element with which these soluble products may combine and thereby be rendered inert. In any event, silicates do constitute a class of

struction that we can visualize the relationship of these spaces to one another. By this method it becomes obvious that the terminal bronchioles become narrower and that their walls become thinner. They lose their definite lining of ciliated epithelium and break up into elongated tubes with scalloped walls. These tubes ultimately communicate with a series of extremely thin walled terminal air sacs, or alveoli. On examining these structures under high magnification it can be seen that they consist of a space surrounded by an extremely thin wall composed of elastic tissue which has the power of expanding and contracting. The inner surface of this wall is covered by a thin layer of flat pavement-like cells. In the substance of the wall there is a network of capillary blood vessels. These structures are also lined by a layer of flat pavement cells. Here and there in the wall there are larger cells which project inward between the flat pavement cells. These larger elements are the dust cells of which we shall speak more later. They are shed off from time to time and take on free migratory existence, having the power of independent movement.

The purpose of this more or less complicated mechanism is to permit air containing a relatively large amount of oxygen to come into intimate association with the blood and to permit an interchange of gases between the blood in the vessels and the air inside the spaces of the lung. Blood brought into this organism through the pulmonary arteries contains considerable amounts of carbon dioxide which is dissolved in its fluid portion. This carbon dioxide is generated in all parts of the body where any activity has taken place. It is a waste product which must be eliminated. Leaving the pulmonary capillary, the carbon dioxide diffuses out through the walls of the terminal air spaces by passing through the cells lining the capillaries and the second layer of pavement cells lining the air spaces. From this location it can be eliminated with the expired air. There is also a flow of gas in the opposite direction. The inspired air contains oxygen. This passes inward from the air space to the blood; the interchange again taking place through the same two layers of cells. The coloring matter in the red blood corpuscles possesses a marked affinity for oxygen. A loose combination is formed and the red blood corpuscles

then carry this gas back through the pulmonary vein to the left heart by which they are pumped out through the general circulation into the organs of the body. Wherever work is being done oxygen is necessary. The red blood corpuscles give up their oxygen to various kinds of cells as they function. This whole process is known as respiration.

Obviously, to be performed efficiently, the membranes in the lung which separate air spaces and blood vessels must be extremely thin and readily permeable to interchange of gases. Any disease process which thickens this membrane and prevents such interchange would reduce the efficiency of the mechanism. As we shall see, the development of fibrous tissue in response to irritation frequently thickens these delicate membranes. If enough of them are involved life can no longer continue, but fortunately only portions of the lung may be so affected until irritation has been continued for a very long time.

Lymph Nodes. At the root of the lung are located the tracheobronchial lymph nodes. They are divided into three main groups. One of them lies below the angle between the two bronchi; another group lies above the left bronchus in the angle which it forms with the trachea; and a third lies in the corresponding angle made by the right bronchus and trachea. These lymph nodes constitute a drainage reservoir to receive foreign materials which may have penetrated into the lung. Such material comes to the lymph nodes through a system of lymphatic vessels.

Lymphatic System. Mention has already been made of the tracheobronchial lymph nodes, located at the root of the lung which receive the drainage from this organ. We have still to consider the vessels which carry foreign matter out of the lung to the lymph nodes. These vessels are exceedingly minute channels lined by a single layer of flat pavement cells. They resemble blood capillaries in their general structure but they are even smaller in diameter and their walls are thinner. Through them there flows a thin, watery fluid known as lymph. Apparently the flow of this fluid is more or less regulated by the motility of the parts through which the vessels pass. There may also be negative pressure exerted on the proximal end of these vessels producing a certain degree of suction.

In the lung we find that there are two main sets of lymph vessels. One of them, known as the superficial set, runs around the outer surface of the organ, through the pleura and empties into the tracheobronchial lymph nodes; the other, or deep set, courses through the substance of the lung as a series of trunks, situated in the walls of the pulmonary artery, the pulmonary vein, and the bronchi. This system also discharges its lymph into the tracheobronchial lymph nodes. These two systems, the superficial and deep sets, are connected with one another in the periphery of the lungs by a series of short thick lymphatic vessels which allow lymph to escape from the deep set into the superficial one.

Lymphoid Tissue. Scattered along the course along the lymph vessels in the lung, are masses of lymphoid tissue. These consist of aggregations of small, round cells known as lymphocytes. Between these cells are minute spaces communicating with a lymph vessel. Foreign bodies carried from the lung by phagocytes make their way through these masses of lymphoid tissue into the lymph vessel. They may become enmeshed in this tissue and held there for a variable period of time, or they may pass on into the lymphatic vessel. The number and size of the lymphoid nodules increases as one progresses from the periphery toward the root of the lung. It is found that lymph nodules occur at points where lymph trunks join one another. These points also correspond to places where large blood vessels and bronchi branch.

The tracheobronchial lymph nodes are more complex in their structure than the simple lymph nodules inside of the lung. They might be considered as an aggregation of simple lymph nodules. They are surrounded by a capsule of connective tissue inside of which is a wide channel surrounding the whole node. The entering lymphatic trunks empty directly into this wide channel. Leaving this channel there are other small vessels which penetrate into the substance of the node and again empty into a large, dilated lymph space at the root of the node. From this last mentioned channel other lymph trunks carry the lymph away from this node to other nodes in the same system. Between this network of minute lymph channels, there are

large numbers of lymphocytes whose function we need not consider in this connection.

The lymph node has been called a filter. It should really be called a sedimentation basin because it does not hold up foreign particles by virtue of the smallness of its spaces, but rather by slowing down the rate of flow of the lymph and allowing foreign bodies to settle out. The system might be compared to a stream with a series of ponds along its course. Pulp wood thrown into the stream would be carried along fairly rapidly in the narrow channel, but on reaching one of the ponds much of the wood would tend to drift onto the shore and remain there for an indefinite period; some would pass directly through the pond to settle out in one of the other ponds further down the stream.

TREATMENT OF INHALED FOREIGN BODIES

Inhaled dust or other foreign bodies like bacteria may be caught on the sticky mucous membrane of the nose or throat and swept backward by ciliary action to the pharynx from whence they may be expectorated. If such particles penetrate into the trachea and bronchi, the action of cilia in these structures tends to carry them outward toward the pharynx from which they may also be expectorated. If the foreign material escapes the above mentioned protecting devices and penetrates into the terminal air spaces, the following series of mechanisms come into action.

One of the dust cells which, as we have seen, develops in the wall of the air space will become detached and make its way toward this foreign body. When accidental contact has been established, the dust cell puts out processes from its substance to surround and engulf the offending particle. The cell then makes its way by a slow creeping motion over the walls of the air spaces until it reaches the nearest mass of lymphoid tissue. Here it tends to pause and in most instances it penetrates into the substance of the nodule. Like the drift wood in the stream, the cell with its foreign material may be held up in the first pond or it may pass down the stream to be held in some other pond further along the course.

Experimental study has shown that the character of the

foreign material has a well marked influence on the behavior of the dust cell or phagocyte. With substances like carborundum or soft coal dust, a single cell will ingest all the particles with which it establishes contact. The result seems to be that the cell becomes over engorged and its power of locomotion is greatly impeded. As a consequence we find that cells filled with such materials tend to remain in the peripheral air spaces. Only a few which have been less greedy succeed in making their way very far into the lymphatic system. In the case of quartz on the other hand, it has been observed that individual cells tend to take up relatively few particles and that once they have ingested the particles the cells seem to be stimulated so that they migrate more rapidly than usual. Quartz dust is known to be an irritating substance and it seems reasonable that it should stimulate the cells in the manner described. When irritation has been carried on sufficiently long many phagocytes die and liberate their engulfed quartz particles. New phagocytes must be attracted to the spot to take up the material thus freed. As a consequence of this stimulating effect upon the motility of the phagocytes, we find that quartz tends to be concentrated in the lymphatic system much more rapidly than other types of dust.

As will be seen, the same cell which handles the fine dust particles also ingests bacteria like tubercle bacilli. These organisms are also carried into the lymphatic system and concentrated at the same point where dust has been collected. The action of the two irritants in the same location is more pronounced than that produced by either one alone.

Observation of human lungs and those of experimental animals has shown that where the amount of foreign material inhaled is excessive there is a marked tendency for such substances to be carried into the peripheral portions of the lung. The reason for this is not clear but possibly it may be due to the massaging action of respiratory movement. If one compels a guinea pig to inhale a considerable cloud of a substance like carmine dust, which can be readily recognized, one finds on killing the animal some fifteen minutes later that much of the dust is in the subpleural air spaces. In other animals, allowed to live longer, the major portion of the dust has made its way to this location. In soft coal

miners with long exposures to a readily recognizable black dust who have been away from their occupation many years before they die, most of the dust will be discovered in the pleura and along lymph vessels radiating from it.

MR. D. E. CUMMINGS: I wonder if it wouldn't be of interest if Dr. Gardner would describe the efficiency of the pulmonary mechanism with regard to the size of the particles which might enter the air spaces in the lung, and also if it wouldn't be worth while to describe the mechanism (in addition to the cilia) that tends to remove the particles from the terminal bronchi of the lung.

DR. GARDNER: I neglected to speak about what happens to the material that has been deposited in the lymph nodes at the root of the lung. A great deal of dust stays there; in fact the larger part of it stays there, but there is an overflow channel from these nodes into a large lymph vessel that finally empties into the veins. The blood would then carry any overflow material from these nodes back to the right side of the heart from whence it would be distributed again to the lung. In this location the larger particles would be filtered out of the blood vessels and carried into the air spaces where phagocytes would carry them by way of the lymphatics back to the tracheobronchial lymph nodes. Thus if dust once gets out of the tracheobronchial nodes it is carried by the blood back to the lungs. But the very finest particles do not leave the blood stream through the capillaries of the lung. Some of them pass on into the pulmonary vein, are carried to the left side of the heart and then distributed to the other organs of the body like the liver and spleen. As a matter of fact, in far-advanced silicosis we occasionally find that there are nodules forming in the spleen and in the liver. This is particularly true of the guinea pig, probably because of anatomical peculiarities, but it can also happen in some human beings.

Mr. Cummings also called attention to another thing that I forgot—the limitations of the ciliary mechanism for various sized particles. We know that there are very few particles greater than ten microns or 10/25000 of an inch in diameter that succeed in passing the protective mechanism in the upper respiratory tract and finally reach the

deeper parts of the lung. In fact relatively few particles as large as this maximum are found in the lung. The majority are only three or four microns in diameter. While we know that the very small particles do penetrate in large numbers, we do not know whether all of them remain there. Whether the cilia may be more effective in carrying particles, one micron and less in diameter, out of the lung is still problematical. It is an important question, however, and one which we hope to investigate because we have shown experimentally that the smaller the particle the greater its biological activity. This is a point of not only academic but of practical significance which will probably be brought out in this discussion today or tomorrow, because it has to do with the size of particles which should be included in dust counts for estimating industrial hazards. If the cilia effectively eliminate all of the very small and hence most active particles we need not bother to include them. If, on the other hand, the cilia and other protective mechanisms do not eliminate them then we must modify our methods in dust determination so that they will be included in the counts. We propose to do some experiments in which we will attempt to discover the truth of this matter.

There are those who claim that a great deal of dust which has reached the terminal air spaces in the lung is carried out again along the pathway of ingress and expectorated. We know that a coal miner for example, living in the relatively clean atmosphere of a health resort for a period of years continues to expectorate black sputum for a long period of time. The pigmentation must be due to the dust which he inhaled during his employment as a miner, because in a place like Saranac Lake there is relatively little or almost no soft coal in the atmosphere. I doubt whether a normal person without any infection in his lung will continue to expectorate such dust, for dust which was inhaled ten, fifteen or twenty years previously in the coal mine tends to be anchored and held in the normal lung. On the other hand, if the miner should develop a pneumonia or other acute inflammation of his lungs, the once anchored dust will be freed and liberated. The pus that forms as the result of pneumonia, tuberculosis or other in-

flammation will be expectorated and will carry out the dust that has been freed in the inflammatory process. I am not prepared to agree that a normal bronchial tree will continue to eliminate dust over an indefinite period of time. Possibly Dr. Willis would still disagree with me. I will give him a chance to reply if he would like to.

DR. WILLIS: I just pointed that out as a matter of observation and I just want to draw attention to the fact that anatomical conditions do exist in certain animals and that it may well be the basis. Of course, the dust does tend to anchor, but if it does anchor in lymphoid tissue I think it passes a grade for a certain amount of elimination, but until we know whether that is true, why, of course, it is mere speculation to say that is the way it comes out.

DR. GARDNER: I may state that I have exposed guinea pigs and rabbits to dust inhalation for periods of a year and then allowed them to live as long as they would, perhaps three years, after the discontinuation of the dust exposure. A guinea pig does not cough and does not expectorate as a human being does; dust that might be eliminated through the trachea would be swallowed again. If it were swallowed a good deal should be absorbed through the intestinal wall where it would tend to localize in masses of lymphoid tissues in connection with the intestines. There are such lymphoid tissues throughout the intestinal tract of all animals. We have not found any proliferation or pigmentation in this lymphoid tissue. Therefore, if the dust were eliminated through the trachea and then swallowed it either passes directly through the intestine leaving no trace, which would be hardly likely, or else it is not swallowed.

DR. WILLIS: It seems to me to take that position of the non-absorption through the intestinal tract we would have to compare it with tubercular bacilli. It takes several thousand times as large a dose of tubercle bacilli by the intestinal tract by infection than it does any other ordinary unit. It may be you would have to have a larger portion of the intestine contain dust, for dust to be absorbed by the intestinal tract and even in a guinea pig, if it should swallow its dust, it wouldn't be in sufficient quantities to make

a dent. That is purely theoretical and has no big significance.

DR. GARDNER: The next thing on our protocol is a discussion of pathology.

MR. TARRELL: A few minutes ago I understood that Dr. Gardner invited questions. If I may be permitted, there is one question that occurs to me at this time with reference to the elimination of different dust particles. You mentioned the rapidly moving phagocytes and slow moving phagocytes. From a safety standpoint which is more efficacious, the rapidly or slower moving phagocyte?

DR. GARDNER: It has been our experience in studying various dusts, as we shall attempt to show in the discussion of the reaction to different materials, that the man who inhales quartz dust and as a consequence has rapidly moving phagocytes, tends to develop disease more abundantly and becomes disabled much more quickly. In case of carborundum or aluminum oxide on the other hand, the phagocytes move slowly or not at all. The existing evidence would indicate that these dusts do not produce disabling disease of the lung. The rate of movement of phagocytes is not the whole story but it is the initial step in the development of dangerous pulmonary fibrosis. Answering your question directly, I would say that the stimulation of migration of phagocytes exerts an unfavorable effect by concentrating irritating dusts in positions where they excite dangerous proliferation. Are there other questions?

MR. NELSON: Doctor, can you say something about the efficiency of first line defenses in the nasal tract and trachea?

DR. GARDNER: Jarvis, for example, in Vermont made a study of the degree of silicosis in mouth breathers and in nose breathers and attempted to show that the mouth breather develops more silicosis than the nose breather. There have also been other references of this nature in the literature in various parts of the world. It is said that a man who inhales dust over a long period of time tends to develop a chronic inflammatory condition in his upper respiratory tract, that when such chronic rhinitis and

tracheitis exist, the action of the cilia is so impaired that more dust passes these barriers than would in an individual with a normal protective mechanism. In our guinea pigs which have been subjected to the inhalation of dust over a period as long as four years, we have failed to discover anatomical evidences of disease in the cells of the upper respiratory tract. They appear normal. Whether they are functioning as well is quite another matter which I am not prepared to discuss. I would infer that a chronic inflammation would tend to impair function and efficiency of the mechanism and that if a man breathed through his mouth he would obviously tend to inhale more dust because he would not have the full benefit of this great surface in the nasal cavity that Dr. Willis mentioned. Another question?

DR. CLARK: Can you give an estimate of the amount of surface which is represented by the lung itself? We have it for the nasal surface. Can you give an estimate on the amount of space there is?

DR. GARDNER: I don't remember. Do you, Mr. Cummings?

MR. CUMMINGS: I believe it is something like two hundred square inches.

DR. BANYAI: Do you agree that the length of the dust particle may be as much as two or three hundred microns, as for instance in the case of asbestos particles?

DR. GARDNER: Yes. We have seen very long asbestos fibres in the lung. Possibly ciliary activity is not adequate to move foreign bodies of this shape. However, we do not find many fibres of asbestos as long as this. The very long asbestosis bodies which are found in the lung we believe have probably grown in length as well as in diameter about a fibre which came to rest in the terminal air spaces.

DR. BANYAI: Is there any primary predilection of the space in the lungs besides the pleurae due to the particular mechanism of the respiratory function?

DR. GARDNER: I will consider that in a few minutes, as it is on the protocol. Other questions?

MR. NELSON: What is the ultimate destination of the carborundum cell that you located at the terminal air space?

DR. GARDNER: The majority seem to stay where they have drifted into the subpleural air spaces. In an animal exposed a year and allowed to rest with no further dust exposure for three or four years, we find practically all the dust cells in the air spaces beneath the pleura. I will show some pictures to illustrate this point in a few moments. The cells tend to remain inside the air spaces and certain air spaces become almost completely filled with masses of phagocytes packed with carborundum particles. With coal dust the localization is a little different. We will go into these matters with lantern slides. Any further questions?

DR. BELLIS: Would the accumulation of large numbers of cells of that type interfere mechanically with the function of the air spaces?

DR. GARDNER: Probably it would because, as we saw before, there is an irritation of the wall of the air space that contains a number of these phagocytes. The wall becomes thickened so that the gaseous interchange does not take place normally. However, it seems to be the case that only certain air spaces tend to accumulate these dust cells and there are many others which remain apparently normal, so that the function of the lung as a whole is not impaired. The factor of safety in the lung is great for one can carry on perfectly well with only a quarter of his respiratory tissues.

DR. WILLIS: I wonder if you would care to mention the probable proportion of dust that is held up by the defensive mechanism.

DR. GARDNER: I don't know what it is. It probably varies with the different types of dust. We know that as you found and as I found, it is a difficult thing to get very much coal dust into the lungs of guinea pigs. When you make them breathe carborundum, a great deal more gets in. If they breathe quartz dust in the same concentration, a tremendous amount gets in. I think that the amount must be concerned with physical characteristics of the dust itself.

I have always reasoned in my own mind that if the dust was dry it would tend to be inhaled. If it wet easily it would tend to stick to the wall so that a good deal would be held up in the upper respiratory tract. Frankly we haven't the information which we need on this subject; it should be studied much more carefully than it has been.

DR. WILLIS: It has been estimated that perhaps not over 20% of the dust on the average is in the lungs. I wonder if you have come across any dependable evidence for the accuracy of that statement.

DR. GARDNER: Mr. Cummings can quote Dr. Drinker's work in that respect and he also has some other information.

MR. CUMMINGS: Dr. Drinker attempted to answer that question by creating experimental dust chambers in which he measured the concentration of the dust in the air very accurately. He then had cats breathe this dust concentration and measured the concentration in the exhaled air. He used for the purpose of his study a dust, the concentration of which he could determine by chemical means. When he used magnesium oxide he found that the degree of retention depends on the concentration of the dust in the air. With the increase of concentration the degree of retention increased and his work showed that for moderate concentrations, comparable to the amounts of dust present in an ordinarily dust industrial air, that approximately 50% of magnesium oxide was retained in a cat's lung. He then carried this work over to short exposures in man and found that the results were roughly comparable to those for the cat. As the concentration in the air increased, the degree of retention increased. Dr. Gardner has pointed out that magnesium oxide and silica produce an entirely different response, and as yet Drinker has used only dusts that can be determined by simple chemical reactions.

MR. TARRELL: Have you made any estimate as to how much impairment of the vital capacity of the lung results from the lodgment of carborundum particles?

DR. GARDNER: No, we have not carried on such a study. I have learned that it is now being investigated in Roches-

ter by Dr. McCann and his associates. I think we may look within the next year for some definite information. Dr. Meyers of Minnesota did make a study of the vital capacity of men suffering from various forms of pneumoconiosis but his figures were not particularly significant. Vital capacity studies were also carried on in South Africa, but they did not feel that their results were of sufficient value to deserve much emphasis. Obviously, if a man has a very advanced silicosis complicated by tuberculosis, a great deal of his functioning lung tissue is out of commission and his vital capacity would be greatly reduced, but for the early and intermediate stages of the disease the test is not of great practical value.

DR. RUSSELL: I wanted to ask if the retention of magnesium oxide included retention in the upper respiratory tract as well as the lungs.

DR. GARDNER: Yes.

DR. RUSSELL: The point is that much of this dust is retained in the upper respiratory tract. We don't know the proportions in that in relation to the amount retained in the lung proper. I was afraid the impression would be left that all of that dust was retained in the lung tissue itself. Dr. Gardner has explained that the protective mechanism of the upper respiratory tract is quite efficient in most cases.

PATHOLOGY

DR. GARDNER: If there are no further questions we will go on to the next part of our protocol which deals with pathology. The first title here is *action of dusts*, chemical, mechanical, etc.

When man first began to observe the development of fibrosis or reaction in the lung of individuals inhaling dust in considerable quantities, it was natural to assume that hard sharp particles would be dangerous because they would cut the lung surfaces and other delicate structures. Further study, however, showed that not all hard sharp particles were dangerous and when Collis and some of the German observers analyzed the statistics of men working in various dusts they found that those working in rock containing

large amounts of silica were the ones who developed the most extensive disease, the most rapid formation of fibrous tissue. Gye and Kettle demonstrated that the soluble form of silica was a tissue poison; that in high concentration it would kill an animal; that in weaker concentration it would not kill but it would injure and that this injury was followed by the formation of scar tissue. On this basis has arisen the so-called solubility hypothesis to explain action of dusts. It has been assumed on the basis of Gye and Kettle's observation that crystalline silica dissolves in the alkaline fluids of the body and liberates poisonous soluble silica which is responsible for the development of scar tissue. It has never been possible to demonstrate the solution of silica in the tissues for its proof is beset with technical difficulties about which Mr. Cummings will have more to say later. Therefore the solution hypothesis still remains unproven. In its support Belt has called attention to the observation that the number and size of silica particles inside silicotic nodules apparently diminish with the age of the nodule; that the older the lesion is, the fewer particles it will contain. I think his work may be criticized, for many of the particles may become coated with body fluids so that they are no longer recognizable. However, if the tissue elements in a section of a silicotic nodule are destroyed by incineration and strong acids the silica particles are readily visible with polarized light. This is true both of early and of many old lesions. The morphological evidence of solution is of doubtful significance. We still have to prove then that silica does dissolve in the body. However there is indirect evidence which indicates that the action of silica may be chemical or physico-chemical in its nature. We know that not all hard and sharp particles are equally injurious while typical silicotic nodules can be produced with pure silica either in its crystalline form or in an amorphous state. We know that carborundum, a substance whose particles are equally hard and sharp, does not produce the same type of reaction. We know that aluminum oxide, about which we will have a good deal to say later, will not produce a progressive fibrosis of the tissues. In fact aluminum oxide seems to be an inert substance which can stay in the tissue for long periods of time without producing any

in the same way as dust particles. Lodged in the terminal air spaces they will be phagocytosed by wandering cells and transported to the nearest lymphoid tissue. These foreign particles differ from dust in that they are alive and capable of multiplication. As a consequence they tend to excite a more rapid and more extensive reaction. In the lymph nodules there is a new growth of cells similar to those seen in silicosis. The continued proliferation of these cells, results in the formation of a nodule, or, in Latin, a tubercle. Poisons liberated from the bacilli kill many of the cells in the center of the nodule with the formation of a special type of degeneration known as caseation, from its resemblance to cheese. This degenerative process may extend to involve the whole nodule, or at any time it may subside and be replaced by lime salts which are attracted to the area from the blood. The deposition of calcium indicates that the tuberculous process is healing. Not all of the tubercle bacilli remain within the lymphoid tissue within the lung. Some of them escape through the lymphoid tissues to the tracheobronchial lymph nodes. In this location they produce more tubercles. The reaction here is usually more extensive than that in the lung because of the previous multiplication of the bacilli in the former location. The formation of tubercles beneath the pleura of the lung and in the tracheobronchial lymph nodes constitute the essential reaction of childhood tuberculosis. The lung tubercles may appear in any part of the organ, but they are usually found in the middle zone. They may be single, or they may be multiple.

Their outcome depends upon the number and the virulence of the tubercle bacilli producing them. If many fully virulent organisms are inhaled, they set up a primary tubercle such as has been described, but very quickly the infection spreads and becomes progressive. Often under such circumstances the outcome may be fatal within a few months. More usually, however, only small numbers of tubercle bacilli, perhaps attenuated by partial drying and exposure to sunlight, are inhaled. In this case the primary tubercles in the lungs and tracheobronchial lymph nodes tend to heal, usually with the formation of a fibrous tissue and the deposition of lime salts. Such healed primary foci

of infection often persist throughout the life of the individual. They may be detected by the X-ray.

Even though they produce no apparent damage they exercise a very definite effect upon the cells of the body as a whole. The establishment of the primary focus renders the body partially immune to subsequent infections with the tubercle bacillus. It can be shown experimentally that a much larger dose of tubercle bacilli is necessary to produce effective reaction in a previously infected guinea pig than is required in a normal animal. Morever, the immunity tends to slow down and render chronic the course of the subsequent tuberculous infection.

A second effect of this childhood tuberculous infection is to produce a condition known as hyper sensitiveness. This is manifested by an inflammatory reaction when the animal previously infected is again treated with tubercle bacilli or its products. The familiar tuberculin test is an example of such hypersensitiveness. If a minute amount of tuberculin is injected into a normal subject, no significant reaction occurs. But if the same amount of tuberculin is injected into a person or an animal previously infected with tubercle bacilli, a progressively increasing inflammation makes its appearance at the side of the injection. Such tuberculin tests are used in surveys to demonstrate the amount of infection in a community. They merely indicate that somewhere in the body there is a focus of infection. This may be inactive and of no clinical importance, or it may be a progressive and dangerous type of tuberculosis. As far as the test is concerned, it simply indicates some previous effective contact with the tubercle bacillus. The frequency of childhood infection varies with the environment. Among the population of European cities, practically one hundred per cent of persons above twenty years of age react to tuberculin. In the United States the percentage is variously estimated from forty to ninety per cent. In rural districts there are some groups which show very few reactions. In others they may run as high as forty per cent.

APICAL TUBERCULOSIS

The second recognizable form of tuberculosis of the lungs is found in the apex of the lung. The relationship of this disease to that of the childhood type is still debatable. The infection at the apex may arise as a result of spread from the primary childhood foci or it may be due to a new infection from without. Whatever the source of the bacilli they tend to localize in the apex of the lung. The reasons for this localization are still unknown, and the various theories offered to explain them need not be discussed here. Because of the influence of the primary childhood infection, immediate reaction to a new implantation of bacilli in the apex of the lung is inflammatory in character. It consists of a localized patch of pneumonia due to the tubercle bacillus. The extent of the reaction and the severity of the attendant symptoms will depend upon the number of bacilli localizing in the area and upon the degree of sensitiveness to the disease. The latter factor is determined by the interval elapsing after the establishment of the childhood foci. The outcome of an apical tuberculosis may be localized progression with possibly the formation of a small cavity. Such a result might be expected where the dosage of bacilli is relatively small. Under favorable conditions such a lesion would tend to heal after a period of time. The healing would be attended by the formation of considerable amounts of scar tissue and the calcification of considerable areas of degeneration. Contraction of the scar tissue produces some deformity of the lung, and such changes are readily visible by X-ray. The healed focus may persist in this condition throughout the life of the individual, never giving him further trouble. As evidence of this, one finds that in doing routine autopsies on persons dying of various causes, apical scars are extremely common. It used to be said that the lungs of ninety per cent of urban residents would show such changes. On the other hand, an apical focus partially healed may become reactivated after a long period of latency. The factors responsible for reactivation are not well understood. When this occurs, the disease again spreads locally through the air spaces, to involve larger portions of the upper lung, and possibly to extend

throughout the lung. If a small cavity should rupture into a bronchus, tubercle bacilli are discharged into a position from which they may be carried to the lower portion of the lung. This brings us to a consideration of the final stage, namely, chronic pulmonary tuberculosis.

CHRONIC PULMONARY TUBERCULOSIS

The distribution of tubercle bacilli by the bronchial tree may be confined to the immediate vicinity of the old apical tuberculosis or it may be disseminated throughout all portions of the lung. The character of a new reaction produced in these locations will again depend upon the number of tubercle bacilli involved, and the degree of sensitiveness of the tissue. If there are many bacteria, and the soil is highly sensitive, the reaction will take the form of an acute broncho-pneumonia. Clinically and pathologically this may simulate a pneumonia produced by other bacteria. The patient may be acutely ill, and possibly die during this episode. On the other hand, he may survive, and, if conditions are favorable, much of the disease will resolve. Sometimes large cavities may be formed; at others the disease heals with the formation of a considerable amount of scar tissue, and more chronic cavities. If the number of bacilli is fewer and the tissue are less sensitive, the resultant disease produced by aspiration tends to be nodular in type, accompanied by the formation of scar tissue and, sometimes, cavitation.

Hemorrhage is a not infrequent accompaniment of any of these forms of chronic pulmonary tuberculosis. It may result from the erosion of a large blood vessel which passes through an area undergoing cavity formation, or it may be due to oozing from minute blood vessels in the walls of cavities.

MILIARY TUBERCULOSIS

This is so named because the tubercles seen are about the size of the millet seed. Miliary tuberculosis is due to a distribution of tubercle bacilli by way of the blood stream. It may occur as an accidental complication of any of the

above forms of tuberculosis. The organisms gain access to the blood stream either through the activity of migratory cells or as the result of an erosion of a large blood vessel. The bacilli will be distributed to portions of the body supplied by the blood vessel involved. For example, if rupture occurs into a branch of the pulmonary artery, miliary tuberculosis will develop in the portion of the lungs supplied. If, on the other hand, there is a rupture into a pulmonary vein, the blood will carry the bacilli back to the left heart and thence to the other organs of the body. Frequently they lodge in the brain and its lining membranes. As a consequence, tuberculous meningitis is a frequent complication of miliary tuberculosis. The outcome of this disease again depends upon the number of bacilli involved. Occasionally a very few organisms pass into the blood stream and set up only a few tubercles in various organs. These heal without demonstrable symptoms. On the other hand, where large numbers are poured into the circulation an acutely fatal disease results which may terminate within a few weeks.

SILICOSIS AND TUBERCULOSIS

It is well known that the silicotic individual is peculiarly susceptible to infection with the tubercle bacillus. Clinical investigators are generally of the opinion that the infection is a new one from the outside acquired subsequent to the development of the silicosis, but by animal experimentation we have been able to show that the inhalation of pure silica dust will light up and reactivate a partially healed childhood type of tuberculosis in guinea pigs. Furthermore there are many cases studied by X-ray which suggest that a partially healed apical focus of tuberculosis has been caused to spread by the inhalation of dust. In South Africa, Dr. Ervine demonstrated several beautiful examples of such reactivation by serial X-ray examination of the same individuals. For several years men with apical scars continued to work in the gold mines with little demonstrable change in their X-ray pictures. In time these areas began to spread, and finally the typical snowstorm picture of silicosis with tuberculosis was produced. Dr. Russell gave me an oppor-

tunity to study some of the lungs from his Barre granite cutters. The number of cases was too small for statistical analysis, but I found that in a few instances the tuberculosis was apparently extending downward from an old focus in the top of the silicotic lung. Most of the lungs, however, showed an atypical distribution of the infection. The older infectious lesions where large cavities had developed were in the lower lungs and not in the apex. The latter group I interpreted as due to infections acquired subsequent to the period of dust exposure.

In the laboratory we have studied the effects of inhaled dust upon tuberculous infection comparable to the childhood type of tuberculosis seen in man. We have not yet been successful in producing changes analogous to apical tuberculosis which behave with sufficient constancy for experimental observation for either the disease would heal so completely that it would no longer be affected by inhaled dust or it would remain so active that it spread in the control animals with no dust exposure. I shall, therefore, confine my remarks to primary tuberculous infection as modified by dust inhalation. When such infection is produced in normal guinea pigs with an attenuated tubercle bacillus of low virulence it tends to heal spontaneously. A year or two after infection most of the subpleural tubercles have either disappeared or have been reduced to nodules of scar tissue with more or less calcification. In the tracheobronchial lymph nodes there is usually more evidence of the infection. There is ordinarily no spread to the other organs. Animals infected in this manner usually react positively to injections of tuberculin in the skin throughout the remainder of their lives, indicating a persistence of some slight activity of the infection.

If an exposure to quartz dust is commenced on the day after infection with such an attenuated tubercle bacillus the disease follows its usual course for four or five months when rather suddenly the subpleural tubercles take on renewed activity. The disease spreads locally through the lung, and small cavities develop at the site of the original lesions. At the same time bacilli gain access to the blood stream and are transported to the liver and spleen where progressive tuberculosis develops. The result is a generalized chronic

tuberculosis which may last for two or more years but which ultimately terminates fatally.

If on the other hand animals with fully developed silicosis are infected with attenuated tubercle bacilli an acute form of tuberculosis is produced which kills them in from one to three months. The cause of this alteration of the tuberculous infection by the coexistence of silicosis is most interesting. In fact it was the observation of this phenomenon in human beings which attracted us to the problem. Thus far it seems obvious that the dust itself does not influence the bacillus. If quartz or other dusts are added to artificial culture media no significant changes in the rate of growth of tubercle bacilli planted in that media are observed. We as well as other observers have noted that in quartz containing media the lag period regularly ensuing before new growth starts is reduced, but this we believe is due to a buffering action which silica is known to possess. In other words the silica absorbs acids liberated by the bacilli so that these substances do not impede growth.

Furthermore it has been discovered that the tubercle bacilli recovered from acute or chronic cases of silico-tuberculosis do not differ materially from those originally injected. For example, a culture is made from an acute cavity in a guinea pig with silicosis subsequently infected with attenuated tubercle bacilli. These bacilli are injected into a normal guinea pig. They produce only non-progressive tuberculous infection. The same result is produced if the ground up lung tissue of the animal with acute silico-tuberculosis is injected directly into a normal animal. Apparently neither the silica nor the silicotic tissue produces any permanent change in the infecting tubercle bacillus.

We are forced to conclude that silica particles excite some change in the tissues which temporarily favors the growth of the organism. As a matter of observation it can be stated that the tuberculous process does not begin to spread until the degenerative changes begin to appear in the silicotic nodules. We have interpreted these observations to signify that the degeneration of silicosis is the factor responsible for the growth of the tubercle bacillus. Whether this hypothesis will be substantiated by further experiments remains to be seen.

Finally the property of stimulating tuberculous infection seems to be specifically associated with various forms of silica. Soft coal and marble dusts are without appreciable effect upon primary tuberculous infections with attenuated *tubercle bacilli*. Asbestos, a silicate of magnesium, causes the tuberculosis to progress for a short time, but it soon heals by fibrosis and rarely results in death. Granite dust composed largely of the silicates feldspar and mica, with only about 25 per cent of free silica retards the normal process of healing of such infection. Prolonged inhalation of this dust will reactivate a partially healed primary tuberculosis in a few members of a large group of animals. Experience with human beings exposed to granite dust would indicate that longer exposures than it has been possible to employ with guinea pigs might produce more pronounced effects. Carborundum dust, the carbide of silicon, behaves in an unusual way which cannot be explained by the facts thus far in our possession. Although the manufacturers' analysis of this material shows that it contains less than one per cent of free silica, yet it is almost as potent as quartz in its stimulation of a tuberculous infection. The only explanations possible at the present time are either that the material contains more than the stated amount of free silica or that the body can act upon this substance to liberate silica in an active form.

The picture produced by the combination of tuberculosis and silicosis is not simply one in which the two conditions coexist side by side, but it is often that of a new disease. The silica particles and the tubercle bacilli may be carried by the phagocytes into the same lymphoid nodule. Both exert their effect upon the same tissues, and a large nodule having characteristics of both conditions is produced. Furthermore there is a greater tendency to diffuse reactions in the walls of the air spaces so that wide areas of the lung tissue may become uniformly fibrous. The causation of such reactions is often difficult to recognize, particularly in cases seen late in the course of the disease. This is true both in the roentgenographic and in the postmortem method of examination.

There has recently been considerable discussion of acute silicosis which has appeared after a few months' or years'

exposure in sandblasting, and pulverizing plants, and among the manufacturers of abrasive soap powders. I have had opportunity to examine sections of the lungs of several sandblasters and pulverizers all said to have a silicosis produced in less than two years. Microscopically I have felt that all of them have showed evidence of a complicating tuberculous pneumonia, but it is atypical in its manifestations. Dr. Kettle has told me that in the sections of acute silicosis from English scouring powder manufacturers there was also evidence of such infection. Many of these cases from both sides of the Atlantic were diagnosed as uncomplicated acute silicosis by X-ray. I do not wish to infer that excessive quantities of exceedingly fine silica may not produce an unusually rapid form of silicosis but I do wish to emphasize the fact that the tuberculous factor may accelerate the dust reaction and produce a form of disease which is difficult to recognize.

Another way in which the coexistence of tuberculosis may influence the development of pneumoconiosis is through its effect upon the lymphatic drainage system. Healed childhood infectious lesions often seriously damage the tracheobronchial lymph nodes. Obviously lymph cannot flow in a normal manner into a node whose channels are compressed and distorted by fibrous and calcified tubercles. If such an individual enters a dusty trade the accumulating particles cannot be removed from the air spaces of his lung as in the normal person. Many of them must tend to be held either in the air spaces or in the other portions of the lymphatic system. It is my belief that such a man will develop silicosis rapidly and that this may explain why some persons may require forty years' exposure to granite dust before developing significant changes in the lungs while others seem to accomplish the same result in four or five or perhaps ten years. It has been claimed the "individual susceptibility" to inhaled dust is the explanation for these differences in reaction time, but surely these words explain nothing. Until we have more carefully studied evidence from a large series of post-mortem examinations we can do no more than speculate upon this subject.

In this connection, it is often assumed that a group of men exposed to a given concentration of dust will develop

pneumoconiosis in approximately the same time. For example, in the old days of dry drilling in South Africa when no protective measures were in force, the *average* period required for the development of silicosis was four years. Today when the wet process is in use and every precaution is employed to reduce the dust concentration, the *average* period is fourteen or fifteen years. But in both instances there are exceptions. Some drillers become affected in less than the average time and others are apparently immune for many times the average period. It is a mistake to assume that such an entity as a normal human being exists. He may appear normal and be able to carry on under the usual conditions of environment. When exposed to unusual surroundings apparently minor defects may render his body quite incapable of coping with the situation.

Thus the evidence would indicate that inhaled silica dust may either reactivate and cause to become progressive a pre-existing focus of latent tuberculosis or it may favor the growth of a new tuberculous infection from the outside. It may produce a new type of disease which is neither silicosis nor tuberculosis. In either case the inference is clear that unusual care should be taken to prevent the same person from inhaling both tubercle bacilli and silica dust.

In South Africa the method of excluding from exposure to silica dust every person who shows detectable traces of tuberculosis in any form is rigidly adhered to. Dr. Russell and I were surprised to find applicants rejected whose X-ray films showed evidence of healed childhood tuberculosis. Others diagnosed as "more fibrosis than usual" or what we would call "increased prominence of linear markings" were likewise refused employment. Men with obvious healed apical scars were summarily rejected. Their experience with compensation costs has led them to insist upon men who are free from every evidence of pulmonary tuberculosis, active or inactive, which can be detected by X-ray or physical examination.

This they are doing for their own protection, for they have learned that silica dust may transform an inactive focus into an active one. The particular individual may become incapacitated for work and claim compensation. Furthermore, when his infection becomes active he may ex-

pectorate tubercle bacilli in the working places of the mine and thus become a source of danger to his fellow workers who are exquisitely susceptible to the tubercle bacillus. But even these measures are not proving successful, for tuberculosis is increasing though the silicosis rate is decreasing among the mine population. The reasons for these changes are not apparent. But they are now recruiting their white miners from the rural districts of South Africa rather than from Europe, as heretofore. Such individuals have often escaped previous contact with the tubercle bacillus, but if they have developed lesions of infection they are excluded by X-ray examination. Possibly the Miners Phthisis Bureau has erred on the side of apparent safety and has introduced into its mines a generation with no immunity (because of no childhood infection) to tuberculosis. But truly it would require more knowledge than we possess today to determine from an X-ray film what primary childhood focus would confer the requisite amount of immunity and yet would not be activated by the inhalation of silica dust.

PNEUMONIA

Throughout the world the incidence of pneumonia is extremely high among groups exposed by occupation to dust. This is true not only of silica but of many other dusts. Dr. Russell can speak for the coal miners. Among guinea pigs exposed to dust, pneumonia has proved to be a terrible scourge. Sometimes a group of 100 to 200 animals may be completely destroyed and the whole experiment must be repeated. It has been our experience that the pneumonia rate is highest in guinea pigs exposed to silica but it is also high where other dusts are involved.

I believe this is a logical place to pause for a few moments. If I can answer questions now or if anyone else wishes to discuss this phase of the subject I would be glad to have him do so.

MR. KUECHLE: I had an opportunity to go to Pitcher, Oklahoma, this June and Dr. Merewether found that a man with syphilis contracted disabling silicosis with the same exposure in just half the time a well man did.

DR. GARDNER: Let Mr. Cummings discuss that. Mr. Cummings has been in Pitcher.

MR. WHITE: You spoke of infantile tuberculosis from which recovery is taken and that those who are found to have had that on application for entering a mine should be eliminated. If we follow that practice of elimination of applicants who had had that infantile tuberculosis, my information is that the infantile tuberculosis which has healed is so prevalent that we would eliminate probably a very large portion of our applicants and the question arises whether we could follow that practice as a practical matter and get the men for work.

DR. GARDNER: I would like to discuss that a little further. I am glad you brought it out.

MR. WILCOX: You said you wouldn't advise the taking of those persons into industry which unduly exposed them, not industry generally.

DR. GARDNER: Mr. White's point that so many of these people would be inclined to show evidence of childhood infection, particularly among some labor groups, that he wouldn't have anybody left to work his particular industry if he excluded them, is a practical one. In South Africa it was the practice to exclude all such cases where evidence of infantile infection could be demonstrated, but there they are working with a population which had not been generally tuberculized. Our experimental infections were more recent and were not particularly well healed. The case is different when you deal with a population long in contact with white civilization. Among the older European races, for example, the adults probably carry those lesions which are so completely healed that they would never be reacti[illegible] Many of them are sterile and they no longer contain tubercle bacilli. It would be a matter of judgment to determine in the individual case whether a particular spot was liable to break down or whether it was not. If it consisted of a small deposit of lime salt not bigger than a buck shot at the edge of the lung, one would certainly be inclined to disregard it. If, on the other hand, it were as big as one's finger nail, not completely calcified, and sharply marked off, one might consider that it was a potential source of danger.

One would also have to take into consideration the type of industrial population, the duration of time with which the suspect might have had contact with tubercle bacilli and the consequent amount of immunity that this group, or the particular individual might be likely to exhibit. This is a difficult point and one that one wouldn't care to answer categorically, I admit.

MR. WHITE: In the northern section of the state where our iron ore mines are located, we depend very largely on Finn labor and Northern European, Scandinavian. We are informed that those races have a much greater tendency to tuberculosis than the ordinary American citizen. How can we apply your rule with reference to that race?

DR. GARDNER: It is a difficult problem. I think you would have to decide the individual case on its merits and on the evidence submitted from the tuberculin reaction and from X-ray examinations. I would not want to give you a blanket rule that could be applied to every case. We know that Finns do die of tuberculosis when they contract it. Their tendency is a bad one, rather than a good one. How to evaluate the factors of living conditions against those of immunity created by previous childhood infections is the thing that would offer difficulties. Perhaps Dr. Warfield could help us in the discussion of this practical point.

MR. WHITE: We are dependent upon local population for our men. We cannot import them from other sections of the United States. It is impractical. Hence our industry has that burden. We have no way of controlling the living conditions of those men once they go home. We can't say that they must live in sanitary conditions. We know that the Finns are very much inclined not to live in sanitary conditions, and yet under state laws we have no control over it. So that it presents a practical problem as to whether there are certain limits in industries of various kinds and I refer now particularly to our own, it may be in others, beyond which we can't go. We may do everything that medical science and resultant rules of the Industrial Commission devolved and yet there is a hazard which we cannot eliminate but which is incident to that work. Now, that being so, the question is whether ultimately, I won't

say today, the thought must come that having done all we could, we should not then be penalized for that which we cannot overcome, especially that racial tendency.

DR. GARDNER: In the case of your Finns, they are a race who have been long in contact with tuberculosis. They have less resistance, apparently, than some other races, for reasons which are not obvious. I think that you would be perfectly safe in accepting a man who has a healed childhood tuberculous infection demonstrable by X-ray who still reacted weakly to tuberculin, who was in other respects physically fit. If there were any suspicion about this individual's X-ray films, if there were any hesitation in your mind that his latent tuberculosis was on the verge of activity, he should be eliminated from the group. Furthermore, I feel that any contacts in the home should be removed from your community, if possible. I realize that this might be very difficult, but if you find that there are people in your community with open tuberculosis they might readily infect a silicotic miner and the silicotic miner then becomes a carrier of disease in the mine and a distributor of it to other silicotic individuals. For your own protection it becomes your obligation to hunt down every possible source of tuberculous infection that you can find and eliminate it from contact with your workers.

MR. WILCOX: We have no law on that subject.

DR. GARDNER: You have no law, but I am simply outlining a situation which might exist. The solution of the problem is difficult, I realize, but it must be solved subject to local conditions.

MR. DOE: Dr. Gardner, do you feel if a man reacted to tuberculin and the X-ray showed he was in pretty good condition and he was rejected for that employment, might that constitute black-listing in your judgment?

DR. GARDNER: They do it in South Africa, because they feel that it is money in their pockets to do so. They have the situation in much better control than we can possibly expect to have it here. They have one industry and the whole compensation scheme has been built up around that industry. The whole thing is concentrated in one place.

They have the Miner's Phthisis Bureau which has absolute authority to make examination previous to and during the period of occupation. If the Bureau rejects an applicant for employment he is excluded; if it diagnoses silicosis during his employment he is warned and may leave and accept compensation in a lump sum award. He can go to a non-scheduled mine, but he is not eligible to compensation then.

MR. DOE: Suppose you had a man with childhood infection which we will say was a border line whether he should or should not be employed, and he was rejected for employment in a foundry, would it be your view that that would be black-listing or entirely justifiable on a medical ground?

DR. GARDNER: If there is sufficient evidence presented from the X-ray pictures and from a tuberculin reaction that this was a potential source of danger to the man, I should think it would be perfectly justifiable to eliminate him.

DR. WILLIS: I think it is pretty near mandatory to do so. It would perhaps introduce a menace to the men and to the industry. I think I would have to have more evidence than the X-ray and tuberculin reaction would give. I would want a careful physical survey of the patient and a careful evidence of the history. There are so many factors, that it impresses me that every industry and perhaps every individual who comes up for review or for employment or for retirement because of illness; every individual has to be taken as an entity and that, it seems to me, is the biggest problem here, both for industry to prevent more and more infection and for the individual to be protected. I think it is the most careful evaluation of the individual who is immediately concerned and it will go farther toward eliminating a lot of tuberculosis in that particular sort of industry in the future than any other one way I know of.

DR. GARDNER: I do also.

MR. DOE: Which is of greater importance from the standpoint of the man, is it more important for him that he should be excluded for his fellow workman as in contradistinction from his employer?

DR. GARDNER: I think it is more important for his fellow employe. He takes a chance with his own life. The chances are about ninety to one that his disease will get him if he has tuberculosis and silicosis. If you had several hundred employes working in silica dust and you should allow a man with open tuberculosis or the one who develops open tuberculosis, work with them you would imperil not only the one man, but all these others. No, I would say for the protection of the many that the one man should be excluded.

MR. KUECHLE: Wouldn't that mean in a community of diversified industry the better course of procedure would be not to accept the suspicious type?

DR. GARDNER: That is what they have done in South Africa. They have excluded people who Dr. Russell and I felt might perfectly well be allowed to enter the mine and expose themselves. They showed very slight changes in their X-ray, more fibrosis than usual, as we designated it, or perhaps a latent or childhood focus or a widening in the mediastinum connective tissue. But they could afford to be strict for they had plenty of laborers and their source of supply was not limited. Nevertheless it surprised us to find out how very rigid they were in the enforcement of their requirements.

MR. DOE: If that was to be effective in a given community, what would you advise, a central clearing house for information of that kind?

DR. GARDNER: I believe very firmly that such a program is only workable with a central clearing house in charge of qualified persons and with authority to make rulings from which there can be no appeal. Without such an authoritative board the plan cannot work, because even medical opinions have been known to differ. The crux of the matter, of course, is to find the men properly trained and qualified to act on such a board.

MR. TARRELL: Dr. Gardner, does your recommendation to exclude a man from being employed who has evidence of a childhood infection of tuberculosis apply to all dusty trades, or are you limiting that to any particular type of dust?

DR. GARDNER: No, I certainly would not exclude the man that might work in pure alundum dust. We have not sufficient knowledge about all types of dust and their reaction by any means; the surface of this subject has only been scratched.

MR. TARRELL: What do you say about such a man working in an exposure of aluminum oxide?

DR. GARDNER: As far as I know today—no, I don't know anything about the effect of aluminum oxide upon tuberculosis. I know that aluminum oxide will not produce significant damage to the normal tissues, but I do not know whether it will alter the reaction to tubercle bacilli. I do not think it will, but I have no proof. I have not yet had an opportunity to study its effects in this connection. There is a good deal of clinical information on the subject, but there have been no autopsies reported.

MR. TARRELL: Have you made any study of the effect on the respiratory tract of aluminum oxide?

DR. GARDNER: We have not done inhalation experiments. We have made some intravenous injections of aluminum oxide, sections of which I will show you in a few minutes.

DR. G. L. BELLIS: Is there any limitation to the amount of aluminum oxide or to other so-called inocuous dusts that can be cared for by lung tissue; does the amount of dust and the length of time in which the worker has been exposed have any effect on lung tissue, in relation to causation of disease, either mechanical or chemical?

DR. GARDNER: Within certain limits it has been held a small concentration of silica dust inhaled over a long period of time, would produce the same effect that a higher concentration over a shorter period of time. But this statement has to be qualified, because with smaller concentrations of dust the eliminating mechanism is more effective. It is not thrown out of order so rapidly and a person can handle very appreciable amounts of dust for a long period of time.

DR. BELLIS: My question had to do principally with dusts claimed by some to be inocuous dusts, for instance,

aluminum oxide dust. Is there any limit to the amount of dust that the lung will take care of without damage to itself?

DR. GARDNER: I think one probably could create such a heavy concentration of dust that an individual would suffocate in it, but of course, such a condition would be unusual.

MR. TARRELL: Is the process of elimination the same with non-silicotic dusts as with the silica?

DR. GARDNER: No, probably not within wide limits. If one inhaled something less than a suffocating concentration of dust it would tend to pile up more rapidly in the lung and would throw more and more air spaces out of commission, not by fibrosis, but simply mechanical plugging. The subject would be apt to develop a chronic bronchitis or possibly a pneumonia. But I do not think that with very high concentrations of aluminum oxide pulmonary fibrosis would develop.

DR. WILLIS: You have a more or less analogous situation with the cement industry, where it is quite well known that huge clouds of cement dust running into hundred millions of particles appear not to do any harm at all to the individual inhaling them. I would like to give a case or two that have come under my observation just recently, of men who have been running emery wheels. Of course, that is the source, probably a source of aluminum oxide, emery dust. It is quite important whether those wheels are artificial or natural stone and of considerable importance to know the attendant substances. In Detroit there have been two young negro men I happen to know about who have died, one after eight years of dusty work in running an emery wheel and one after six years' exposure to emery dust. One of them had symptoms for a little over a year and one of them three or four months. There is no proof at all that emery dust, aluminum oxide, was what had caused his silicosis. He obviously had pneumoconiosis, but there wasn't any proof that dust had done it, because there was no way of finding out the environment that had surrounded his occupation and that had been in attendance in his early occupational life. It seems to me over and over

MR. TARRELL: Now, assume that with the aluminum oxide dust you have a certain percentage of metal. dust, iron dust, how would that effect your answer as to the susceptibility?

MR. CUMMINGS: We have very little evidence of reaction produced by iron dust itself. More significant would be the small percentage of silica over a long time. I feel that the best criterion for the action of any such dust would be the evidence of pulmonary fibrosis on a good roentgenogram.

MR. TARRELL: What do you mean by saying there is no evidence as to the effect of iron upon the development of tubercle bacilli?

MR. CUMMINGS: I mean by that metallic iron.

MR. TARRELL: Isn't it a fact developed by experiments in Europe that iron favors the growth of tubercle bacilli?

MR. CUMMINGS: Yes, there have been some recent reports on that.

MR. TARRELL: They all indicate that iron favors the development of the tubercle, does it not?

MR. CUMMINGS: Yes, with an experimental infection in experimental animals any foreign body, with its resultant irritation, may tend to make the infection more active. However, the conditions of experimental infection are quite different than those in man. I would rather have Dr. Gardner answer that question.

MR. TARRELL: You are familiar with the experiments conducted by Dr. Calmette?

MR. CUMMINGS: Yes.

MR. TARRELL: Director of Pasteur Institute?

MR. CUMMINGS: Yes.

MR. TARRELL: What did his experiment disclose?

MR. CUMMINGS: His experimental work has been quite extensive.

MR. TARRELL: I mean his experiment work as to iron favoring growth of the tubercle bacilli.

MR. CUMMINGS: I think you refer to the fact that iron salts in small concentration were added to the medium on which tubercle bacilli were grown. Long, in this country has also made the same observation. Small percentages of iron salts seem to favor growth of tubercle bacilli in test tubes. However, conditions of growth in test tubes and those present in the body are not to be compared. For instance, the experiments to which you refer show that a small amount of an iron salt will increase the rate of growth of tubercle bacilli in the test tube, whereas double or treble that quantity of iron salt will definitely inhibit growth. There is then a very definite concentration which seems to be beneficial for the growth of organisms.

MR. TARRELL: I think that is all.

MR. DOE: Mr. Cummings, may I ask you one more question? In your previous answers with regard to the number of million particles you said in answer to one question that that was under ten microns in size and over what size? In the Barre, Vermont survey what did they use in that regard?

MR. CUMMINGS: The Barre survey was made with the technique used by the Public Health Service and as I pointed out yesterday it is our impression that for all intents and purposes particles above two microns and below ten were included. From a physical and mathematical standpoint 2 micron particles are about the smallest size visible with that technique. Consequently, the measure of dust concentration used included particles within that range, from two to ten microns.

MR. DOE: Everybody knows that there could be more particles per cubic foot of air. Where there are particles you can see there must be particles you can't see.

MR. CUMMINGS: That is right.

MR. DOE: So that in giving your estimate as to what is a safe atmosphere over a long period of time you mean five million particles more than two microns in size and less than ten?

MR. CUMMINGS: Yes, on determinations made by a technique similar to that used in the Barre survey.

MR. DOE: And if another technique were made by which the smaller particles were also counted, as I understood your graphs yesterday, that would approximately double the number of particles?

MR. CUMMINGS: It might increase it by more than double the number given. However, that would be taken into consideration by the use of a conversion factor. It would not increase the actual hazard.

MR. DOE: The hazard would be the same if you counted by the other method?

MR. CUMMINGS: We would simply be using a different ruler with finer divisions.

MR. DOE: That is all.

MR. KUECHLE: Is there a different correlation between particles discovered by the light field method and the dark field method?

MR. CUMMINGS: At the last conference of the people involved in dust counting it was agreed that we would obtain conversion factors for changing over from the present technique to the new one if it were justified.

MR. KUECHLE: Have there been any definite figures determined?

MR. CUMMINGS: There have been.

MR. KUECHLE: What is the correlation as near as you know today?

MR. CUMMINGS: I would rather have Bloomfield answer that.

MR. KUECHLE: I understood from Dr. Gardner's statements yesterday morning that particles above ten microns probably do not get into the peripheral air fields of the lungs.

DR. GARDNER: With the exception of the long fibres of asbestos which may go down endways.

MR. KUECHLE: Would you also say that particles below three microns were infrequently discernible in those air cells?

DR. GARDNER: My discussion on that subject was one with reference to the advisability of revising our counting methods. It has been granted that while these small particles might easily penetrate into the terminal air spaces of the lung, some observers believe that ciliary action would tend to carry them out again very quickly so that they would not remain and I said we hoped to carry on experiments to prove whether this were true or not. If it were true that these very small particles which have been shown to be so extremely active were retained in the lung, then it would be necessary to revise our technique to measure the hazard so that we may determine how many of these very small ones were present in the industrial atmosphere.

MR. KUECHLE: Would that then mean, Doctor, that at the present time when you talk about dust concentration at any point that you always designate the method that was employed in the dust counting so that we may definitely limit the size of the particles between maximum and minimum?

DR. GARDNER: Of course in most dust investigations at the present time the bright-field illumination is being employed, and it is to be assumed that up to the present time at least, all dust counts have been made on this basis. In our experimental work we would certainly report that we have used the dark-field.

MR. TARRELL: In the article published in the American Review of Tuberculosis in 1922 and 1923 you reported an experiment with granite and marble dust being injected in guinea pigs and then after exposure to the granite and marble dust for a year you infected these guinea pigs with tubercles and reported what that disclosed? Do you remember what that was?

DR. GARDNER: That paper to which you refer was one entitled "Reactivation"?

MR. TARRELL: Yes.

DR. GARDNER: In that case you have the cart before the horse. This reactivation experiment was one in which infection was given first and not allowed to heal and subsequently at intervals after the infection, dust exposure

was commenced. In that experiment we discovered that marble had practically no effect in reactivating a latent tuberculous infection but that granite was much more potent and I don't recall the percentage of animals but a good number of them did reactivate their tuberculosis and caused it to spread.

MR. TARRELL: Well, as I remember your article, a certain percentage of animals were not given the dust and you quoted your figures in non-dusted animals as fifteen over different intervals. It remained the same and after giving the tubercles to these dusted animals it increased. The first chart in granite was 51; the second chart 94, and the third chart 102.

DR. GARDNER: You refer to another paper. I have forgotten the year of its publication. In this experiment the infection was given today, let us say, and dust exposure was commenced immediately so as to allow the dust to act during the period of development of tuberculosis. Under these circumstances we found that the number of tubercles developing was proportionate to the irritating properties of the dust itself. There was a slight increase above the normal controls in the series inhaling marble dust. There was a greater increase in the series inhaling granite dust.

MR. TARRELL: There was an increase from 15 to 62 over the normal control to the marble administered?

DR. GARDNER: Yes.

MR. TARRELL: And 15 to 102 in the granite?

DR. GARDNER: Yes.

MR. TARRELL: That is all.

DR. H. S. WILLIS, Detroit Health Dept., Northville, Mich.

There are quite a number of items on the agenda on which some of us will talk at length, and others which other members of the group will discuss at some length. Dr. Gardner has already given you a very clear exposition of the anatomical relationships underlying the development of pneumoconiosis and I would be inclined to pass over that entirely because he has covered it thoroughly.

It is well to bear in mind the fact that a normal, healthy

upper respiratory tract is very efficient in withholding dust from the lung, and the best evidence that we have, which is not as yet absolute, would have you believe that perhaps not over 25% of the dust in the air reaches the alveoli of the lungs. That is so when the defensive mechanism in the upper tract is intact. This may be seriously damaged, of course, with anatomical defects or with mouth breathing, or it may be overburdened with huge amounts of dust. As to the pathology itself, Dr. Gardner has so thoroughly covered the question that there isn't much to add except to emphasize one or two points further.

He has told you the parts of the lung that are ordinarily involved: namely, that pneumoconiosis is primarily a disease of the lymphatic apparatus of the lung; that it concerns itself with reaction in the lymphatics and in the lymphoid tissue, masses of which are distributed along the arteries, veins, and bronchi, particularly at the points where those structures divide. He has told you also that dust tends to accumulate in the lymph nodes at the root of the lung, the tracheobronchial lymph nodes, and that, in general, after the lymphatic apparatus is more or less involved, the disease manifests itself in the more central portions of both lungs, usually a little more on the right (according to many observers, particularly roentgenologists). The disease manifests itself bilaterally in all portions of the lung except the extreme apices and the very bases, and there are many instances in which the entire lung is involved. He also mentioned the fact that early in the process of exposure the pleura and peripheral parts of the lung are involved and that there is some evidence to point to an anatomical basis for the localization of the dust in the pleura.

When one takes a dust-containing lung out of the body and looks at it, one from either an experimental animal or a human being, he will notice that the dust is arranged in a rather definite manner, a rather definite pattern over the pleural surface. There may be elongated lines and dots or a series of dots and lines. There are polygonal areas of dust under the pleura, that correspond to the position of the lymphatics as they course through the pleura and it corresponds also to the outline of the finest division (of the

anatomical unit) of the lung, according to Miller of Wisconsin. This unit or lobule is a structure with a border of lymphatics as its limit. Lobules are thus set off from one another which have bronchioli near their center that pass peripheralward from the deeper portions of the lung. The tendency for dust to be deposited in these limiting areas is based possibly on two anatomical facts. The first is the fact that the bronchiolus sends its divisions in all directions, and that those which pass toward the edge of the lobule meet similar ones from neighboring bronchioli in neighboring lobules. Any dust in the air in these passages is likely to become trapped in these terminals. A second factor which may contribute to this deposition of dust lies in the existence of masses of lymphoid tissue which normally lie in these regions and which act as it filters.

I would like to have you bear this in mind too, that the physiological and anatomical mechanism which localizes dust in these various areas of the lung is the mechanism which transports and localizes tubercle bacilli as well, so that tubercle bacilli are inclined to be carried and deposited in the same anatomical areas in the lung as is dust.

Two points about the pathological reaction! First, as Dr. Gardner has amply emphasized, the reaction is essentially a *fibrosis*, and there has recently been a number of contributions in the literature which point out the fact that, if there is an alkali in association with silica dust, the results are likely to be very devastating; that an alkali inhaled with silica will stimulate the formation of fibrosis to a very remarkable degree.

The second point about the pathology which we ought to remember is that it is a *progressive* affair. A good many of you are familiar with the report of Watkins-Pitchford from the South African mines in which he gives an account of miners who left the mines for service during the war. When they left the mines, they had no signs of silicosis. They were in the war for four years and came back to the mines when a number of them had very definite roentgenological evidence of silicosis. In other words, silica was there and in the intervening four year period it had stimulated the formation of recognizable reaction in the lung. Another evidence of its progressiveness has been

brought out by Dr. Russell and co-workers in their study of granite workers in which he analyzed the records of quarrymen who had left the granite industry. Of that group the average age at death was something like 51 years. The average duration of occupation in the mines or quarries had been about twenty-two years and the average duration of time between discontinuance of occupation and death was something over eight years, but the whole group had silicosis and nearly all of them tuberculosis as well; so, although the worker is removed from his dusty occupation, the tendency of the disease is to very definitely be progressive.

On the contrary there is a curious fact in connection with this observation. Jarvis observed that, during a prolonged strike among granite workers, there was apparently a recession of pneumoconiosis. Also in South Africa physicians assume that the reason the colored workers (natives) do not develop as much pneumoconiosis or silicosis, and do not develop it as readily as the white workers do, is because they work intermittently. I do not know how one can reconcile those two facts; on the one hand progressive disease in a large number of instances goes on down to death, although exposure has ceased; on the other, intermittency of employment appears to stay the disease. It is a puzzle but I think it should be mentioned.

The next item on the agenda is a question of terminology. When Zenker used the term pneumoconiosis he derived it from the Greek and aimed it to be a term to include any effect of dust on the lungs. Since then it has been an inclusive term and the terms silicosis, anthracosis, siderosis and so on have been used properly to designate a certain sort of pneumoconiosis.

When one comes to talk about tuberculosis and pneumoconiosis, one is simply floored because one hardly knows where to begin. It is a large subject and one which may be discussed from a great number of angles. Think of tuberculosis first as a very protean disease which may attack all classes or ages, and all tissues of the body. It may be acute or chronic. It is difficult to catalogue any disease which is as protean in its nature. It is a disease so full of clinical and pathological irregularities that exact placing of it may be an exceedingly difficult matter.

However, there are three factors that we might think of in respect to relation between pneumoconiosis and tuberculosis and with respect to the effect which pneumoconiosis has on this infectious disease. In the first place, the well recognized *incidence of tuberculosis in silicotics;* it is high; the number of people with silicosis who die and at autopsy (not just on the death certificate, but at autopsy), show tuberculosis is sufficient evidence to link the diseases causally and very definitely. Yet much of the data that we present from time to time on the death rate of tuberculosis and pneumoconiosis is open to serious criticism, because it is based on death reports. A very good example of that is seen in some work that was done ten years ago in Connecticut relative to the death rate of axe grinders. It was reported that 1900 per hundred thousand employed died early of tuberculosis. Drury, who made that report made a perfectly plain statement that his diagnosis of tuberculosis was taken from death reports only and that it included quite a hodge-podge; anybody dying of phthisis, anybody dying of what was known as consumption, anybody who died of what was known as "miner's phthisis" or "grinder's consumption"; anybody who died of tuberculosis; he took the whole group and called them tuberculosis, and in that group there was not one single reported death as being due to pneumoconiosis alone. I think that gives you a very good reason for being suspicious of such data when we know that pneumoconiosis must alone produce many deaths, and many of which are attributed to tuberculosis alone. Such work emphasizes very clearly the high incidence of mortality from pulmonary disease among groups who work in dusty trades or in axe grinding, but it does not prove that those people all have died of tuberculosis.

However, the high incidence of tuberculosis proved to be associated with pneumoconiosis is sufficient to indicate that the infection comes very frequently as a complication.

Experimental data proves the same thing. The data which the sections and lantern slides Dr. Gardner and Mr. Cummings showed yesterday pointed out how common or easy it is for tuberculosis to undergo exacerbation or to

be increased in amount in the lungs that have been made silicotic.

In Toronto there is some work going on which has not yet been reported, to the effect that *the addition of silica to media* in which tubercle bacilli are grown produces a definite increase in the growth of these bacilli. The same media without silica grows tubercle bacilli at a given rate and when silica has been added the growth is enhanced. That, of course, is again a test tube experiment and is not to be carried over bodily to clinical observation, but it is a fact worth mentioning.

A VOICE: You mentioned silica being added to the test tube?

DR. WILLIS: Silica. I understand that it is crystalline silica although I have no specific knowledge of this.

A VOICE: Silica increases the growth?

DR. WILLIS: Yes. Many other substances do the same thing; iron does it in certain concentration, for instance.

Do the dusts which fail to produce demonstrable pneumoconiosis produce an effect on tuberculosis? That is a question of great importance. It is a question to which there is no absolute answer. In general one would say *no* to this question. At least there is no proof that dusts which fail to produce pneumoconiosis or fail to produce fibrosis of the lung produce any susceptibility to tuberculosis. The only two types of dusts, so far as we know, that do produce pneumoconiosis are crystalline silica and asbestos, which latter is a silicate. Aside from those two dusts, there is no proof that any one predisposes to the development of tuberculosis. Certainly in many dusty industries the death rate from tuberculosis is not appreciably increased over that for occupied persons of comparable ages. I should like to mention one or two experiments that were undertaken in this connection, with inhalation of dusts which do not produce pneumoconiosis.

Guinea pigs were exposed for something over a year to soft coal dust in great concentration and then were injected with human tubercle bacilli which had a known virulence; they were infected along with a group of animals which had

had no dust. There was no material difference in the amount of tuberculosis which developed in those two groups.

Then for something over three years we exposed a large group of guinea pigs to what was essentially silicon carbide (one of the dusts from the Norton Company). We exposed them to extraordinary heavy clouds, often containing over one hundred million particles per cubic foot, often in clouds so thick that you could hardly see the animals as they sat at the bottom of the apparatus. We exposed them for three years for four or five days a week and six to eight hours a day and then infected them with tubercle bacilli of known virulence, along with normal, non-dusted animals, and in the dusted animals there was no increase in tuberculosis. The degree of tuberculosis in the two groups was quite the same. It should also be said that, in both these experiments, we were unable to demonstrate any evidence that fibrosis, any evidence that pneumoconiosis had been produced by those two dusts. I mention them because I think you would like to lay them, in your thinking, alongside these experiments of Dr. Gardner, with dusts which do produce a pneumoconiosis and have a deleterious effect on tuberculosis.

Now, in thinking of the effect of dust on the lungs, of the formation of silicosis, we have at least three factors to consider. One, of course, is the *percentage of silica in the dust* and another is the *amount of the dust*. It is obvious that, if silica is only one or two or three per cent but the dust is of sufficient concentration, workers may possibly acquire enough silica to do damage. In practical experience, however, it hasn't worked out that way in the cement industry where free silica exists from one to five or six per cent and where the dust is in very great concentration. One needs to get no nearer than a half mile to a cement plant to realize how intensely concentrated the dust is. Those people do not develop any serious grade of pneumoconiosis and they certainly do not appear to be predisposed to tuberculosis. On the other hand, pure silica may be present in dust to the extent of 50% or more without leading to serious effect on the lungs, provided the concentration (the dust count) is low. So both those factors must obviously go together.

A third factor, namely, *adulterant dusts*, has been men-

tioned, not in this conference, but quite a number of times elsewhere. Haldane brought it up fifteen years ago and Mavrogordato has played it up as has Heffernan of England. That is the question of the effect of inhalation of silica to which some other dust has been added. Clay, or in some instances, coal dust is said to either cause the silica to remain in place in the alveoli and not be transported to the lymphatic system very readily or to facilitate its rapid elimination. Theoretically that is a very important consideration. Of how much practical value this procedure might be remains undetermined as yet. Certain facts indicate its inutility. The only industry in which it has really been put to test over a long period has been that of the potters. There is a great deal of clay of many sorts in potter's dust and there is also a varying amount of silica dust, but potters develop pneumoconiosis slowly and progressively, irrespective of the presence of clay dust. The question of these adulterant dusts is one that should be thought of and worked on a great deal more than has been done up to the present time.

The next point in the outline is the effect of dusts on non-tuberculous diseases. It is well known and admitted by everyone that the morbidity rates and mortality rates from respiratory diseases in people working in the dusty trades is higher than it is amongst occupied males of the same age groups in other industries. I hope Dr. Russell will develop that point more. His group has emphasized the fact that, amongst pneumoconotic people who subsequently develop tuberculosis, there is, for a while before the tuberculosis appears, an increase in the amount of apparently non-tuberculous pulmonary diseases. What the factors are in this increase in the morbidity and mortality rates among dust workers one doesn't know. Some of them are unanalyzable. There are certain known factors, such as low economic scale of living, particularly in sand blasting and trades in which pay is relatively low. The people live in unhygienic surroundings. On the contrary the skilled workmen in the granite fields have unusually good wages and good sanitary surroundings and living conditions. Among coal miners excessive drunkenness exists. That may or may not play a part. It certainly might easily contrib-

ute to the incidence of pneumonia which is high. These are still questions on which we cannot give any very definite information, but people in dusty trades do have a high incidence of pulmonary disease; that is granted.

What are the effects of dusts which do not produce pneumoconiosis—the effect on tuberculosis and other diseases? Well, those are uncertain factors, uncertain quantities. It has been claimed, for instance, that, in a group of tobacco workers in the Mannheim District in Germany, the incidence of tuberculosis is very high; this has been attributed to tobacco dust, but investigation reveals essentially these facts—that people entering that particular industry are physically weak people, largely because the work is light and the pay small, and the vigorous, healthy people seek employment which gives them a better income than this particular occupation would. So the group that elects this particular industry are physically below par and this, coupled with the fact that the income is low, has been given as an explanation of the high tuberculosis death rate. Those are the facts that have been evaluated as a cause of the higher death rates from tuberculosis. There is a good deal of asthma amongst the workers, coal miners at times, and especially among workers in the vegetable dust, as well as a high incidence of tumor in certain German mining districts.

This brings up another question which is of some importance, because on at least three occasions it has been reported that acute disease in large numbers of workers has followed exposure to vegetable dust. It has been shown in each instance that fungi or other infectious material has been carried with the dust and that the inhalation of quantities of living infectious material has been responsible for the disease in question.

There has recently been a very interesting and significant development of a new disease amongst workers in the woods of Northern Michigan. It represents a new industrial hazard which is a product of the depression. Many logs at the logging camps have been allowed to lie about for two or three years, and, as the men have worked the old logs, they have found a very fine, almost impalpable powder of black-brown color lying in huge amounts between the bark and the body of the tree. This arises in great clouds when

the bark is removed from the log or when the log is sawed. Quite a number of those people have developed evidence of pulmonary disease with physical and roentgenological signs that suggested tuberculosis of an acute sort. Their symptoms, however, were predominantly those of asthma and bronchitis. Those men, nearly fifty of them, have been put into sanatoria, and all of them have got well. Now, the upshot of the whole thing has been that examination of the sputum has failed to reveal tubercle bacilli but has revealed a fungus. Examination of this fine powder has found it to consist solely of pure culture of spores of this parasite. In the ordinary process of the industry the logs would not lie around long enough for this fungus to grow, but as they have not been used, the bug has an opportunity to grow and this product, this fungus, this spore, produces in the lung a very definite pulmonary disease, which, fortunately, clears up promptly after removal of the cause. It has been reported by Dr. John Towey, and it has been suggested that it be named Towey's disease. It points out this fact, that, in association with vegetable dusts, there may well be infectious materials, which may be responsible for disease.

The next point on the agenda is roentgenological evidence of silicosis. I do not pose as an expert in roentgenology and, with a few brief remarks I am going to pass that over to Dr. Russell. The classification of silicosis into groups or stages, one, two and three, has been protested against vigorously by a number of roentgenologists, particularly by Pancoast and Pendergrass. They have felt that, inasmuch as the different industries are associated with different types of pneumoconiosis, those stages do not apply. The ordinary grouping that has been described has been that in association with rock dust (quartz). The first evidence is simply a haze (especially in asbestos is) throughout a fairly large area of the pulmonary field, usually bilateral, and this in a film which may show (1) widening of the mediastinum, (2) accentuation of the linear markings, (3) fine fibrous lines, and (4) fine shadows in the periphery of the lung. As time goes on, a degree of nodular predominance appears so that these little nodules (lymphatic nodules) become enlarged and appear on the film as opaque shadows which have been described as snowstorm appear-

ances. A little later there is fusion of many of the snowstorm like nodules and an increase in the amount of diffuse fibrosis, both of which may lead to consolidated areas that are sometimes quite large. These definite opacities appear in the film. Then finally when tuberculosis supervenes masses of consolidation become more conspicuous. It is quite difficult to separate a patchy consolidation of simple pneumoconiosis from that in which tuberculosis has supervened. It should be borne in mind that silica dust in its various forms leads to differing roentgenological appearances. In granite cutters, for instance, thickening of the trunks, widening of mediastinal shadows, increasing density of shadows in the parenchyma all appear without the nodular, snowstorm shadows.

I am not going to say more about roentgenological features because Dr. Russell will speak to you shortly and will undoubtedly discuss this question. One might raise a question in connection with roentgenological features of silicosis. Can one make a diagnosis of pneumoconiosis in the absence of roentgenological signs? This question comes up to physicians quite often and it is a question that a physician is really in difficulty to answer. I should say "no" to that question because, unless there is roentgenological evidence of pneumoconiosis, there is absence of the characteristic symptoms of the disease. This question has been up in South Africa and committees have made certain requirements for the diagnosis of silicosis *at autopsy*. In South Africa there must be a macroscopic nodule, at least *one* nodule, in every four square cms. of lung tissue. This may vary, depending upon whether the lung is distended or contracted (fibrotic), but it indicates at least that there is an effort being made to require definite evidence for the diagnosis. If they do that in South Africa and make such a requirement for the autopsy diagnosis, it seems logical to assume that one cannot make the diagnosis in life unless some definite criteria avail. As to the differential diagnosis between pneumoconiosis and tuberculosis, there are also certain difficulties. Physical examination is important. History is important. I am not going to discuss it because Dr. Russell will consider it, I am sure. Physical examination is of the greatest importance in evaluating the status of the

patient. Occasionally miliary tuberculosis of the lungs is diagnosed as pneumoconiosis because throughout the pulmonary field miliary tubercles are scattered and these appear as innumerable little dots and points or opacities. Miliary tuberculosis or pulmonary carcinomatosis (a very rare disease) must be carefully differentiated from pneumoconiosis. I have a case in mind just now that may illustrate that fact. A young man of 21 had worked two years in emery dust, polishing headlights in one of the Ford plants. He reported that the dust was so thick that he could not see his neighbor who stood two or three feet away. He worked in that dust daily for two years before he had a hemorrhage, which is not common in pneumoconiosis. He came for examination which was equivocal. X-ray showed a snow-storm appearance that looked like very extensive pneumoconiosis. His sputum continued to be bloody (which is not like pneumoconiosis), and finally tubercle bacilli were discovered in the sputum. He developed a cavity in the lung, was treated by pneumothorax, and in the course of a year and a half his so-called silicosis disappeared from both lungs.

We all know the symptoms and we know that, when tuberculosis supervenes in the course of pneumoconiosis the patient begins to feel ill. Heretofore, he had not felt very badly. A little shortness of breath and a little cough were present, but he had had very few symptoms. But now tuberculosis begins, and he is very likely to lose his appetite; he is very likely to cough and expectorate and to lose weight, and he feels quite weak and lethargic. Such symptoms appear eventually in the late stages of uncomplicated pneumoconiosis, particularly when the circulation and the heart begin to lag, but only in full blown cases. The physical signs help a good deal. When tuberculosis occurs there are rales in the chest, whereas the chest is almost always dry in simple pneumoconiosis. A very important factor in the differential diagnosis is examination of the sputum. Sputum should be looked at over and over and over again, if there is any suspicion that tuberculosis may be present. It is only in that way that the diagnosis may be made in many instances.

There are a good many more points in this outline. Noth-

ing has been said about prevention, but Dr. Russell and Mr. Bloomfield are in a much better position to discuss those features than I am.

MR. WILCOX: Have you any view on the matter of concentration of dust? I speak of it because we are developing a dust and fumes code in Wisconsin and we have a number of our members here.

DR. WILLIS: In general it has been shown that a concentration of 200 to 250 particles per cc. or five to eight million particles per cubic foot are within limits of safety. That means particles below ten microns in diameter.

MR. DOE: What percentage of silica?

DR. WILLIS: In a question like that the important thing is the type of dust. We know perfectly well that one hundred million particles or possibly two hundred million particles of cement dust does little or no harm, but we know that one-tenth or one-twentieth of that amount of silica may do considerable harm. So in any discussion of the limitation of numbers of particles, we must obviously consider the type of dust as well. I think it is safe to say that five million particles of silica under ten microns fall within the realm of safety in any practical circumstances, even if the silica be present in 35 or even 50% strength.

MR. DOE: If it is agreeable to you and to Dr. Willis, I would prefer not to ask any more questions until Dr. Russell has talked, because many of the things I might ask him would be quite fully covered.

MR. WILCOX: Would that be agreeable to you, Mr. Tarrell?

MR. TARRELL: Yes, surely.

MR. WILCOX: It is Dr. Russell's wish that Mr. Bloomfield take his part on the program before Dr. Russell discusses this matter. If that is agreeable, we will have Mr. Bloomfield at this time.

MR. J. J. BLOOMFIELD, *Sanitary Engineer*, United States Public Health Service: MR. CHAIRMAN, I should like to discuss those properties of a given dust which determine its

capacity to produce pulmonary pathology; that is, the nature of the dust, or its chemical and mineralogical composition, the particle size and finally the quantity of the dust dispersed in the atmosphere.

Dr. Gardner pointed out yesterday, in discussing his work in experimental pathology, that apparently there are no two dusts which exert the same influence on the lungs. Mr. Cummings gave you an excellent discussion of the nature of different types of dust, and I, also, should like to comment briefly on this same point.

NATURE OF DUST

Research on the problem of industrial dust inhalation has indicated that so far as their fibrosis-producing qualities are concerned, dusts may be divided into three groups: (1), those composed completely of combined silica, that is, silicates, such as pure asbestos; (2), those containing free silica in the crystalline form known as quartz, (granite contains approximately 35 per cent of quartz); and lastly, (3), dusts containing free silica in a non-crystalline form such as diatomaceous earth. It has also been observed that the harmfulness of a quartz-containing dust is in direct proportion to its quartz content.

However, even today we find the terms quartz, silica and free silica used interchangeably. Perhaps it may not be amiss to define these terms briefly at this time. Silica is the name given to the oxide of silicon (SiO_2). A distinction is made between free silica and combined silica. Free silica is the term used when the silica occurs in the form of a definite compound having the formula SiO_2. Generally free silica when thus used means quartz, but as a matter of fact there are seven minerals that are composed of free silica. For example, in addition to quartz there are other free silicas now finding industrial use, such as tripoli, (used as a facing powder for molds in foundries), opal, an amorphous hydrated variety of silica occurring abundantly in diatomaceous earth, and other forms not quite as abundant in nature as quartz.

Combined silica is the term given to silica that occurs in minerals in chemically combined form, being united

with certain bases, such as soda (Na_2O), lime (CaO), and a number of others; in short, it is the silica in silicates. According to long-established convention, the chemist reports the silicon present in rocks and minerals as silica; he makes no distinction between free silica and combined silica, even though both be present.

Let me illustrate these principles to you by means of granite. The average granite is an aggregate made up chiefly of three minerals in the following proportions; feldspar, 60 per cent; quartz, 30 per cent; and mica, 15 per cent. *Chemical analysis shows that this average granite* contains 70 per cent of silica. Of this 70 per cent, 30 per cent is present as quartz (free silica) and the other 40 per cent is present as combined silica, being locked up in chemical combination in the other minerals that make up the granite.

From this illustration it is apparent that we should be more explicit in our terminology when referring to the nature of a dust. If a dust contains quartz, and we are referring to this mineral, then we should call it quartz and not just silica or free silica. As I have just pointed out to you, there are other free silicas in existence that are today of industrial use, and these other free silicas differ in their physical properties from quartz and may possibly differ from quartz in their action on the lungs of workers exposed to the inhalation of these dusts.

Perhaps I can emphasize the importance of the necessity for an exact knowledge of any dust under consideration by pointing out a few of the pitfalls one occasionally encounters in field work. Yesterday, Mr. Cummings, in dealing with the subject of the nature of dust, mentioned that tripoli was an amorphous variety of free silica, whereas quartz was the crystalline type of free silica. That is true of the tripoli mined in Illinois, which is composed of clusters of crypto-crystalline silica, often know as amorphous silica, whereas the Missouri deposit is composed of spongy, globular clusters of distinctly double-refracting quarts. [1. (a).] So that when one is dealing with a tripoli facing powder used in foundry practice one should make sure by a careful

(a). Numbers refer to Bibliography at end of this paper.

analysis of the dust just which form of tripoli is under consideration.

Again, in the study of the health of workers in a cement plant [2] the Public Health Service found it necessary to conduct mineralogical analyses of the dusts in the various departments of the plant because of the physical and chemical changes undergone by the various raw materials used in cement manufacture. Clay, one of these raw materials, was found to contain about 7 per cent quartz and the dust in all the departments preceding the calcining of the materials, contained about this amount of quartz. However, in the kilns, the quartz combined with the other elements in the materials that go into cement manufacture, to form complex silicates, and our dust samples obtained in the departments following the kilns showed less than one per cent of quartz. So that we had to bear in mind this dissimilarity of quartz dust exposure in analyzing our clinical and other data on the health of the cement worker.

I should like to present briefly one more example of the errors one may make in not resorting to a careful mineralogical analysis of dusts encountered in the various processes of manufacture of a single article. In 1926, Heffernan reported a study in the Journal of Industrial Hygiene, on "The Exposure to Silica Dust Without the Occurrence of Silicosis". (3). This occurred among brick makers in Derbyshire, England. The raw materials used in brick making contained, according to Heffernan, 85 to 89 per cent silica, mostly in the form of gannister sand. The finished brick was found, on rational analysis, to contain about 83 per cent silica. Now in the departments preceding the kilns there is apparently very little dust generated, due to the wet methods of working the materials. On the other hand, considerable quantities of dust are given off, in the trimming and polishing of the bricks, following the firing of the molded bricks in the kilns. Now, it is a well-known fact that when quartz or other free silica is subjected to high temperatures, such as obtain in a brick kiln, in the presence of other compounds, the quartz will combine to form silicates or at least will become inverted and change its properties. Heffernan's paper does not state whether the silica in the finished brick was free or combined, but merely

states that it contained 83 per cent silica. It may be inferred that the reason no silicosis occurred among the gannister sand brickmakers in Derbyshire was probably due to the fact that in the departments where quartz was present the dust exposure was negligible, as stated by Heffernan, and in those departments where considerable dust was generated the workers were no longer exposed to a dust containing quartz. A careful mineralogical analysis of the dust in each department, similar to the analyses conducted in our cement study, would have thrown considerable light on this puzzling situation presented by Dr. Heffernan.

Yesterday, Mr. Cummings pointed out some of the pitfalls which may be encountered in dealing with the nature of dusts and at the same time made a plea for a method of analysis which would be more accurate than either a chemical or petrographic examination. I should like to call to your attention that for the past nine years the Public Health Service has been making use of an accurate method of analysis of dusts in connection with its various dust investigations. This is a combined chemical and petrographic analysis or a so-called mineralogical analysis, best carried out by a competent geologist. Since no two dusts offer the same problem it is difficult to lay down general rules for such an analysis. Suffice it to say that each sample must first undergo a careful examination under the petrographic microscope, and in addition is further subjected to a complete chemical analysis with frequent petrographic examinations throughout the entire process. For example, only by such analysis have we found it possible to determine accurately the percentage of quartz present in quartz-containing dusts.

With your permission I should like to show a few lantern slides to illustrate some of the factors I have discussed dealing with the study of the nature of dusts. During the course of our numerous dust studies we have had many samples of dust analyzed to determine their composition. As is well known, our present knowledge concerning the harmfulness of inhaled dusts is more complete with reference to quartz-containing dusts. For this reason, and also because space does not allow the presentation of the other

minerals present in each kind of dust, the table depicted shows only the quartz content of the dusts obtained in the various industries which we have studied.

TABLE 1

PERCENTAGE OF QUARTZ PRESENT IN VARIOUS INDUSTRIAL DUSTS

	Percentage of Quartz
Rock drilling dust (bituminous coal mine)	54.0
Granite cutting dust	35.2
Rock drilling dust (anthracite coal mine)	31.0
Brass foundry dust	19.0
Dust from raw mills in cement plant	6.5
Slate mill dust (Vermont red slate)	3.0
Silverware polishing dust	1.7
Anthracite coal dust	1.5
Bituminous coal dust	1.2
Cement dust	< 1.0
Slate mill dust (Vermont green slate)	trace
Tale mill dust	none
Marble cutting dust	none

It is evident from this table that rock drilling occupations in the coal mining industry and certain occupations in the granite cutting industry and in brass foundries would be in the hazardous class, if judged solely by the proportions of quartz in the atmospheric dust. I should also like to point out that there are some slates which have been found to contain as much as 30 to 40 per cent quartz, although in the slate industry which we investigated the dust was found to contain from a trace (green slate) to 3 per cent (red slate) quartz. It is also of interest to learn that the coal dust, both in bituminous and anthracite mines, was found to contain small amounts of quartz, due to the fact that the coal exists in hard rock deposits.

To illustrate more clearly the importance of an exact knowledge of the nature of a dust in the study of the health of workers exposed to industrial dusts, I should like to discuss the next lantern slide. Realizing the importance of this problem the United States Public Health Service inaugurated, in 1923, a series of dust studies under the general direction of Assistant Surgeon General L. R. Thompson. These studies were all conducted in the same manner in order to permit as detailed a comparison as possible between the different investigations. Briefly, these meth-

ods of study may be divided into six parts, as follows: (1) examination to determine the general physical condition of the workers under observation; (2) special physical examination to determine the prevalence of specific diseases of the respiratory system and the lung pathology resulting from exposure to the particular dust hazard; (3) record of the nature and severity of the disabling illnesses; (4) analysis and detailed study of the occupational environment; (5) occupational mortality statistics relating to the specific dust; and lastly (6) autopsies. In brief, the chief value of each of these studies lies in the fact that it represents careful and detailed observations on a fairly large group of persons whose working environment was accurately determined, especially with reference to the nature and quantity of the dust exposure.

TABLE 2

SICKNESS[1] FROM RESPIRATORY CONDITIONS IN SIX DUSTY TRADES

Diagnosis	Annual Rate per 100 Years of Observation						Number of Cases					
	Granite	Cement	Hard Coal	Soft Coal	Cotton	Silver Polishing[2]	Granite	Cement	Hard Coal	Soft Coal	Cotton	Silver Polishing[2]
Asthma	0.2	...	0.4	....	0.9	0.4	3	....	4	....	7	2
Bronchitis	0.3	5.0	9.0	14.4	2.0	2.1	4	38	100	71	16	15
Pneumonia	0.5	0.4	0.2	1.0	0.2	0.1	7	3	2	5	2	1
Tuberculosis	3.7	0.1	0.7	....	....	0.1	50	1	8	....	....	1
Suspected Tuberculosis	0.8	....	....	....	....		11	....	....	....	....	
Diseases of the nasal fossae	4.9	8.7	8.4	....	26.0	7.4	66	66	94	....	208	53
Influenza and grip	4.8	20.6	18.1	1.2	18.6	6.6	58	157	202	6	149	47
Pharyngitis and tonsilitis	0.7	7.6	5.7	4.7	2.9	0.7	9	57	64	23	23	5
Pleurisy	1.1	0.3	0.9	0.2	0.2	0.4	15	2	10	1	2	3
Other respiratory diseases	....	0.1	1.7	1.4	0.4	....	....	1	19	7	3	....
Total respiratory diseases	16.5	42.7	45.1	22.9	51.2	17.9	223	325	509	113	410	128
Years of Observation	....	....	....	....	....	....	1346	763	1116	494	801	715

[1] Lasting two consecutive working days or longer.
[2] Males only, to agree with the other studies.

In Table 2 is presented a comparison of sickness for two working days or longer from respiratory conditions in six dusty trades, the annual rates being based on 100 years of observation. [4]An examination of this table shows that in

the anthracite industry and in the cement plant there is a high rate of illness from grip and certain other minor respiratory affections. In the bituminous coal industry, bronchial diseases and pneumonia are excessive; in the cotton industry grip and diseases of the nasal fossae are predominant; whereas in the granite industry, tuberculosis and pleurisy are excessive. The dusty occupations in silver polishing show no excess from any cause. I should like to comment on the fact that in the case of the bituminous coal miners the excessive pneumonia rates may be due to other factors than dust, as recently pointed out by Brundage and myself in the study of the frequency of pneumonia among steel and iron workers, in which bituminous coal miners comprised a considerable portion of the workers studied. [5]

So much for the importance of a knowledge of the exact composition of dusts encountered in industry. I shall be quite satisfied if I have been able to impress upon you the necessity for a very careful consideration of this phase of the dust problem. I shall next discuss the factor or particle-size of industrial dusts.

SIZE OF DUST PARTICLES

It was pointed out yesterday by some of the speakers that particles of a size greater than 10 to 12 microns in longest dimension are very seldom found in the lungs. This absence of larger particles is partly due to the fact that the numbers of such particles greater than 10 microns in size present in industrial air is, as compared with the lower sizes, comparatively small and due to gravity and the protective action of the mucous surfaces of the upper respiratory tract, these larger particles do not penetrate to the terminal portions of the respiratory tract. Hence, in studying the size of dusts we need only concern ourselves, as a rule, with those dust particles that are less than 10 microns in longest dimension.

Yesterday, Mr. Cummings, in discussing this phase of the dust problem, showed you a particle-size distribution curve on a sample of quartz dust taken not directly from the air of an industrial establishment but from the gross material used by him in dusting animals at their labora-

tory. Mr. Cummings also discussed fully his technique of size separation by the elutriation method and suggested that such a method be used in the future for particle-size studies. He also intimated that to date no studies of particle-size of dusts in industry have been made. I should like to point out one or two things in connection with his remarks. To begin with the use of his suggested method of particle-size measurements is impractical for field studies, due to the fact that the method is very laborious and tedious. In his case it was necessary to resort to such technique since he was interested in obtaining fractions of known sizes for experimental purposes. We are not interested in actually separating dusts into sizes but merely in determining the percentage distribution of the various sizes of dust particles existing in industrial atmospheres. For that reason we make use of a simpler technique than the one pointed out by Mr. Cummings. I shall describe this technique in a few minutes. Mr. Cummings also suggested that particle-size studies of dusts in industry should be conducted. I should like to point out that such studies have been made by the Public Health Service and that during the past year I have reported on two occasions the results of our studies of the sizes of dust particles present in industrial atmospheres. [4] I shall present these results to you in my discussion of the quantity of dust in air, since these results fit in very well with that phase of the problem. For the present I shall confine myself to a brief description of our technique in obtaining and measuring aerial dust.

Samples of dust in air may be obtained by the use of the Owens Jet Dust Counter. [7] The advantage of this instrument over other devices is that the Owens apparatus projects the atmospheric dust in unaltered condition directly on a microscope cover-slip. This cover-slip may then be properly mounted and examined microscopically; using a magnification of 1,000 diameters (oil immersion objective) the horizontal diameter of a representative number of particles is measured by means of a calibrated filar ocular micrometer. [3] With this magnification it is possible to measure particles as small as 0.5 microns in diameter, while

particles smaller than 0.5 microns are easily distinguished at this magnification and their presence recorded.

Yesterday, Mr. Cummings told you about using the microphotographic method of making particle-size studies. Such a method may be used in dealing with fairly uniform sizes of dusts, such as he obtained by the fractionation method. But it must be kept in mind that industrial dusts vary in size from less than 0.5 microns to more than 10 microns in diameter. According to Green [9] and Chamot in order to obtain good microphotographs the particles should be in one plane, free from Brownian movement and well-dispersed. Since industrial dusts are not uniform it is difficult to have them all in one plane. We have attempted to make particle-size studies of industrial dusts and enlisted the aid of the research workers of the Eastman Kodak Company, who, after much difficulty, obtained some good microphotographs of dust for us. The particles shown in their prints were at a magnification of 5,000 diameters. A study of the size-frequency distribution of the dust obtained by the photographic method revealed practically the same results as obtained by the direct microscopic measurement previously described. So that for all practical purposes the simpler and less expensive filar measurement is one of practical application and fulfills the requirements of our problem.

DUST CONCENTRATION

The last factor which I desire to discuss in connection with the study of the industrial dust problem is indeed a very important one, the one dealing with the concentration of dust in the air. It is apparent that when the dust concentration is high the exposed person will inhale a greater quantity in a given period of time than he will when the dust concentration of the atmosphere is relatively low, and since the rate of production of the fibrosis is partially dependent upon the rate at which the dust is inhaled, this latter item plays an important part in predicting the relative danger of different environments. Hence, the need for the evaluation of the quantity of dust in the industrial atmosphere is obvious.

From the practical hygienic viewpoint, we feel that the

particle count is at present the best quantitative index of the degree of atmospheric pollution. The decision as to the size range of the particles which should be included in the dust count will be somewhat dependent on the size of the dust particles actually found present in the industrial atmosphere. It is obvious that the size of the smallest visible particle will depend on the magnification and type of illumination used in the microscope, the refractive properties of the dust and to some extent, on the visual acuity of the observer. We must bear in mind that our chief interest in this problem is in the industrial hygienic aspect. Primarily we are interested in differentiating between the dust content in ordinary normal atmospheres, not known to be harmful, and certain industrial dusts which are known to be associated with lung damage. As I will show you presently, this differnce is sharply marked so far as the dust particles between approximately ½ and 10 microns in diameter are concerned; but the difference between such normal and abnormal air is masked and lost when we include in our determination the particles of ultramicroscopic size which are present in vast numbers in all air.

As I have already pointed out to you earlier, we need not concern ourselves with those particles greater than 10 microns in longest dimension, since the number of such particles present in most industrial air is, as compared with the lower sizes, comparatively small. Let me cite you what available data we have on the lower limit of particle sizes of industrial dusts. In South Africa, Moir [10] examined microscopically 120 dust particles obtained from two specimens of silicotic lung and found that only 13 per cent of the particles were less than 0.5 microns and about 36 per cent of the particles to be less than 1 micron in diameter. The majority of the particles (60 per cent) were between 1 and 3 microns in size. The median size of the dust was found to be 1.2 microns in diameter. Practically the same results were obtained by Watkins-Pitchford, [11] who examined and measured the silica particles in sections of silicotic lungs illuminated by polarized light. Drinker, [12] in comparing the size-frequency of the particles found by Moir with the particles found by him in the sputum of men employed in ore mills, found a close

correspondence. The findings of Moir and Watkins-Pitchford have also been corroborated by Mavrogordato, [18] who examined dust both with light and dark-ground illumination, in sections of human and animal silicotic lungs as well as the dust recovered from these lungs.

In connection with the lower limit of particle-size of dust of pathologic significance the following pertinent question arises: Aside from the evidence direct or indirect of the non-retention of minute particles of dust by the lungs, what evidence is there that appreciable percentages of ordinary industrial dusts ever fragment into those minute sizes less than 0.5 microns in diameter? It is a well known fact that in most of the fine grinding operations in use today, such as in the preparation of paint pigments, considerable energy must be expended to obtain a product, the particle-size of which is less than 0.5 microns in average diameter, and this not in an industry where dust is an evil by-product but where finely divided dust is the chief aim of the whole industrial process.

The best answer to the question just raised, namely: what is the particle-size distribution of industrial dust,

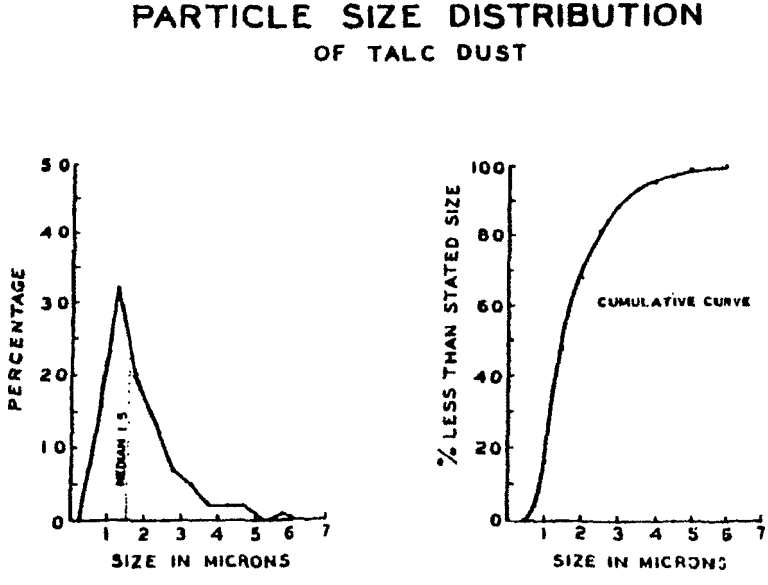


Figure 1

would be data of actual measurements of such dust. Let us see what the available data on this question shows. In 1929, Fehnel [14] reported some particle-size dust measurements in connection with a dust study of hard rock drillers in New York City. As a result of his study, Fehnel reported the findings on three samples, which showed the dust which was less than one micron in size to vary from 1 to 15 per cent. Most of the dust in these hard rock drilling operations was, according to Fehnel, between 2 and 5 microns in size.

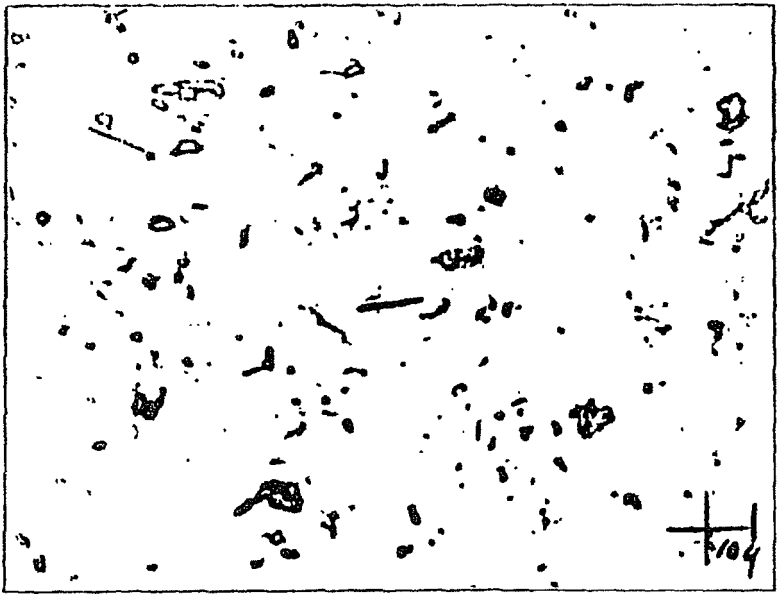


Figure 2
Microphotograph of Talc Dust Obtained with the Owens Jet Dust Counter. Magnification 650X.

Badham, [15] in studying the dust hazard among sandstone workers in Sydney, measured some 16,000 particles of dust in the air of work places and found that 67 per cent of these particles were about one micron in size. From his study Badham states: "It would appear that below 10 microns there is no selective action by the dust cells of the lung and that the particles found in the lung have the same size-frequency as those in the air breathed . . ."

During the past few years, in the course of the numerous dust studies we have conducted, we have collected samples of dust suspended in industrial atmospheres. Recently we completed a study of 26 samples of 11 different kinds of industrial dust obtained with the Owens apparatus. These dusts ranged from that present in sandblasting operations to the dust present in slate and talc milling plants, where the material is ground to a very fine state of subdivision by recirculating the dust in a closed system for many hours. An analyses of the measurements obtained on these 26 samples showed that only 2 per cent of the particles were less than 0.5 microns, 21 per cent less than 1 micron, and the majority of the dust (71 per cent) was found to be between one and three microns in average diameter. In figure 1 you may see the particle-size distribution of talc dust and this curve is representative of the results obtained from the measurement of the dusts so far studied. It is seen that the results of the measurements on the talc dust show that only 16 per cent of the particles were less than 1 micron, the majority of the particles (65 per cent) being between 1 and 2.5 microns in size, while the median size of this dust was found to be 1.5 microns. Figure 2 is a microphotograph of the same specimen of dust.

From the evidence I have just presented on the particle-size distribution of industrial dusts in air, as well as from the previously mentioned studies of dust recovered from lung tissue, it is apparent that we need only be concerned with those dust particles between ½ and 5 microns in size, and from a practical viewpoint the lower limit of particle-size to be counted may well be taken at about one micron. The method of dust counting which we have been using for the past 14 years is capable of revealing particles as small as one micron quite readily, and in the hands of an experienced observer this method, as I will soon show you, reveals quartz particles as small as 0.7 microns in size.

Many methods have been devised and used for the purpose of determining the quantity of dust in air. Suffice it to say that for the purpose of dust sampling in either high or low dust concentrations, the Greenburg-Smith Impinger apparatus now finds universal favor. [10] This

instrument has been used by the United States Public Health Service in all of its dust studies during the past nine years, and is also being used by other workers in this field in this country and abroad.

In this instrument, the air to be sampled is drawn through a glass tube and impinged at a high velocity on a glass plate which is kept beneath the surface of the water or other suitable fluid in the collecting flask. The dust is momentarily arrested, wetted by the collecting fluid, and in this manner trapped.

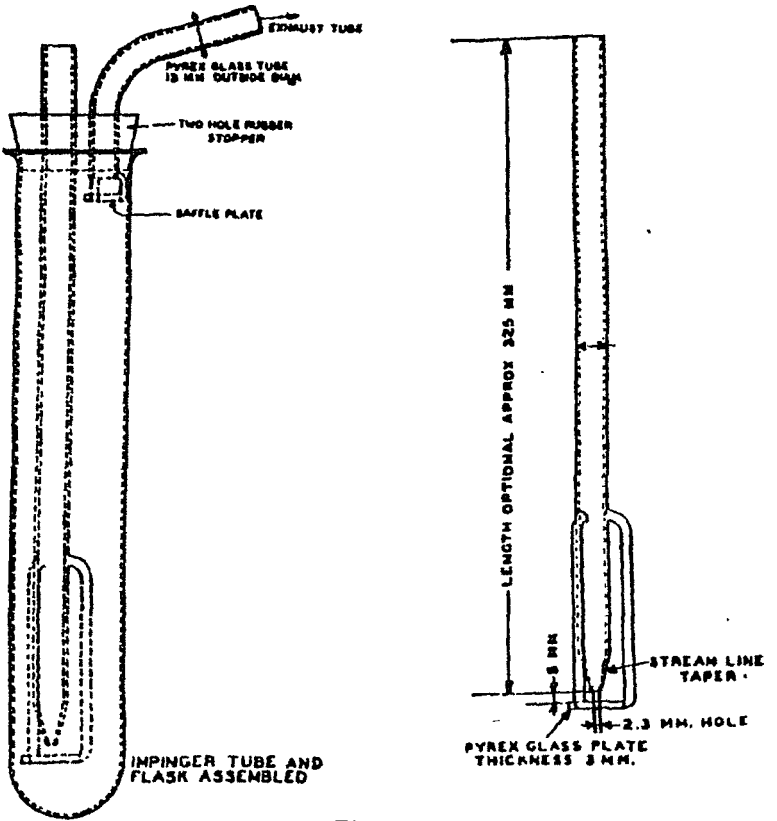


Figure 3

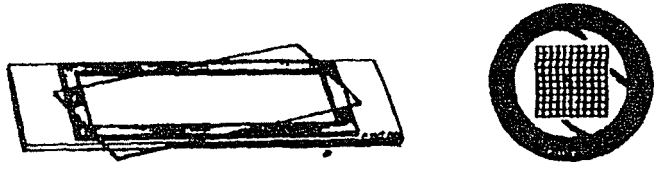
The Impinger apparatus consists essentially of two portions: First, a source of sufficient suction to draw the air to be sampled through the sampling device; and second, the sampling device or impinger itself, which consists of

a container and the impinger tube and plate. As a source of suction one may use either an electrically-driven pump or a compressed air ejector device. In figure 3 you may see the essential portions of the apparatus, which consists of a straight piece of Pyrex glass tubing 13 mm. in outside diameter and approximately 325 mm. in length. The tube is drawn down in stream line form at its lower end, to a tip with a 2.3 mm. orifice. A circular glass impinging plate approximately 3 mm. in thickness and 25 mm. in diameter is attached to the lower end of the impinger tube at a distance of 5 mm. from the orifice by means of three glass rods. The collecting medium (distilled water) in the sampling flask is of sufficient volume to keep the impinger plate immersed at a depth of approximately 3 centimeters. In sampling, the outlet or suction elbow of the sampling flask is connected with the source of suction by means of a suitable length (25 feet) of non-collapsible rubber tubing. The duration of the sampling period should be such as to yield a satisfactory suspension of dust for analysis and is thus dependent on the concentration of dust in the atmosphere. Under the usual industrial conditions, samples of from 10 to 30 cubic feet of air yield sufficient suspended dust for analysis. Since a sampling rate of 1 cubic foot per minute is maintained, this will require a sampling period of from 10 to 30 minutes.

The collecting efficiency of the apparatus is dependent upon adherence to the previously cited impinger tube dimensions and the sampling rate of one cubic foot of air per minute. Experimental tests of this instrument against finely divided silica dust suspensions in air have consistently yielded efficiencies of 98 per cent at the specified sampling rate.

Since practically all dusts are, to some extent, soluble in water, it is good practice to analyze the samples as soon as possible. Such practice tends to prevent any undue flocculation as well as any solvent action on the dust particles. In the laboratory the dust suspension in the sampling fluid is filtered through a 325-mesh screen and then diluted so that the number of dust particles in the microscope field is equal to approximately 50 to 75. Two or more 1-cc. portions are placed in Sedgwich-Rafter cells for counting. One of these

cells is shown in Figure 4. The microscope is of the ordinary type provided with a suitable eyepiece and objective and fitted with an Abbe condenser. A Whipple disc eyepiece micrometer, of the kind shown in Figure 4, is placed in the microscope eyepiece and the microscope tube length is adjusted so that the side of the ruling in the eyepiece is 1 mm. in length. (We employ a 7.5 X eyepiece, 16 milli-



Sedgwick-Rafter Cell Whipple Disc

Figure 4

meters objective and a tube length of 178 millimeters). As a source of illumination we use an ordinary type of microscope lamp with the Abbe condenser system dropped below the usual focusing point and the iris diaphragm adjusted so as to provide a high degree of visibility for refractile objects. In making counts the microscope should be focused throughout the depth of the cell since some of the dust particles may remain in suspension. Since the counting cell is 1 mm. deep and the area in the microscopic field is 1 square millimeter each count represents the amount of dust in a cubic millimeter of the sampling fluid. Knowing the original dilution of the sample and the number of cubic feet of air sampled it is an easy matter to compute the number of dust particles in the sample per cubic foot of air. It is of course necessary to make control dust counts on the sampling fluid.

In taking dust samples the location of the sampling place, the time during which sampling is conducted and the duration of sampling are all selected with the idea in mind of yielding the definite data required by the study in progress. It is impossible to specify any set rules for this portion of the procedure. Obviously the requirements of the study in progress govern the procedure to be employed; this procedure can best be judged by the investigator on the job.

Yesterday, Mr. Cummings discussed quite freely some of the alleged weaknesses in the technique of dust counting

which I have just described to you. This technique is the so-called "light ground" illumination method as contrasted to the "dark ground" method advocated by Mr. Cummings. In our standard method the object is examined by the aid of transmitted light and appears on a lighted or white ground field, whereas in the dark-field technique the field is dark and the objects appear as if they themselves emitted the light by which they are seen. Now, Mr. Cummings made many statements yesterday upon which I should like to comment. To begin with he said that our method of counting is not easily duplicated by various observers. I wish to point out that my dust counts have been repeatedly checked by workers at Yale University and the Division of Occupational Diseases of the Connecticut State Department of Health. In fact, in the latter Division counts are made as a routine procedure, independently, by two different observers and these have always checked very closely. So that we are convinced that persons with normal vision and training in dust counting will find no difficulty on this score. As Mr. Cummings said it may be true that I have good visual acuity, but apparently so do many other individuals trained to conduct this type of microscopic work.

Now I'd like to compare some of the facts known today concerning the light-field and dark-field methods of counting dust. To begin with in our standard light-field technique, illumination is not as important a factor as in the dark-field method. Mr. Theodore Hatch of Harvard University, who has had considerable experience in conducting dust studies, has recently informed us that in the dark-field method of counting advocated by Mr. Cummings, if the time of counting a dust sample is prolonged and if the illumination isn't properly standardized, one may obtain results 300 or more per cent in error. Our method is known to reveal only those particles of hygienic significance. As I have already indicated to you earlier all evidence points to the fact that we need only concern ourselves with those particles ranging in size from about ½ micron to 5 microns and for practical purposes the 1 micron particle may be accepted as the lower limit of particle-size to be counted. Mr. Cummings said that with our technique we can not see particles smaller than 2 microns in size and cites as proof

that he and Mr. Fehnel of the Metropolitan Life Insurance Company, who assisted him in developing his method, received confirmation of this belief from makers of microscopic instruments. I suppose what they are all basing this statement on is the knowledge that with a magnification of about 100 diameters and light-field illumination, the resolving power (the ability to see fine details) of the microscopic system is about 1.5 microns. Now anyone with any experience at all in the use of a microscope should know that the limit of visibility, (the possibility of seeing if an object is present), which is what we are interested in, is much lower than the limit of resolution. To satisfy myself on this point I have taken quartz dust, a highly refractive dust and hence offering a severe test of visibility, and obtained a fraction ranging in size from 0.4 to 1.6 microns and averaging 0.9 microns. This fraction was obtained by Mr. Cummings' elutriation method. I have placed a sample of this dust on a ruled cell and measured 100 of the particles at a magnification of 1,000 diameters, sketching each particle, in its exact location in the ruled area, on a piece of paper. Next I examined these same 100 particles by our standard technique and was able to see those particles of a size of 0.7 microns and larger. In other words, if we recall the particle-size distribution data on industrial dusts I spoke of earlier, we can actually count, by our method, 85 per cent or more of the dust present in industrial air. You will certainly agree with me that the small percentage of dust we fail to count by our method is neglible when one takes into consideration the fact that with Mr. Cummings' dark-field dissecting condenser method one can be several hundred per cent in error if the illumination is not carefully adjusted. On the other hand we know nothing of the lower limit of particle-sizes revealed by the dark-field method. It is a well-known fact that such methods of dust counting are apt to reveal ultramicroscopic dust particles which are present in all air, thus tending to mask the significance of the results.

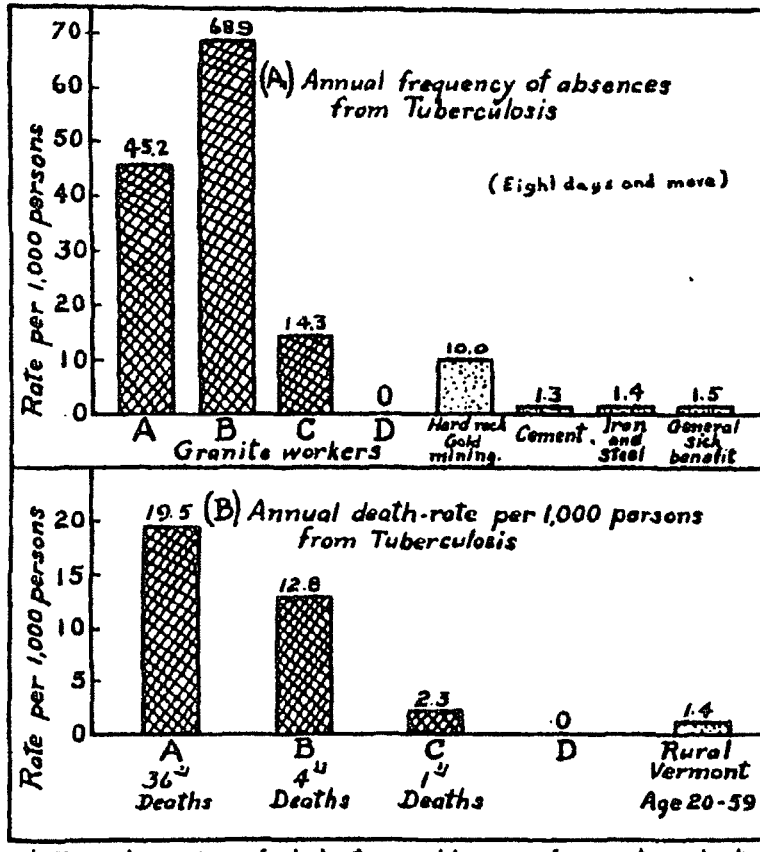
In your General Orders on Dusts, Fumes, Vapors and Gases I notice in the section dealing with dust that you define countable particles determined by United States Public Health Service technique, as those ranging in size

from 2 to 10 microns in longest dimension. I don't know how you happened to decide on this lower limit of 2 microns but from all that had taken place at this conference yesterday I am now able to confirm my suspicions. Certainly you didn't obtain this figure of 2 microns from the Public Health Service when it assisted you in framing the dust code at your Milwaukee meeting in 1930. I know, since I happened to be one of those testifying at that hearing. I should like to suggest that if you decide to leave the section dealing with dust in its present wording, that you omit any reference to our standard technique, since as I have already pointed out to you, we are able to count particles of a size less than 2 microns with our technique.

Mr. Cummings has also informed you that the Public Health Service has tentatively adopted his dark-field technique for dust counting. So far as I know, and I should be pretty well informed on this point, the Public Health Service has only one technique and that is the standard technique it has been using for the past 14 years in all its dust work and a description of which was recently republished in the Public Health Reports. [10] I have been using this technique for nearly ten years and have examined some 1500 samples of dust with this method. We have been able to establish certain standards of dustiness by the use of this dust counting method and have found it to be of practical application. It may be possible that some day we may wish to alter our present technique, but if we do, it will first be necessary to standardize any proposed method of substitution and obtain comparative results between the present method and the new one. In this connection I should like to call to your attention that any results obtained by the proposed method of dark-field counting, or by any other dark-field technique, cannot be interpreted in the light of our own findings; that is, our present standards of permissible dustiness can not be applied to results obtained with the dark-field methods, and hence for all practical purposes these latter results are of questionable value.

The best criterion of the value of any method of measurement is the demonstration of its successful use in a practi-

cal application. Such a test of our dust counting method has been offered us in our various dust investigations and especially in our granite study. Let us briefly examine the results of this study. The whole group of workers was di-



↓ From beginning of study to working up of report — about three years — among 972 workers: A, 614; B, 104; C, 146; D, 108.

Figure 5

(A) Annual frequency of absences from tuberculosis (eight days more); (B) Annual death rate per 1,000 persons from tuberculosis.

vided into four sub-groups, depending on their average dust exposure. In figure 5 one may note the annual frequency of absences due to tuberculosis and the annual death rate per 1000 persons from tuberculosis among the workers in

these four groups. This figure also compares this data with similar information for other industrial groups.

In group A, which included hand-pneumatic tool operators and in which the exposure average about 59 million particles per cubic foot of air, it was found that practically 100 per cent developed an established silicosis within 10 years from the time of beginning employment. Also, in this group the highest rate was found for cases diagnosed on physical examination as having active tuberculosis.

Furthermore, a definite relation was established between length of service in the industry and the prevalence of tuberculosis.

In group B were included those workers other than hand-pneumatic tool operators who were also exposed to more than the average plant dustiness. Taking the group as a whole, the average dust concentration was nearly 45 million particles per cubic foot of air. This group showed the same reflection of a dust hazard as Group A.

In Group C, consisting of those occupational groups exposed to the average plant dustiness (about 20 million particles per cubic foot of air), silicosis developed much more slowly than in the groups just discussed and there appeared to be very little excess in the rate for tuberculosis, with no tendency for an increase according to length of service. Analysis of occupational mortality over a period of 25 years, however, indicated that some of the occupations in this group may have been exposed to a real dust hazard.

Group D was made up of those occupations in which the dust exposure was less than that of the average plant dustiness. The average exposure for the group was less than 10 million particles per cubic foot of air. Although a certain amount of silicosis was found even in this group, there was no indication of serious results, even when the workers had been employed for many years.

It is clear from these data that there exists a high correlation between the dust counts and the effects of this dust exposure on the health of the granite workers. It is obvious, therefore, that the technique of dust analysis which I have described to you constitutes a valuable index of the hazardousness of dust inhalation and one from

which the degree of hazard may be judged with practical certainty.

Before concluding the discussion on dust concentration in industry I should like to present to you some of the results I have obtained in numerous industrial establishments during the past nine years. In Table 3, a summary

TABLE 3

AVERAGE DUST COUNTS IN CERTAIN DUSTY TRADES.

Industry and Occupation	Dust Exposure in millions of particles per cubic ft.	Average per cent of quartz in dust
Talc Mining and Milling:		
jack hammer drillers	2,160	none
packers	50	"
muckers	45	"
crushermen and cylindermen	14	"
Slate Finishing Mills:		3
floormen	1,598	3
loaders	1,276	3
disc crusher operators	312	3
Quartz Grinding Plant:		99
mill operators	173	99
laborers	83	99
packers	55	99
Granite Quarrying and Finishing:		35
Leyner drillers	144	35
jack-hammer drillers	112	35
hand pneumatic tool finishers	59	35
machine pneumatic tool finishers	36	35
plug drillers	37	35
attendant labor (indoors)	17	35
Anthracite Coal Mining:		1.5
miners and helpers	232	1.5
attendant labor	31	1.5
Bituminous Coal Mining:		1.2
coal cutters and loaders	112	1.2
attendant labor	4	1.2
Marble cutters	33	none
Cotton Cloth Manufacturing:		none
carders	9	none
weavers and spinners	5	none
Silverware Manufacturing:		1.7
dusty trades	5	1.7
non-dusty trades	2	1.7

is presented of the average dust content of the air in a few of these dusty industries. This table clearly shows that the highest dust exposure was in the talc mines, slate finishing mills, quartz grinding plant, coal mining and granite cutting industries. Owing to the high percentage of quartz present in the dust of quartz grinding and granite

cutting plants, as compared with the dust in the other industries listed in Table 3, quartz grinding and granite cutting are revealed to be the most hazardous of the occupations we have studied.

DUST SUPPRESSION

Yesterday there was a discussion on the floor concerning the advisability of allowing a worker with a healed or latent tuberculosis to continue in a dusty occupation. I am in no position to add anything to this discussion, since it is not in my province, but it seems to me that if we could remove the dust evil at its source we would not need to be concerned about workers with healed tuberculosis or other lung conditions conducting work in industry where dust is generated. Unfortunately we haven't very much constructive engineering data on the subject of dust removal. Nor have we an abundance of information on the permissible amounts of various dusts which may be tolerated with impunity. We have such data for granite cutting dust, cement, coal, certain kinds of slates and talcs, but we still know very little concerning the effects on the health of workers exposed to other industrial dusts, such as feldspar, tripoli, opal, pure talc, and innumerable other dusts. There was a study recently reported on the effects on workers exposed to diatomaceous silica dust in a California deposit now utilized commercially. [11] However, no dust counts were reported so that wo do not know the severity of the exposure nor is there any information given on the total length of exposure. I'd like to suggest that in any future industrial dust studies all phases of the problem be investigated, somewhat along the technique used in our dust studies, so that we may be able to measure the relative importance of all the involved factors. In the California study just cited we do not know the degree of dust exposure that apparently brought about the physical changes in the workers that was reported by the investigators. To me this omission is a very serious one since we have no definite basic data to use in designing dust removal equipment for this particular industry, for it is very

expensive and at times impossible to remove all the dust generated in a process.

With your indulgence I should like to discuss very briefly some of the methods used in combatting the dust hazard. The application of any one particular method, of course, will depend largely on the industrial process creating dust. The protection of workers against certain dusts known to be toxic may at times be accomplished by the substitution of a non-toxic material for the toxic one. As an example of such a procedure we have the possible use of a metallic or other type of artificial abrasive for sand in the sandblasting process, in those operations in which it is not essential to use sand, a substance high in quartz content. Again, the mechanical enclosure of the dust-creating process also serves at times to protect the worker. An excellent illustration of this type of protection is afforded by the modern sandblast barrel used in the cleaning of small objects. Sometimes it is possible to protect workers by the substitution of wet for dry processes. In one instance in our granite study an operator using a diamond point pneumatic tool worked the stone wet; the resulting dust amounted to 22 million particles per cubic foot. The same operator was then requested to work the stone dry; as a result, the amount of dust reached the high figure of 45 million particles per cubic foot. In the weaving of asbestos cloth it has been possible to reduce the amount of dust in the air by wet weaving to one-fourth of the amount present when the process is conducted by dry methods. However, wet methods are not always to be relied upon for the complete suppression of dust. For example, in a study of the dust hazard in the wet and dry grinding shops of an ax factory, Winslow and Greenburg [18] have shown that protection afforded by wet grinding, as compared with dry grinding using an exhaust system is, in most instances, illusory. The same result was found in our granite study in determining the exposure of tool grinders in that industry. Another example of this procedure in allaying dust is the use of a spray of water in Leyner and jack-hammer drilling in hard rock, although the new type of Kelley Dust Trap is now finding much favor for this kind of work. [19]

In certain cases, such as in the sandblasting of large

castings in sandblast rooms, the only practical safeguard to the worker is to provide him with a mask or helmet of the positive pressure type. In the sandblasting investigation recently conducted by the United States Public Health Service in cooperation with the National Safety Council, we found that with a well-designed and well-maintained mask or helmet of the positive pressure type a supply of 6 cubic feet per minute of dust-free air will give ideal protection to the worker. However, one must always bear in mind that the ultimate criterion of protection should be the dust determination of the air within the helmet during blasting and not the quantity of the air supply itself.

In certain cases, where the exposure is brief and the work is of such a nature that it is impossible to use positive pressure air devices, one can give the employee protection by furnishing him with an efficient respirator of the filter type.

In most dusty processes, however, the most effective means of dust elimination are by the use of properly designed local exhaust ventilation systems. Since in many instances it is a difficult, costly, and at times unnecessary procedure to remove all the dust in the vicinity of a worker, we first need to determine the minimum amount of a certain dust which the worker can apparently tolerate with impunity. Such information can be made available by the type of studies carried out by the Public Health Service, which I have already mentioned frequently. For example, in the granite study already referred to you will recall that apparently 10 million particles of granite dust (containing 35 per cent quartz) per cubic foot of air could apparently be inhaled with impunity for 30 or more years. At the time our study was made there were already a few plants utilizing local exhaust ventilation in connection with pneumatic tool operations, those operations falling in our Groups A and B. [20] Studies of the efficiency of these dust removal devices disclosed that with proper maintenance these local exhaust devices were capable of keeping the dust concentration at the worker's breathing level at a safe minimum, as may be noted in Table 4.

TABLE 4

COMPARISON OF ATMOSPHERIC DUST CONDITIONS BETWEEN TWO GRANITE-CUTTING PLANTS EQUIPPED WITH LOCAL EXHAUST VENTILATION AND PLANTS NOT SO EQUIPPED.

Occupation	Average dust count in millions of particles per cubic foot of air; winter observations		
	Plants without efficient local exhaust system	Plants with efficient local exhaust system	
		Plant X	Plant Y
All pneumatic hand-tool operations	55.2	23.5	9.5
Surface cutting	45.0	15.3	10.6
Tool grinding	30.0	5.9	12.1
Sand blasting	6.9	3.5	5.5
General plant atmosphere	22.6	5.6	8.9

A further study of the efficiency of the local exhaust devices used in the more modern plants labelled as "X" and "Y" in the table, yielded the results depicted in Figure 6. This figure shows the relation between the dust concentration in the air at the worker's breathing level when using various pneumatic tools and the air velocity at the local exhaust ducts. From this figure it is apparent that a velocity of 1,500 linear feet per minute is necessary to keep the dust concentration at the worker's breathing level below 10 million particles per cubic foot of air, the amount found in our study as not associated with any disabling illness.

Later on workers at the Harvard School of Public Health reported the results of their laboratory studies of the design of dust control systems for use with pneumatic cutting tools. [21] These workers corroborated our findings as to the degree of air velocity at the exhaust ducts necessary to keep the dust concentration at the breathing level to an amount less than 10 million particles per cubic foot of air and, in addition, were able to specify the type of hood to be used which would give such a velocity with a minimum volume of air.

Before concluding my remarks I should like to leave one word of caution with you. You will recall in our granite cutting bulletin we stated as a result of our investigation that apparently 9 to 20 million particles of granite dust in

a cubic foot of air, containing about 35 per cent quartz, can be inhaled with impunity even for 30 or more years. We do not state anywhere in our bulletin on this study that an exposure to any other dust of the same concentration and quartz content could also be tolerated without harm. Yet there has been a tendency on the part of many

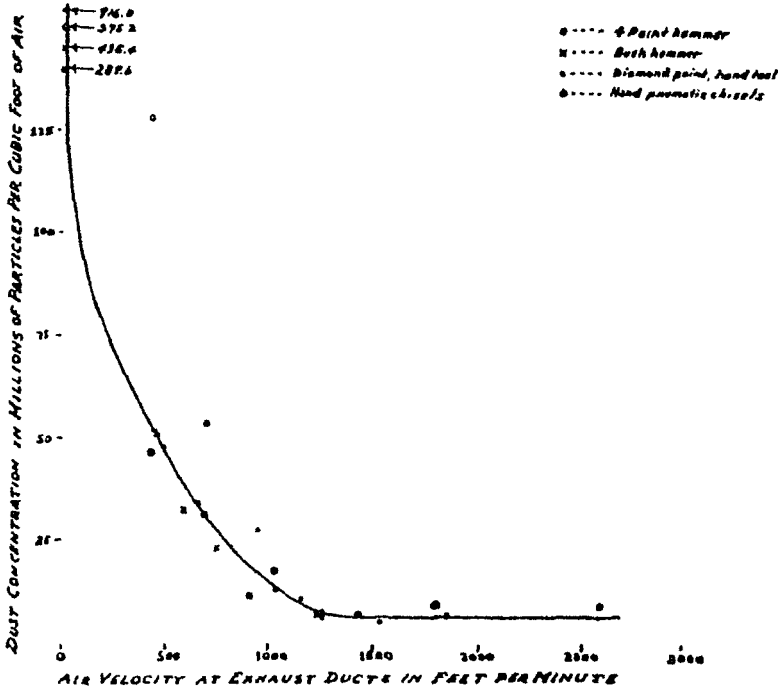


Figure 6

Graph showing the relation between the degree of air velocity at exhaust ducts and the amount of dust inhaled by granite cutters using various pneumatic tools.

individuals to interpret our findings in that light and to set up standards for other dusts in other industries based on our granite cutting data. This may be sound practice but it also may lead one into error if taken too far. For example, to reason that since 15 million particles per cubic foot of air of a 35 per cent quartz-containing dust has been found to be safe, that therefore about 4 million particles per cubic foot of a 95 per cent quartz-containing dust, such

as sandstone is also a harmless dosage, may be good arithmetical reasoning but may lead one into a serious error. My suggestion is that we should determine the permissible limit of dustiness for each individual dust.

In the interim, for those dusts that we do not have any basic data of threshold dosage we can demand from industry that it maintain conditions in the workrooms equal to those already found in the best plants of a similar industry in actual operation.

I shall now be happy to answer any questions that may have suggested themselves to you during the course of my remarks.

DR. SCHLOMOVITZ: Did you make any study of the dust in the air passing through respirators?

MR. BLOOMFIELD: We have conducted studies on positive pressure masks and helmets as used by sandblasters during actual work in a sandblast room but we have not conducted studies in the field nor in the laboratory on the efficiency of respirators. Excellent work on respirators has been done by the United States Bureau of Mines and at Professor Philip Drinker's laboratory at the Harvard School of Public Health.

DR. SCHLOMOVITZ: You don't happen to know the size of particles that pass through the mask?

MR. BLOOMFIELD: Last month Professor Drinker presented a paper at the National Safety Congress in Washington, in which he stated that a careful measurement of the size of dust particles entering and leaving respirators shows a slight but important reduction in average size of the dust particles. In fact, there are now respirators on the market which on testing by Professor Drinker were found to have efficiencies in excess of 90 per cent against silica dust of a size less than 2 microns. So that apparently some respirators do filter out the small particles.

MR. WILCOX: Is there any recognized activity among these manufacturers of protective devices, respirators and so forth, to try to develop something that will give relief?

MR. BLOOMFIELD: Very much so. The manufacturers of equipment and protective devices are quite active. In

our sandblasting investigation we found equipment being sold today which is capable of keeping the dust count to a concentration of less than 2 million particles per cubic foot of air at the breathing level.

MR. WILCOX: That is where the air is fed by tubes?

MR. BLOOMFIELD: Positive pressure masks and helmets are now being sold, which if properly maintained and supplied with a sufficient volume of dust-free air will protect a worker fully inside a sandblast room and in addition there are now on the market sandblast cabinets, tables, barrels and other equipment, which if maintained in a proper state of upkeep, will also give ideal protection to a worker without the use of masks or positive pressure devices. We have found such conditions during the course of our sandblast investigation.

MR. KNUTSON: Have you any suggestion to offer with regard to safety devices that might be used in connection with the sandblasting of castings weighing several tons?

MR. BLOOMFIELD: One can sandblast such castings in large rooms, exhaust the rooms properly and furnish the worker a good positive pressure respiratory device. There are installations of the automatic type which do not necessitate working inside the blasting zone, but such equipment is not practical for huge castings.

BIBLIOGRAPHY

1. Bayley, W. S.: Non-Metallic Mineral Products. Henry Holt & Co.
2. Thompson, L. R., Brundage, D. K., Russell, A. E. and Bloomfield, J. J.: The Health of Workers in Dusty Trades. I. Health of Workers in a Portland Cement Plant. Public Health Bulletin No. 176. (1928)
3. Heffernan, Patrick: Exposure to Silica Dust Without the Occurrence of Silicosis. Journal of Industrial Hygiene, Vol. 8, No. 11, Nov., 1926.
4. Russell, A. E., Britten, R. H., Thompson, L. R., and Bloomfield, J. J.: The Health of Workers in Dusty Trades. II. Exposure to Siliceous Dust (Granite Industry). Public Health Bulletin No. 187. (1929)
5. Brundage, Dean K., and Bloomfield, J. J.:The Pneumonia Problem in the Steel Industry. Journal of Industrial Hygiene, Vol. 14, No. 10, Dec., 1932.
6. Bloomfield, J. J.: The Dust Content of the Atmosphere in Various Dusty Industries. Published Nov. 18, 1932, in the United States Daily, Washington, D. C.

7. Owens, J. S.: Jet Dust Counting Apparatus. Journal of Industrial Hygiene, p. 522, April, 1923.
8. Hatch, Theodore, and Choate, Sarah P.: Statistical Description of the Particle Size Properties of Non-uniform Particulate Substances. Journal of the Franklin Institute. March, 1929.
9. Green, Henry: A Photomicrographic method for the Determination of Particle Size of Paint and Rubber Pigments. Journal of the Franklin Institute, Vol. 192, No. 5, p. 637, Nov., 1921.
10. Moir, J.: Report on a Specimen of Dust from Silicotic Lung. General Report of the Miners' Phthisis Prevention Committee, Pretoria, 1916, Appendix 9, pp. 138–140.
11. Watkins-Pitchford, W.: The Situation, Outline and Dimensions of Mineral Particles Visible by Polarized Light in Sections of Silicotic Lungs, mounted in Canada Balsam. General Report of the Miners' Phthisis Prevention Committee, Pretoria, 1916, Appendix 8, pp. 135–136.
12. Drinker, Philip: The Size-Frequency and Identification of Certain Phagocytosed Dusts. Journal of Industrial Hygiene, Vol. 7, No. 7, July, 1925.
13. Mavrogordato, A.: The Value of the Konimeter. Publication of the South African Institute of Medical Research. No. 17.
14. Fehnel, William J.: A Study of Silica Dust in Hard Rock Drilling in New York City. Journal of Industrial Hygiene, Vol. 11, No. 2, Feb. 1929.
15. Badham, Charles, Rayner, H. E. G., and Broose, H. D.: Dust Sampling in Sydney Sandstone Industries. Report of the Director-General of Public Health, New South Wales. December, 1927, p. 74.
16. Greenburg, Leonard, and Bloomfield, J. J.: The Impinger Dust Sampling Apparatus as Used by the United States Public Health Service. Public Health Reports, Vol. 47, No. 12, March 18, 1932.
17. Legge, Robert T., and Rosencrantz, Esther: Observations and Studies on Silicosis by Diatomaceous Silica. American Journal of Public Health. Vol. 22, No. 10, October, 1932.
18. Winslow, C.-E. A., and Greenburg, Leonard: A Study of the Dust Hazard in the Wet and Dry Grinding Shops of an Ax Factory. Reprint No. 616, Public Health Reports, October 8, 1920.
19. Hatch, Theodore, Kelly, George, S., and Fehnel, J. Williams: Control of the Silicosis Hazard in the Hard Rock Industries. II. An Investigation of the Kelly Dust Trap for Use with Pneumatic Rock Drills of the "Jackhammer" type. Journal of Industrial Hygiene, Vol. 14, No. 2, Jan., 1932.
20. Bloomfield, J. J.: A Study of the Efficiency of Dust-Removal Systems in Granite-cutting Plants. Reprint No. 1324, Public Health Reports, Oct. 18, 1929.
21. Hatch, Theodore, Drinker, Philip, and Choate, Sarah P.: Control of the Silicosis Hazard in the Hard Rock Industries. I. A Laboratory Study of the Design of Dust Control Systems for Use with Pneumatic Granite-cutting Tools. Journal of Industrial Hygiene, Vol. 12, No. 3, March, 1930.

November 17, 1932. Afternoon Session

DR. ALBERT E. RUSSELL, *Surgeon*, United States Public Health Service; *Surgeon*, U. S. Bureau of Mines.

It is a great pleasure to be here with you and to take part in the discussion of this very interesting and intricate subject. What has gone before has been very interesting and seems to have brought us up now to a consideration of the physical condition of the worker and the clinical pictures produced by the effect of inhalation of dust.

It has been my pleasure to work in the public health service in a study of the dusty trades. Beginning in 1924, we made observations in the cement industry, the granite industry, coal mining, both bituminous and anthracite and other dusty trades. We have found a number of very interesting things and two of these studies have been published in bulletins No. 176 and No. 187. In the report of cement study we made the statement that although in most of these dusty trades fibrosis of the lungs was produced, we were of the opinion that this fibrosis, even though it was similar to that produced by other dusts, did not represent the same degree of disability. I think our subsequent studies and observations of workers in other dusty trades, have borne that out. I will show you today a number of slides of X-rays from different industries showing reaction to dust as shown in X-rays and we will discuss it more at length at that time.

In doing our studies we started out with a program which would include dust counting and dust analysis, records of absentees from work and finding out the reasons for absence. We were greatly interested in the sickness and longevity, and the amount of time lost from work due to sickness. A complete physical examination with special attention given to the chest conditions, was made on all employees where it was possible. First, I will take up some of our findings in the morbidity records produced by exposure to dust. Second, you will recall that Mr. Bloomfield discussed that we had groups of people exposed to different concentrations of dust. Our most extensive study was made in the granite industry in Barre, Vermont, and it was my pleasure to be there and to have charge of this

study throughout its course. Our sickness records and later our X-ray and other findings, seem to divide the workers into certain groups according to results we were getting and later when Mr. Bloomfield made his dust counts we found a very close correlation of dustiness and the amount of sickness and disability and deaths in direct proportion to the concentration of dust. With the first slide we will start considering the morbidity.

This slide (see Plate 3, p. 10, Bul. No. 187, U. S. P. H. S.) is a photograph of a man at work cutting stone. You have heard a lot about different methods of dust counting, but this will reveal the fact that the worker is exposed to quite a bit of dust which can be seen with the naked eye. This tool is the hand pneumatic tool which vibrates very fast and in working on the hard granite rock he bends over looking at his work and brings his nose closer to the dust he is creating. The general atmosphere in that plant was rather high and is not shown in the photograph, nor do you get it in visibility unless there are direct rays of light shining in the plant. You may walk through it and it does not seem to be very dusty. That is where the dust counts tell more accurately as to the extent of the dustiness.

This slide (see Fig. 2, p. 20, Bul. No. 187, U. S. P. H. S.) shows the different occupations that we had in Vermont. These bars indicate the intensity of exposure. Our groups divide themselves into the first and second group, ten to twenty million; then from twenty to sixty were the two second groups. You can see the general plant atmosphere was about twenty million particles per cubic foot of air. There were a number of occupations with comparatively low exposure. Keep that in mind in considering the morbidity records which we will discuss later.

The South African standard of permissible dustiness is indicated in the black bar at the bottom. The groups I told you about we called A, B, C, and D, This next slide (See Fig. 17, p. 88, Bul. No. 187, U. S. P. H. S.) is a graph which shows the number of persons with and without silicosis in the four dust groups indicated in the other graphs by length of service. The bottom line indicates length of service and each line indicates a certain group of workers. Almost invariably by the end of fourteen

years practically all of the workers in the higher dust counts had evidence of silicosis. Some of it was rather slight in the X-ray and very little in the physical and other characteristics, but with the X-ray there was almost invariably evidence of silicosis at the end of that period.

You have seen this slide (See Fig. 22, p. 118, Bul. No. 187, U. S. P. H. S.) before today when Mr. Bloomfield discussed it. It carries out the line of thought, the incidence of sickness or absences was lower in these groups of workers beginning with group C and D in the low exposure line and higher in groups A and B. The tuberculosis death rate for males in rural Vermont is indicated here. Group D was less than that and is perhaps due to the small number of people. Group C is about the same, but groups A and B are quite a bit in excess.

This graph (See Southern Med. Journ. Sept. 1932, pp. 919-927) shows the frequency of disability lasting longer than one week on account of sickness, exclusive of accidents or from respiratory diseases in industry. The industries are specified. The industrial groups; the first group is gold mining in the Black Hills of South Dakota. Their rate was 208. General manufacturing is shown and is more or less an average. That includes industries in a number of northern states. This would seem to indicate that that group of workers had that much sickness in excess of what we might call the normal. The respiratory disease, the incidence of sickness in the lower part with granite cutting in Vermont heading the list. Gold mining in the Black Hills second, Portland cement third, anthracite fourth, with general manufacturing, which we might say is about an average at this point, you can see in those four industries the incidence of sickness is this much in excess of average. I might say in relation to the gold mining in the Black Hills in South Dakota that we do not know the extent of the exposure to dust because no dust counts were made. We might compare it to dustiness in other mining, but we don't actually know what it is there. It is evident that there was an excessive amount of dust which was practically pure quartz. The reason why we haven't as much tuberculosis among the gold mining, I think, is because of the fact that the labor turnover is

rather high and these workers develop silicosis and become partially disabled and go to their homes before developing tuberculosis. The workers in Vermont remain at their homes inasmuch as granite cutting is a skilled trade. The labor turnover in granite cutting is very little, whereas in gold mining it was very high. That will explain many differences that may follow. The gold miners head the list in the incidence of influenza; cement workers second, anthracite coal mining third, and granite cutting four, and general manufacturing at this point. In bronchitis, acute and chronic, anthracite coal miners head the list, Portland cement second, general manufacturing third, which would indicate that the cement workers had a slight amount in excess of granite and anthracite coal miners had still more.

This is a continuation of the same group of workers. Respiratory tuberculosis only is considered in the industries we have been talking about. Granite cutting in Vermont heads the list, gold miners in the Black Hills second. I have explained that the reason why I think this isn't greater is because of the labor turnover; anthracite coal mining third, and general manufacturing at this point. It would seem there is this (pointing) much in excess of tuberculosis in the three industries above. In the iron and steel and Portland cement plant it seems to be about the same as general manufacturing. In the incidence of pneumonia, workers in iron and steel head the list; granite cutters in Vermont second and general manufacturing third. We would think that the excess was not as great as in some of the preceding diseases. I might say a word about the economic conditions. The iron and steel workers are not paid as well as granite cutters. Their living conditions are not as high and that may be a factor in considering tuberculosis and other diseases, particularly those of the respiratory tract. The granite workers in Vermont made $1.00 an hour. They worked eight hours a day, forty-four hours a week; this was the minimum wage at that time. The highly skilled workers made more. The ones able to carve and cut statues made up to $20.00 a day. They lived well. The best group of industrial workers I have ever seen. The incidence of tuberculosis among them was not an economic factor.

This brings us down to the petrographic analysis of the dust. In silver polishing the total silica percentage varied according to different occupations and different materials used, and the quartz content varied likewise. Mr. Bloomfield showed a graph this morning in which the dust count in these particular occupations was quite low, I think about five million particles at the greatest. In cement industry the total silica is expressed as 21% in the raw cement, but the quartz content of finished cement is less than one per cent. It is stated to contain 6% before passing through the kilns but the burning process reduces the amount. In coal mining the rock dust is quite high in quartz because the overlying stratum was sandstone. Quartz content of coal is 1.2%. In hard coal we have the same explanation here with 1.5 silica in the coal with 31% quartz in the rock dust. The granite we have explained has a total of 70% silica with about half of it as quartz. There is a point I wish to mention here. In the United States most of the hard coal as anthracite is found in an area in a few counties in Pennsylvania. Bituminous coal is scattered pretty well throughout the country. In the state of West Virginia there are areas where overlying stratum is sand stone and others where different rocks make up the overhead rocks. The silica content of these rocks varies tremendously in different localities, which makes it impractiable to compare the results from the study of one group of coal miners to another. The amount of silica in the rock dust to which he might have been exposed should be stated. It is very difficult to say that one industry is comparable to another or that one part of the same industry represents the same conditions throughout. We don't know until we have made careful analysis of the dustiness in the various occupations. This slide gives us an average dust count under ten microns in certain dusty trades, of which we will speak later. Cement dust averages about twenty-five million. In granite cutting there are fifty million particles per cu. ft. for the upper two groups and sixteen million for the lower two groups and in anthracite coal mining one hundred twenty four million for the miners and 11,000,000 for attendant labor. Bituminous coal mining has one hundred and two million and 3.3

million for the attendant labor. In the silver manufacturing 4.1 million and 800,000. I think these dust counts are very interesting and we believe that they correlate well with our findings which we will bring out as we go along.

This is a graph (See fig. 8, p. 53, Bull. No. 187 U. S. P. H. S.) expressing the incidence of sickness from all causes in these four dust count groups in the granite cutting industry in Vermont. It is by length of service and the absences are eight days or more and the rate is per thousand. Here we have a group with less than ten years exposure, with the four groups starting at about the same point. Group C and D with lower exposure average about sixteen million. As they grow older the total incidence of sickness seems to diminish. The upper groups, whose exposure to dust averaged about 50 million, their rate of sickness increased in proportion to the length of exposure to dust.

This slide shows the prevalence of tuberculosis, and we considered only active pulmonary tuberculosis, by length of the service and dust count groups. This is based on physical examinations, and X-ray to determine the presence of the disease. Here we have group C, starting with a higher rate than groups A and B. We think perhaps there may be an economic factor here. The attendant labor did not have as good living conditions as did groups A and B. Up to ten years' exposure there isn't much significance but after ten years you see these two groups of workers having increased incidence of tuberculosis in direct proportion to the length of service. After thirty to forty years exposure, it seems to reach its peak; taking for granted that a man is about twenty years old when he starts to work we would expect that the group of workers having tuberculosis would be around fifty years of age. I believe that our average for the age at that particular study was a little over forty-nine years.

This slide I am showing is about an average chest. Sometimes we get a very good presentation of this slide, and it shows more than at other times, due to illumination. This is what is usually found in a more or less average person, that is a person who has not had tuberculosis, silicosis, or any of the other chronic pulmonary diseases. Those

of you who are not physicians and not familiar with an average chest should bear this in mind as we go along and you will be able to follow changes indicated by X-ray. Notice that the diaphragm curves with a regularity on each side. This is the hilus of the lung of which Dr. Gardner has spoken, and the light areas are the functioning portions.

(X-ray No. 4, case No. 397, near p. 92, Bull. 187, U. S. P. H. S.) I called your attention to the regularity of the diaphragm in the preceding one. I want you to see the irregularity in this case. You will notice that through the lung fields there are linear markings spreading out toward the periphery and we think that it was due to the inhalation of dust. This man was a granite cutter who had cut stone about fifteen years. In writing up our report of granite study in Vermont you will recall we didn't attempt to classify our cases of silicosis by the then existing one classification which was the South African one. Our cases did not seem to fit into that classification. This man, since this picture was made in 1924, has developed tuberculosis and died of clinical pulmonary tuberculosis.

This is another granite worker who had been working about twenty-three years in cutting stone; it shows an increase in the shadows with further evidence of disease. You will note in this slide, as in the ones which follow, that there is more pathology indicated in the right lung. That has been attributed to the fact that the right bronchus is a little larger than the left and it curves at an angle of about twenty-four degrees, whereas the left bronchus bends at a little sharper angle and is slightly smaller. You will notice in this case the diaphragm is fairly regular. In the preceding one there were evidently pleural adhesions causing irregularity in the contour of the diaphragm. It is possibly due to a latent tuberculous condition in addition to the effect of dust.

The next slide. The two cases preceding have been cases of silicosis uncomplicated by tuberculosis. You noted in those cases that the apex and upper portion of the lungs were fairly clear. Most of the pathology was in the middle and lower portion.

This case (X-ray No. 43, case No. 195, Bull. 187, U. S. P. H. S.) is one of pulmonary tuberculosis in a granite

worker. You will notice the apices are fairly clear. There is quite a lot of pathology indicated by the markings in the lower portion of the lungs. The man had a cavity at the base of the right lung. We checked most of these cases for sputum analysis and almost invariably we found they were positive. These granite workers all expected to have tuberculosis; they were rather averse to having sputum analyses.

This (X-ray No. 45, case No. 32, Bull. 187, U. S. P. H. S.) is another worker showing similar condition with an active tuberculous process determined by physical examination in the lower portion of the right lung. The usual markings are present and he had the usual symptoms of tuberculosis.

The next slide. This is still another granite worker. This slide shows the thickened pleura in this area together with the usual reaction to dust and to tuberculous infection. You will notice in the earlier cases the shadows were linear, and that they spread out to the periphery from the hilum in each case. The markings here are more conglomerate and less distinct in character.

This (X-ray No. 40, Case No. 174, Bull. 187, U. S. P. H. S.) is a very interesting case in that there seems to be a pneumonic process here. The man had clinical and physical signs of active tuberculous disease and he died a short time after this picture was made. You will note a little irregularity in the diaphragm at this point and the contour of the diaphragm on the other side is not clear. There seems to be an adherent mass at that point.

This (X-ray No. 46, case No. 194, Bull. 187, U. S. P. H. S.) is a very interesting case. He was working and developed a condition which is quite like pneumonia physically. Quite a bit of pulmonary tissue became consolidated, which on physical examination gave all the signs of pneumonia. He was intensely dyspneic, but had no temperature nor toxemia, and as this cleared up bacilli appeared in the sputum about three weeks after.

I believe somebody said that we didn't have any case of mottling of the lungs similar to the South African cases among the Barre workers. This is an exception. It is the only case we had which presented this type of picture. The

linear shadows are not present as in your other cases of silicosis. There is a great similarity in this picture to what we see in Africa in gold miners. Just why this case has this same marking I am unable to explain. He weighed about two hundred pounds and was one of the finest looking chaps I ever saw. He had no symptoms, except a little dyspnea on exercise. I saw Dr. Pancoast after I had been doing that study and he showed me the only other slide like this of a granite cutter with this particular characteristic and he said it was given to him as being a typical granite cutter in Vermont. This is the only one I have with these particular markings. This man was exposed to the same intensity of exposure and same percentage of silica as the preceding case.

DR. GARDNER: For how long?

DR. RUSSELL: Twenty-three years. This man was a Scotchman. In Barre they have a nice variety of nationalities, Italians, principally the Northern Italians, French-Canadians, Scotchmen, American and New England Yankees, few Spaniards and quite a few Scandinavians and a few other nationalities.

A VOICE: What was the subsequent history of that man?

DR. RUSSELL: I don't know. Two years later he was still well. I hope to go back and check up on some of these people this coming year.

This (X-ray No. 63, case No. 18, p. 132 near, Bull. No. 187 U. S. P. H. S.) is a case of an Italian who cut granite twenty-five years. Eleven years prior to the time this X-ray was made he had been living on a farm in Vermont and the reason he came to me was that he was getting to be quite dyspneic and he was worrying about his condition. We took X-rays and this is the picture. He had been in dust-free atmosphere eleven years on the farm. His dyspnea increased and two years after this time he had a fulminating tuberculosis; he was past fifty years of age.

This is another case (X-ray No. 62, case No. 299, Bull. 187, U. S. P. H. S.) with a similar history. This man was Irish, cut granite 26 years; thirteen years immediately prior

to the time this picture was made he had been a night watchman in an insane asylum; a dust-free occupation. You will note that he had deep markings in the bases of his lungs. It has lost that linear character and is more the cottony or confluent type. He was very dyspenic, and was 64 years of age. He found it quite difficult to do any chores around his home and his occupation was quite sedentary, no strenuous work to do and yet he was bothered with dyspnea. The superintendent of the hospital was quite interested in him and very much interested in our work, and about three years after this picture was made he wrote me that the man's dyspnea had increased and that he had toxic symptoms and tubercle bacilli in his sputum.

This (X-ray No. 68, Case No. 339, Bull. 187, U. S. P. H. S.) is a case with a similar history; a New England Yankee. He cut granite 17 years, then went to Oregon and cultivated apples, and he had been out of dust about fifteen years. He came back to Vermont and cut granite for one year and he began to have some difficulties and this is what the X-ray revealed. He later developed a fulminating type of tuberculosis, and like most of those granite cutters with tuberculosis, he did not last so long.

This slide is of an X-ray of a potter. The man had spent more than twenty years in the pottery industry. I think potter's clay contains about 35% silica, about the same amount we have in granite. With a picture like this, I think we can safely say that the man was exposed to too much dust. The flocculent shadows are possible, due to a super-imposed tuberculosis.

The next one is a case of a potter with earlier stage of the silicosis. You will note that the markings are more of a linear type and spread out at the hilus toward the periphery and to this portion with the enlarged hilus glands. There are a few calcified tubercles in the area. He had been a potter twenty-six years when this picture was made.

I don't know much about acute silicosis, but I am presenting this slide as a case of early silicosis with rather short exposure. This man's occupation was that of a foreman of tunneling work, and he was exposed to dust for about a year. This tunnel went through pure quartz rock

and he was in it quite often during the working hours. You will note that he has quite an increase in the linear markings. He had perhaps an old tuberculosis condition in this hilus with a few calcifications. He had no disability.

This is another tunnel worker with rather extensive pulmonary fibrosis together with a little interlobar pleurisy at that point. The exposure of this case was something of about a year. I have seen autopsies from cases from that same tunnel with exposure of a year or less, which produced a fatal silicosis. This is perhaps an acute silicosis.

This is a slide showing a man who had been a lens grinder, grinding pure quartz lenses for a period of eight months and he was disabled with silicosis at the time the picture was made. He was in a tuberculosis sanatorium and the staff physicians were unable to demonstrate the presence of tuberculosis. The patient's complaint, like most silicotics, was that of shortening of breath. The hiluses seem to be greatly choked up. The shadows are rather dense and no doubt he has silica deposited in the lung. He has not had time, in the course of eight months, to develop the fibrosis indicated in the preceding X-ray.

This brings us up to the consideration of pneumoconiosis of a different cause. You remember what I said in the beginning, that you couldn't look at fibrosis in one case and say that was comparable to that of another. You will see this worker has quite a bit of lung markings and yet he has no disability, or apparently not much to worry about in the future. He was a soft coal miner.

This is another soft coal miner who had been getting a little rock dust. We believe this irregularity and the markings are more of a linear character and more discrete than in the previous one. I must admit there is very little pathology there. I am showing it to contrast with the preceding one.

This is an X-ray of an anthracite coal miner. Apparently the anthracite coal miners have more rock dust exposure than the group of bituminous workers at large. This case is interesting, because of the interlobar pleurisy shown at this point (between upper and lower lobes). It is the only one I have ever seen like that. You will note that he has quite a bit of pathology indicated in both lungs, yet the

character of the markings is different from that of silicosis which I have shown you before.

DR. WILLIS: Would you mind commenting on the drop heart?

DR. RUSSELL: We don't know so much about drop hearts, but we have an idea that cases having drop heart have some remote old tuberculosis. It has been the experience in South Africa that workers with drop hearts developed silicosis and tuberculosis much more rapidly than workers who did not have it. You will note in the preceding case the heart had a greater curve to the left than this; notice that the border of the heart in this case is almost perpendicular.

This is another anthracite coal miner showing different markings. You will notice the contrast in this heart and the preceding one. This case and the preceding case had miner's asthma.

This is another miner with still a little different picture. You will note that the markings in these cases don't seem to be parallel. You will note the character of these markings are suggestive of the presence of silica, because of the rather uniform distribution.

This is an X-ray of a cement worker, a man who had been working in a cement plant about ten years. You will notice that it has different characteristics. Notice the light that seems to be coming through in this area with more or less clouding at the areas to the outer portion. There are fine linear markings underneath. I wish to call your attention to the fact that these workers were exposed to about one per cent quartz in their dust. The dust counts, I think, averaged 25 million particles.

Following this there will be some slides of marble workers. Cement dust contains a lot of calcium or lime. The marble workers were exposed to dust which contained about 88% calcium carbonate, and I think the company's analysis of the dust stated that it contained less than one per cent of quartz. Our analysis was taken from quite a different place and we found practically no quartz in the marble dust.

This is an X-ray of a man who had been cutting marble twenty-two years. You will note the increased shadows

around the hilus and around the larger bronchi. But you will note the light areas which indicate there is quite a lot of good functioning tissue. He had absolutely no disability and no evidence of tuberculosis or any other chronic pulmonary condition. You will note in these slides of marble workers there are quite a lot of calcifications. This one hasn't as much as the other. These workers were the same age as the granite workers, occupations very much alike and the dust concentration little less in the marble than granite plants. We found no case of active tuberculosis among the marble workers. Dr. Rogers of the Vermont Sanitarium for tuberculosis stated he never had a marble worker as a patient unless they had also worked in granite. These marble workers had quite a lot of calcification of the costal cartileges.

This is another marble worker. You will note the calcification of the hilus of the lung and some increase in fibrosis. This case, like the others, had no disability. You will note the calcification here.

Those pictures weren't as good as we would like to have had them, but the best we could do out in field work. Field work is quite different than in institutions. We work under handicaps in so many places. In this picture the calcifications are present with quite a degree of fibrosis. The constant exposure to dust over a long period of time carrying bacteria in the lungs would produce fibrosis no doubt, but not necessarily a disabling fibrosis. Marble dust does not seem to predispose to any chronic disease.

MR. TARRELL: Did you follow that patient up to see how soon it was he became disabled?

DR. RUSSELL: Did I say he was disabled?

MR. TARRELL: You said he was not disabled when you took the X-ray.

DR. RUSSELL: That has been a year ago and he was all right when we took it. We haven't heard anything.

This is a marble worker and the calcifications of these costal cartileges are indicated in this picture.

I am showing this next slide because of the calcification of the cartileges with apparent calcification of the pleura.

This man was a slate worker. His history is that he lived in the area of this slate and that for most of his life, aside from farming, that had been his sole occupation. The slate workers in one plant which we observed were not exposed to any quartz. The plant adjacent to this mill had about 3% quartz in the slate it used. You will note in considering slate that the silica content of slate varies in different areas so we can't say that slate has a definite amount of silica without determining it first. The same holds true for granite. There are certain granites which have much less than 35%. I think most of the Vermont granite contains about 35% quartz. You can see the futility of trying to compare X-rays of the lungs from workers in one industry to those of another.

This is an X-ray of a slate miller. You will notice he has a few pulmonary markings, with quite a bit of fine linear fibrosis. We found in the group of slate miller workers one case who had an active tuberculosis. He had been in this slate mill about six years and apparently the tuberculosis was just incidental and not a result of his occupation.

Other slate workers showed hilus thickenings with a few calcifications. Still other slate workers showed a different picture but not extensive pathology.

This is an X-ray of another slate worker showing quite a bit of shadows in the lower portion of the right lung with a few calcifications.

This is the X-ray of a man who had, or has, asbestosis. His occupation was that of cleaning and restoring the asbestos on pipes in one of our government hospitals. He had been working at the trade about six years, I think, and you will see, he has fibrosis of both lungs. The character of the fibrosis is quite a bit different from that of the granite workers. He had disability and the government compensated him for it.

This is a graph showing classification of silicosis. The first classification was made in South Africa. They started out classifying their cases as primary and secondary stages, and as they learned more about the disease they had to add to this classification. They learned that they had a stage earlier than primary and it was called ante-primary,

and as it developed there was still another condition before they could be considered as ante-primary so they called that "more fibrosis than usual." They took an average chest of the average individual as more or less a standard, and there were a number of cases having more fibrosis than the average person and yet not enough fibrosis to be placed in the classes mentioned. When they diagnosed a case as having ante-primary, primary or secondary silicosis, their arrangement is that the man is entitled to compensation and he is removed from his dusty occupation. This is the original classification of silicosis.

In Australia, they used a slightly different classification. They called their chests normal instead of average. In Dr. Moore's paper he gives the reason for cases having more fibrosis than usual, tuberculosis, that is, latent tuberculosis, or healed tuberculosis; cardiac conditions, and dust. The stages are called early and advanced. Their advanced cases seem to be considered more or less in two phases. At the International Silicosis Conference this classification into first, second and third stages was recommended. The South Africans have their classifications written into their laws and they have to stick to it, although they said a more workable or reasonable classification should be adopted and adhered to. The classification of stages designated as negative or average chest, more fibrosis than usual, first, second and third stages was recommended. Tuberculosis complicating any one of these stages would automatically place it in third stage. The first stage of silicosis with tuberculosis would therefore be considered as the third stage.

This slide represents a case which would more or less come under the stage of "more fibrosis than usual." However, the density in here is getting a little beyond that stage, but you will note that the slide illuminates well, indicating that there is apparently quite good aeration of the lung. He was a sand blaster working for the United States Government and he had been in this occupation for eight years. He was a colored man and he had several masks. He not only wore one but two masks neither of which were good. I took this X-ray because a man working in the same place he was had died of tuberculosis. They

had filed a claim with the employees' compensation commission for silicosis for this deceased sand blaster. There was an X-ray of him at the hospital where he had died, and apparently he was entitled to receive compensation, and it was so recommended. This particular case had been working along with him. This picture was taken in 1926; he later developed tuberculosis and died, and in 1929 the widow filed claim for tuberculosis and silicosis. This stage I consider "more fibrosis than usual" and if he had had tuberculosis and more fibrosis at that time it would have been considered a third stage case.

This is a slide of a granite worker I'm showing to bring out the point that when a tuberculous infection exists the rate of progress of silicosis seems to be augmented. I find that I have left out the slide that should follow, but I believe that I can describe the stage. In the right lung there is quite a lot of fibrosis, more than in the left lung. This picture was taken in 1925; last year, in 1931, I X-rayed this man again. This condition was about the same, but he has apparently developed tuberculous infection in this lung and there was undoubtedly much more fibrosis in this lung than in the right lung. Ordinarily and in the preceding cases the disease has seemed to develop a little more in the right lung than in the left.

You have perhaps seen our beautiful Union Station in Washington. This is the X-ray of the man who carved two of the five beautiful statues which surmount the entrances on the outside to the Union Station. He was one of the most skilled granite workers. This is the right lung. He has quite a snow storm appearance in this portion, lower right. At the time this picture was taken he had rales after cough, afternoon temperature and loss of weight, positive sputum, and the usual signs or symptoms of tuberculosis. (See *Southern Medical Journal*, Sept. 1932, pp. 919–927, for X-ray.)

This next slide is from another case from Vermont. The man had apparently in this spot an old smoldering tuberculosis. This case is not typical of our other cases, some of which I have shown you in preceding slides, in that they have bilateral fibrosis. He had this conglomerate affair with little pathalogy indicated in the other lung. Because

it was different I observed him very closely for a period of two and one-half years while there. I visited in the place a year later and found that he had not been sick nor lost one day from work. I was back in Vermont in 1931 and as I was interested in him we took an X-ray and this is the result. This area has spread pretty well all the way to the periphery together with a tremendous increase of the amount of pathology in the other lung. He was feeling pretty well except that he was very dyspneic because so much of the normal functioning lung had been displaced with fibrous tissue. (X-ray No. 14, Case No. 189, Bull. 187, U. S. P. H. S.)

This is the man's photograph. He doesn't look particularly bad. As long as these cases do not have active clinical tuberculosis they look quite well.

This is an actual photograph (Plate 17, Case 37, p. 150, Bull. 187, U. S. P. H. S.) of the lungs of one of our cases in Vermont. This is the apex of the lung at this point. You will notice dark areas through there. The lighter areas at the bottom are tuberculosis. I want to call your attention to the density of the pleura. The pleura, as you know, is a very small mucous membrane, many times likened to the mucous membrane of your lip. In these cases there was great thickening of the pleura. I believe it is due to the fact that we have an exposure lasting over a long number of years and the possibility that the majority of the cases have a smoldering tuberculosis along with it. That is, I believe that the tubercle bacilli is perhaps a factor in this tremendously thickened pleura. I have seen a number of lungs of silicotic patients who had a shorter exposure to a much higher silica dust than were the granite workers and the pleura was not thickened to the extent of the Vermont cases. Pleurisy pain is one of the symptoms of silicosis which we will consider in the discussion of the diagnosis of silicosis. One of the few complaints which the workers have is pain in the chest. It is more or less trivial and not usually disabling.

They seldom complain of it and don't often stop work. It bothers them more in damp weather. Pains in the chest with shortness of breath is about all that these Vermont workers complained of in the course of silicosis until they

begin to develop signs of active tuberculosis, that is, temperature, loss of weight, increase in the amount of cough with productive sputum, etc.

This slide is one showing the death rate in rural Vermont from pulmonary tuberculosis beginning about 1900. In the granite industry in about 1894 they introduced the hand pneumatic tool which is activated by compressed air, as an instrument for cutting granite. You will note that about that time the death rate of granite cutters from tuberculosis began to increase. We think that is caused by the excessive amount of dust which is generated in cutting of stone with this hand pneumatic tool. (Fig. 26, p. 180, Bull. 187, U. S. P. H. S.)

I might say a few words about the diagnosis of *silicosis*. I am frequently asked to speak of the diagnostic points of silicosis. It seems to be the practice of so many people to draw their own conclusions from the X-rays alone. We believe that in the scientific practice of medicine, all points should be considered in making a diagnosis and certainly this should be done when there is the matter of extent of disability to be decided on. In Vermont we had complete physical examination, history of the case which included the entire occupational life, and that with any symptoms he may have had. As I said before, very few of them have many complaints. They even forget that they have a little hacking cough which is perhaps due to mechanical irritation of the upper respiratory tract. Ordinarily, unless they have a cold or infection, they do not produce sputum. They may complain of an occasional pain in the chest and usually of a slight shortness of breath. One of them expressed it to me in this way—"I am beginning to get so I can't get my second wind." When the reserve is called upon they seem to be unable to get their "second wind."

Another phase in the diagnosis of *silicosis* is the consideration of respiratory diseases they may have had. I showed you in the slides that this particular group of workers had more respiratory disease than the usual individual. The physical examination of these workers is frequently disappointing. You may listen to the chest, and hear very little and when you see the X-ray you are sur-

prised at the extent of pathology present, but even so that is no reason for omitting the physical examination. The chest expansion is almost invariably limited in these workers and we find it almost in direct proportion to their length of exposure and which would be, of course, according to the progress of the disease. The granite workers were a husky robust type of people and you would reasonably expect them to be capable of at least 2½ to 3 inches expansion, whereas, most of them had one inch or less. They seem to have an abdominal type of breathing rather than thoracic. I found that examination by palpation was quite disappointing, the fremitus was not usually increased in the same proportion that it is in fibrosis of tuberculosis. Tuberculosis produces a more or less localized fibrosis, whereas, in silicosis it is more or less generalized, and that may explain the difference in the fremitus in the two conditions. The breath sounds in these cases in Vermont were not changed to any particular variety. There seemed to be more softening of all the sounds. The fibrosis of tuberculosis produces more definite changes in breath sounds than does silicosis. I saw some cases of silicosis recently which had developed the disease after very short exposure and they had breath sounds which were quite different from the Vermont cases.

In the acute cases, which I saw recently, the principal portion of fibrosis and infiltration were in the upper lobes of the lungs. The lower part of the lungs were emphysematous. The upper portion seemed to be functioning very little. In the silicosis cases in our Vermont study, rales were not heard unless infection was present. When tuberculosis complicates silicosis, almost invariably rales can be heard after the patient coughs, as in tuberculosis in non-silicotic people. A great amount of fibrosis may conceal the presence of rales to a certain extent in silicotic patients. Our cases in Vermont had very few signs, symptoms or complaints until they began to develop an active tuberculosis. That was a very definite point with them. They then complain of increased shortness of breath, pains in the chest, loss of weight, afternoon temperature, productive cough and the usual other signs of active tuberculosis. These patients seemed to have pulmonary hemor-

rhages more than most cases of tuberculosis. We had several deaths from hemorrhage of the lung and the doctors there told me about other cases that had died there of it prior to our study. One of them had died on the lawn of a doctor's office trying to get help. Hemorrhage is the result of ulceration of these tuberculous areas.

One of the cases of acute silicosis was giving me his history recently, and he was quite perturbed because after he had a coughing spell his chew of tobacco was gritty. This is a new angle on the elmination of dust. He didn't mind the cough, but he didn't like to have his chew of tobacco spoiled. I think that it is the result of ulcerations and erosions of these silicotic and tuberculous lesions and dust was eliminated along with other debris.

The tuberculous complication of all the cases in Vermont, other than the pulmonary type of tuberculosis, were comparatively rare. Most of these cases developed a fulminating type of tuberculosis and did not live so long as the uncomplicated case, thereby lessening the chances of complications. We had one man die ninety days after he quit work and most of the other cases died within a year. We had only one man who lived two years after he stopped work. I think that the comparatively short period of time they lived after developing tuberculosis, would perhaps preclude the development of other tuberculous complications. There were, however, a few cases of glandular tuberculosis, one case of infection of the inguinal glands, and a tuberculous elbow with adenitis of the axillary glands. A few children in these families of granite cutters developed tuberculous meningitis.

I think you people are particularly interested in information relative to the point where a silicotic becomes disabled, or where he should be compensated. In our cases in Vermont we had no disability from silicosis unless they developed tuberculosis. I don't recall, and I don't believe we have recorded a single instance of a case with disability without the disease. Our cases had from 15 to 30 and 35 years of exposure. I don't believe that you can estimate the disability from the X-ray alone and that is why I urge that cases be studied not only by X-ray, but by physical observations as well.

This slide is a case of a man who was the lens grinder who had been exposed to silica only eight months and was disabled, yet from the X-ray it didn't appear he had any disability, but he was bedfast. I believe I spoke of the fact that I have seen a number of disabled cases of silicosis recently. The greatest amount of pathology was in the upper portion of the lung in these cases, which is directly opposite to what we saw in Vermont. Most of these recent cases were exposed to silica dust less than two years, and have disability, whereas, in Vermont the workers were exposed over a period of many years.

There is one other thing I might speak of and I think Mr. Bloomfield touched on it this morning, and that is about the so-called anti-silicotic dusts. There is one thing that Mr. Bloomfield didn't bring out, that I wish to mention at this time. The original report which started the discussion of anti-silicotic properties of dust, did not include dust counts to show that a hazard really existed. There have been no scientific studies made on the subject, but rather a lot of surmising. If there is an antidote for silica dust, we should make every effort to learn about it. It would alleviate much suffering and save many lives as well as much expense to industry. The author of this article did not show that enough dust was present to produce silicosis, and because the workers didn't get silicosis, he concluded the clay kept them from getting it. He gives the analysis of this dust and the total percentage of silica in the material was 88 or 84%; the total percentage in silica in Vermont is 69% with much less quartz than his dusts seem to have had.

The following table shows that granite dust contains less silica and more of the so-called "anti-silicotic" elements than what is found in the materials used in brickmaking.

Analysis of "Dark Barre" Granite

Silica	69.89
Alumina	15.08
Iron oxide	1.46
Iron Sesquioxide	1.04
Magnesia (MgO)	0.66
Soda	4.73
Lime	2.07
Potash (K_2O)	4.29
Water uncombined (H_2O at 110°)	0.31
Water combined (H_2O) (ignition)	0.23
Phosphorous pentoxide (P_2O_5)	Trace

Report of Granite Area of Barre, 1902. George I. Finlay, State Geologist of Vermont.

Materials Used in Brickmaking

Contents of Sample	Brick Factory A	Brick Factory B
Silica	88.90	84.90
Alumina	7.42	9.88
Ferric oxide	0.16	0.19
Manganese, magnesium and tin oxide	0.44	0.85
Soda	1.49	0.84
Lime	0.00	0.34
Water and Loss	2.49	3.30

Heffernan, Journal of Industrial Hygiene, November, 1926.

No study has been reported in which accurate dust counts were made and where it was shown that a silica hazard existed and has been a very definite curative or preventative dust mixed along with it, thereby preventing the development of silicosis. I hope that somebody will give us such a study. I wish Dr. Gardner and Mr. Cummings much success in their endeavors along this line, and I am sure if anything can be brought out as an antidote for silica, they will do it.

There are, perhaps, a number of other things that will come up in the discussion. I believe that is all for the present.

MR. DOE: Dr. Russell, in your discussion of the surveys that you have made, where there was a high silica content and where there was a low silica content, you referred to the presence of fibrosis in both cases. Yesterday I got the impression from Dr. Gardner, that in the non-silicotic dusts there was no formation of fibrosis in the same sense that there was in the case of silicotic dusts. Is it your view that in the non-silicotic dusts the pathology is the same or different?

DR. RUSSELL: I think it is different. In silicosis, you have formation of silicotic nodules as shown by Dr. Gard-

ner. In non-silicotic dusts the silicotic nodules are not present. It is more of a generalized type of fibrosis.

MR. DOE: What I wanted to know was whether it was fibrosis, or whether it was merely the presence of dust that was retained in the lung tissue?

DR. RUSSELL: I think the X-ray penetrates most dusts. I don't believe many shadows are recorded on the film due to dust particles.

MR. DOE: You don't think that most dusts are radio opaque?

DR. RUSSELL: I don't think that most dusts are.

MR. DOE: Are they radio opaque?

DR. RUSSELL: I think they are not.

MR. DOE: So that in the cases of marble workers that you showed, the markings are not the dust, in your opinion?

DR. RUSSELL: I think they are calcifications. That is, combination of fibrosis and lime.

MR. DOE: Then they are partially dust and partially a reaction of the tissues themselves?

DR. RUSSELL: Yes; calcifications are present in tuberculosis.

MR. DOE: When you have a non-silicotic dust such as marble dust, for instance, do you think, Dr. Russell, that the mere presence of those dust particles in the tissue in the manner that you have described, have any predisposing effect in relation to tuberculosis?

DR. RUSSELL: Marble dust?

MR. DOE: Perhaps I don't mean to limit it to marble dust, but to any non-silicotic dust.

DR. RUSSELL: I think that they might predispose to tuberculosis in this way, not directly, but indirectly. In most dusty trades there is an increase in the amount of respiratory diseases and I think it is reasonable to presume that a repetition of respiratory infections certainly would tend to aggravate a smoldering tuberculous condition.

Certainly the tuberculous condition would be better off without having these concurrent infections.

MR. DOE: If a man had a history of two or three pneumonias and a couple of pleurisies, it might be significant?

DR. RUSSELL: Yes. And influenza.

MR. DOE: Would there be any difference in that respect with regard to whether the man had an old tuberculous condition or never had had any tuberculosis?

DR. RUSSELL: I thought you said with an existing or latent tuberculosis.

MR. DOE: I am asking you the question both ways. Whether it is in the one case or isn't in the other, or whether both would be the same.

DR. RUSSELL: I think the incidence of respiratory diseases of any kind, a repetition of them, a number of them, more or less predispose to tuberculosis. We have many patients giving a history of their break down from influenza, etc. Of course, we don't know in those cases whether they had latent tuberculosis or whether it is a new thing. Most of us I think, believe it is latent.

MR. DOE: Is there any way of telling in a particular case?

DR. RUSSELL: Well, the X-ray might reveal the presence of it, but it doesn't always show up on the X-ray. It depends, of course, on the extent of the original infection.

MR. DOE: Well, when you have an individual who has been exposed to non-silicotic dust who becomes tuberculous, are there any means of telling whether the fact that he is tuberculous is associated with the dust?

DR. RUSSELL: I think different cases would vary a lot individually. There might be some characteristics—I think it would be difficult to tell whether or not the case was associated with pneumoconiosis, so to speak.

MR. DOE: In your studies have you made any comparison between the incidence of the disease of tuberculosis

among the population of the locality and in the dusty trades? I notice you had in the Barre survey the incidence of disease in rural Vermont. In other studies have there been similar comparisons?

DR. RUSSELL: Not entirely similar. We have in the anthracite coal the death rate for the county in which we made our study, and the death rate from the coal miners in that area.

MR. DOE: And you had a high percentage of silica at least in the rock drilling, didn't you?

DR. RUSSELL: Yes.

MR. DOE: Have you made any such comparison, Doctor, in the non-silicotic dust studies?

DR. RUSSELL: I don't believe we have. I don't think there has been anything published.

MR. DOE: In the marble study there was no comparison of incidence of tuberculosis with the civil population?

DR. RUSSELL: Not in the report. There were no cases of tuberculosis among the workers at the time we were there. There is a low death rate (from tuberculosis) for rural Vermont.

MR. DOE: Are you satisfied that in the case of the marble study you made, the incidence of tuberculosis was no higher than it was among the rest of the population?

DR. RUSSELL: I think it is quite conclusive that it was not higher among marble workers.

MR. DOE: Would it be your opinion that that would also be true of other non-silicotic dusts?

DR. RUSSELL: I made the statement in the beginning that you couldn't compare fibrosis of one case of pneumoconiosis with that of another. I think each one of these groups of workers is unique in itself. I think with the marble workers, whatever happens to them we couldn't say the same thing would happen to slate workers or any other group. We might hazard a guess but one guess is about as good as another.

MR. DOE: Do you believe that there is any marked difference between the silicotic cases and the non-silicotic cases in that regard?

DR. RUSSELL: Of tuberculosis?

MR. DOE: Yes.

DR. RUSSELL: Yes.

MR. DOE: Well, you have made certain studies of non-silicotic dusts. One is marble and one is slate where silica content was nominal. Is it your conclusion from those studies, that the incidence of tuberculosis is as low as that of the rest of the population in the community?

DR. RUSSELL: I said it was for marble.

MR. DOE: What is the fact as to slate?

DR. RUSSELL: About the same. The same as the population.

MR. DOE: How does the frequency of respiratory infection compare with that?

DR. RUSSELL: We didn't make morbidity studies of the marble workers nor slate workers. According to our histories I think the slate workers have more respiratory diseases than marble workers. I haven't the figures.

MR. DOE: But you wouldn't conclude from that that the tuberculosis rate would be higher?

DR. RUSSELL: The amount of tuberculosis we found among workers was not higher than that of the general population.

MR. DOE: Doctor, will you tell us a little something about what you have found with regard to secondary heart conditions in silicosis?

DR. RUSSELL: I don't believe we have any cases of secondary heart conditions due to silicosis in Barre. Certainly it was not indicated in the X-ray, that there was undue cardiac enlargement, particularly the right side of the heart.

MR. DOE: Did you form any conclusion as to whether that it is common or uncommon, that there could be a

secondary heart condition due, we will say, to third stage silicosis?

DR. RUSSELL: I think it is possible. Quite possible. You have to take into consideration the person's previous condition, his heart condition before the onset of silicosis. A lot of people have a heart disease, and it occurs in and out of dusty trades,—a certain amount, just like tuberculosis in the general population.

MR. DOE: You didn't feel in the Barre survey then that there was any evidence of a secondary heart condition that was attributed to the disease of silicosis?

DR. RUSSELL: No.

MR. DOE: Have you found any such factor in any other study?

DR. RUSSELL: That was the most extensive silicosis study I have done. I would rather you would consult the literature of others who have reported on that to give you their opinion.

MR. DOE: From the studies you have made, Dr. Russell, what would be your opinion as to the advisability or inadvisability of continuing a man in a dusty atmosphere after the presence of silicosis is detected? Suppose you had a "more fibrosis than usual" finding. Would it be your judgment that that man should be excluded from a dusty occupation as soon as that was detected?

DR. RUSSELL: If that were done it would take them out very rapidly. You would have tremendous labor turnover.

MR. DOE: Would it make any difference as to the number of years that he had been employed before the "more fibrosis than usual" finding was discovered?

DR. RUSSELL: I think it would.

MR. DOE: Would it be your view that all such cases should be excluded from dusty employment, if possible? I don't mean to state an impractical situation.

DR. RUSSELL: I think it would be better to clear up the dust than to have to be constantly thinning employes

out. Dust can be prevented and you would have to take men in many dusty trades who are skilled operators. At least they know their trades. If you keep taking them out how would you retrain them for other occupations? It is much cheaper and a better policy to eliminate the dust than to be constantly turning over employes because they develop fibrosis.

MR. DOE: That might be the ideal situation, if one could do that, but assuming that after the best has been done that we are capable of in the present state of knowledge, you take a man with more fibrosis than usual, your idea would be that he should be gotten out of employment?

DR. RUSSELL: If there is an excessive incidence of tuberculosis in that particular occupation. It depends a lot on the extent of silica in the dust. I don't think I could, or anybody else, lay down a definite policy that would be applicable to all instances where you have silica ranging from one to one hundred per cent. Some coal miners show more fibrosis than usual, and yet history of bituminous coal is that they don't have excessive amounts of tuberculosis. It would be folly to take those people out. It would depend entirely on the industry involved and its past record.

MR. DOE: Well, would the quantity of silica in the dust be the determining factor, for instance, if you had a marble worker with more fibrosis than usual, would your recommendation be different than if he were a granite worker?

DR. RUSSELL: Yes, it would be, of course.

MR. DOE: Now, then what do you say would be a reasonably safe maximum and when I say safe, I mean safe from the danger of tuberculosis complication, first, as to silica content?

DR. RUSSELL: I can only give you our experience in Barre. I think it is the only dusty trade where that was well worked out. Groups C and D were exposed to less than twenty million particles of dust, the death rate from those people (from tuberculosis) was about the same as rural Vermont and the death rate in the groups exposed to more than twenty million particles was greatly in excess

of rural Vermont. I can give only my practical experience, as approximately less than twenty million particles per cubic foot of dust containing 35% silica. If a man has evidence of tuberculosis with more fibrosis than usual, I don't think it would be advisable to continue even in that dust.

MR. DOE: Would that have a limitation as to the number of hours—for instance, if you had an occupation that showed less than twenty-five million particles and, say, no greater silica content than you had at Barre, would that employment be safe for that individual for an indefinite period of hours?

DR. RUSSELL: It seemed to be true there; they continue in that occupation for many years.

MR. DOE: Nothing in the studies you have made since has effected that conclusion?

DR. RUSSELL: We haven't done any studies since that time of silica dust. Most were other types of dust. What is applicable to the granite industry I can't say definitely would be applicable to other industries. We believe, however, that men can tolerate twenty million particles in that much silica, (35%).

MR. DOE: Suppose you found a case in your marble survey, of an individual who had been exposed to a dust containing a very nominal percentage of silica, compared to the granite, and that he had been exposed to that dust for a relatively short period of years, say ten years, and he developed tuberculosis, would you say the development of tuberculosis in such a case was secondary to the employment?

DR. RUSSELL: In the marble?

MR. DOE: Yes, in the marble.

DR. RUSSELL: No, I don't think so.

MR. DOE: Would there be any means of, in the present state of our knowledge, connecting a case where the exposure to silica was less than 35% and the number of particles less than twenty million, any means in connecting

up a tuberculosis with such a case with the industry that you know of?

DR. RUSSELL: I think it would be difficult. You have to take into consideration a certain percentage of people have tuberculosis, regardless of occupation.

MR. DOE: That is, in every community there is a certain incidence?

DR. RUSSELL: Yes, certain incidence of tuberculosis.

MR. DOE: Is there any criterion upon which those individuals in that community can be classified as to whether their disease is industrial or not, if the silica content is lower than granite and less than twenty million?

DR. RUSSELL: I don't know of anything. If we haven't the characteristic silicosis proceeding with tuberculosis, I don't see how you can say positively that it is.

MR. DOE: What is the proper method of taking an X-ray to show the characteristic findings of silicosis; is there a proper technique as to under and over exposure being avoided; can you tell us your views on that?

DR. RUSSELL: Personally I like X-ray of one character and other people of another character. It is more or less a personal standard. You realize that there is a big variety of X-ray equipment at the present time. I am using a portable X-ray machine, but I would much prefer to have a larger and better one, a hospital unit. The technique I use for this portable machine could not be used for a hospital. I don't believe there is a standard technique for it, although it has been urged that a certain technique be promulgated and used throughout the country, but with the big variety of X-ray equipment I don't see how it can be easily carried out.

MR. DOE: In a hypothetical situation where you had, say, not a portable machine, but a variety that might be available in the large centers—can you give us any criterion by which we can tell whether an X-ray is properly exposed, not too little and not too much?

DR. RUSSELL: I think the X-ray picture itself would tell you that.

MR. DOE: What are the signs by which we would know?

DR. RUSSELL: If it is over exposed it is difficult to describe. It is easier to demonstrate, if you will, the softer or less intense exposure brings out the finer shadows. You can give more exposure and these fine lines are obliterated.

MR. DOE: Dr. Russell, what is your view on the pathology of the higher incidence of tuberculosis in silicotics?

DR. RUSSELL: I think you had better refer to Dr. Gardner on pathology; he has been discussing that yesterday and today.

MR. DOE: Suppose we accept Dr. Gardner's hypothesis at the moment, that the phagocyte is killed and that necrosis sets in and that is something in the nature of poison which results in the tuberculosis, do you give any comparable situation to that when the dust is non-silicotic in character?

DR. RUSSELL: I don't think it has been shown.

MR. DOE: Dr. Russell, has the Public Health Service done any work in iron mines or has the Bureau of Mines done anything in iron mines with which you are familiar?

DR. RUSSELL: The Public Health Service hasn't and the Bureau of Mines hasn't reported anything.

MR. DOE: That is all.

MR. TARRELL: Dr. Russell, in answering the question of Mr. Doe, as to the standard or the given quantity of silica under which a man might work with safety, you have reference to a group of men have you?

DR. RUSSELL: Yes.

MR. TARRELL: And in one section do you refer to any particular individual person?

DR. RUSSELL: No, they were groups we studied.

MR. TARRELL: If a man has a breakdown of the upper respiratory tract, a breakdown in the line of defense against the inhalation of dust particles, should he work under conditions with twenty million particles in less than 35% silica?

DR. RUSSELL: How are you going to determine the breakdown?

MR. TARRELL: Well, it is a fact isn't it, that the inhalation of dust particles tend to destroy the mucous membrane of the respiratory tract?

DR. RUSSELL: Yes.

MR. TARRELL: That has all been covered. But if a man does have a breakdown and the mucous membrane is destroyed, can that man safely work in an atmosphere of twenty million particles?

DR. RUSSELL: I don't think we said the mucous membrane was destroyed; the ciliary action was limited. The only way you can determine whether or not the cilia are gone is to get a section of the mucous membrane of the trachea to determine that.

MR. TARRELL: The resistive powers of some individuals differ from others?

DR. RUSSELL: Yes.

MR. TARRELL: Then there isn't any definite standard you can set up for separate individuals under which they may work?

DR. RUSSELL: No. The presence of tuberculosis predisposes to silicosis. I told you about this case developing more fibrosis in the right lung and later having a tuberculous infection in the other lung, and the rapidity with which the combined disease developed on that side. Most of such cases in Vermont were the case of father and son, the father had tuberculosis, the son lived with him and presumably became infected. These younger chaps in cases of that kind, seem to develop silicosis more rapidly than the others.

MR. TARRELL: The amount of dust necessary to produce a nasal disease in one individual is not the same in all individuals?

DR. RUSSELL: I think the upper respiratory tracts in different individuals vary a lot. Deflected septum, sinus disease, chronic tonsilitis, would alter the efficiency.

MR. TARRELL: Does that statement apply equally as well to non-silicotic dust?

DR. RUSSELL: I think a person in a non-silicotic dust with a deflected septum, would be more apt to have a respiratory condition than if he didn't have it.

MR. TARRELL: What influence would that have on the development of tuberculosis?

DR. RUSSELL: Well, I think it would be indirectly, as I spoke of before, that that would perhaps have an undue amount of respiratory disease of different varieties, and that in this way it might predispose to tuberculosis.

MR. TARRELL: And by respiratory disease you mean bronchitis, pneumonia—

DR. RUSSELL: Influenza.

MR. TARRELL: Asthma and influenza?

DR. RUSSELL: Yes.

MR. TARRELL: That is all.

DR. OGDEN, of the Illinois Steel Company: Dr. Russell, in all of your slides which showed a true pneumoconiosis other than asbestosis, do you think that there was a possibility of ruling out silica as a determining factor?

DR. RUSSELL: You mean in all of them?

DR. OGDEN: Yes, could it be ruled out as the determining factor, causing fibrosis other than asbestosis?

DR. RUSSELL: I don't think so.

DR. OGDEN: There was always free silica present in each one of those in varying degrees?

DR. RUSSELL: In practically all of them there was some silica present.

DR. OGDEN: It couldn't be ruled out as being the determining cause?

DR. RUSSELL: No doubt the presence of silica with other dusts help to produce fibrosis. The reaction to silica is much more violent than it is to other dusts.

DR. OGDEN: You didn't show any slides of true pneumoconiosis in which there was no silica, with the exception of asbestos, is that correct?

DR. RUSSELL: No, I don't think so. I think there was maybe one per cent in most of them. That brings us up to the point of mixed dusts. Practically all dusts are mixed. These tunnel workers were exposed to almost one hundred per cent silica; there were less of the other elements with it.

DR. OGDEN: That cement slide.

DR. RUSSELL: That was the finished product; about one per cent quartz.

DR. BELKNAP, Milwaukee, Wisconsin: I would like to ask if there is any known clinical functional test that you can give a man with a certain amount of pure silicosis by which you might determine his disability, say, he complained of a certain amount of dyspnea?

DR. RUSSELL: You mean like a tuberculin test?

DR. BELKNAP: No, to get an estimate of disability from pure silica?

DR. RUSSELL: I don't know of any, no. I think in estimating disability you have to take into consideration the whole picture, the man's history and his background, his present condition and the X-ray.

DR. BELKNAP: I meant definitely on dyspnea.

DR. RUSSELL: In our Vermont study we recorded, by use of the spirometer, the vital capacity. Our statistician said that we couldn't do much with it statistically, but it seemed to me that these men when they had silicosis, had a marked decrease in vital capacity and most dyspneic patients had a marked loss of vital capacity. One of the slides I showed you, I think the second one—his chest indicated quite a bit of pathology and he had one of the best vital capacities of the whole group, because he was the best golfer in that area; he had built up a reserve. You can practice with a spirometer and increase your vital capacity.

MR. WRIGHT: In the cases that come for litigation, the spirometer is of very little value because we need full and perfect cooperation of the patient in order to get true vital capacity. You spoke of response to exercise, didn't you, or did you? You spoke of chest expansion. Do you put any stress on the movements of the diaphragm?

DR. RUSSELL: Well, chest expansion and respiration is along the same line as result from the spirometer. It is more or less voluntary.

MR. WRIGHT: But I find that very often in examining patients they may know about vital capacity; they may not know what you are looking at when looking for the movement of the diaphragm in the dark room. They may not know what you are getting at. One thing is response to exercise and another thing is holding of the breath. I find when you don't get cooperation in any of the other tests a man may hold his breath for a normal length of time, which is a very valuable thing. In other words, wouldn't you use the same tests to determine the man's disability in this lung condition that you would in any other tests of his ordinary physical capacity?

DR. RUSSELL: I would. Exercise is quite valuable in determining vital capacity. I think it is really more accurate than the spirometer. I used to meet patients at a certain place and walk up the stairs with them to an office for X-ray and I counted their respirations at the bottom of the stairs and the top. It gave a rough index as to what we might find with the X-ray. The stairway had a landing half way up, and the more advanced cases of silicosis would stop there for a few extra puffs, and that is something they can't control. You can determine your own respirations before and after and compare them to his. I think it gave a fairly good index, and of course, other exercise is similar.

MR. DOE: Dr. Willis asked for an opportunity to correct a portion of what he said this morning. I would like to have him given that opportunity.

DR. WILLIS: This morning the question came up right at the last concerning the concentration of dust and dust counts which were the limits of safety. I think I said

that I would recommend five million. What I wanted to follow that up with was this: That is pretty nearly a compromise between Dr. Russell's data which he held at ten million, as I remember it, in his publication from the granite industry, and the earlier investigations in South Africa, which made it anywhere from five to eight million.

MR. DOE: Dr. Willis, when you refer to a safe limit are you assuming a certain silica content in the dust?

DR. WILLIS: That was a 35% level because there is really no other basis to go on except those two well established figures.

MR. DOE: Is there any method of getting a combination classification; for instance, we will say that five to ten million of 35% silica is safe from the standpoint of any hazard of contracting tuberculosis. Can we make any similar classification, doctor, with reference to other quantities of silica?

DR. WILLIS: You mean 50% or 10%?

MR. DOE: Exactly.

DR. WILLIS: Well, it certainly sounds like you could logically, but there is no established fact that you can. It is perfectly logical to assume that it ought to require a higher concentration of 10% silica to produce pneumoconiosis than it would a 50%, but aside from the range which Dr. Russell has shown, I don't know of any well worked out authenticated data.

MR. DOE: It is the general conception, is it not, Doctor, that the higher the silica content, the more rapidly the disease will occur?

DR. WILLIS: His work would certainly indicate that.

MR. DOE: But there is no definite graduated scale which one can apply?

DR. WILLIS: No, there is no definite graduated scale which one can apply.

MR. DOE: Was it your view that in the classes C and D where the incidence of tuberculosis was not above that

of rural Vermont, would you say that if one of those men exposed to the degree of concentration that was involved in those cases, which as I recall was sixteen million, had contracted tuberculosis, would there have been any means of attributing that to the industry, in your opinion?

DR. WILLIS: That is a hard question. I believe that unless you can illustrate that the concentration was sufficient to produce silicosis that it is only an assumption that that had a deleterious effect on tuberculosis. Again there is no absolute proof.

MR. DOE: Doctor, in your statement this morning and now, do you use in making a statement you just made, the terms silicosis and pneumoconiosis as interchangeable?

DR. WILLIS: Pneumoconiosis would in that case mean silicosis. Yes.

MR. DOE: Assume a dust which contains less than 2% silica. From what you have just said I assume that the concentration might be very much higher with safety, than it could be if the dust contained 35% silica. Can one classify dusts into silicotic and non-silicotic dusts by saying that a dust that contains as little as less than 2% dust falls into the non-silicotic groups of dust? Can you give us any guide as to the margin of safety in that group of dust containing less than 2%?

DR. WILLIS: We can only cite the data available for coal miners and cement workers. In other words, in coal miners the silica content is perhaps one and one-half per cent and in cement it varies from one to about five. In cement work the count is terrifically high. I think it was fifty million or so. When the silica percentage is low the dust count must be extraordinarily high to accord a hazard that a low count with high percentage would accord.

MR. DOE: Has any work been done with which you are familiar that gives any comparable result to the Barre study of silicotic dust, with relation to non-silicotic dusts?

DR. WILLIS: No, except these several pieces of work that Dr. Russell has mentioned.

MR. DOE: There are here a series of X-rays.

MR. TARRELL: I want to ask Dr. Willis his qualifications as a roentgenologist.

MR. DOE: I will ask you Dr. Willis, how many chest films do you see annually?

DR. WILLIS: I suppose about 3500.

MR. DOE: You are the director of a tuberculosis sanatorium at present?

DR. WILLIS: I am on the staff.

MR. DOE: How long have you been engaged in tuberculosis work as a specialty?

DR. WILLIS: Well, I should say since 1920.

MR. DOE: What did you do at Johns-Hopkins relating to this subject?

DR. WILLIS: Well, I did a fair amount of experimental work on the question of anthracosis and silicosis in animals and I also, from time to time, saw clinical cases of pneumoconiosis, and I wrote a review on the subject of pneumoconiosis, particularly as it relates to tuberculosis. As I said, I don't mean to qualify as an expert witness in X-ray work. I thought this was just a question of opinion.

MR. DOE: Yes. Now, Dr. Willis, what were the conclusions that you reached on the experimental work you did on coal miners?

DR. WILLIS: That inhalation of coal doesn't have any appreciable effect within the limits of experiments; it does not produce any fibrosis of the lung and in animals so exposed are not more susceptible to tuberculosis than other animals not so exposed.

MR. DOE: Your other study?

DR. WILLIS: The other study was a study of silicon carbide produced by the factory which Dr. Clark represented yesterday. That was an experiment in which animals were exposed to inhalation of dust over three years and the results were essentially the same as those with the inhalation of coal dust. I didn't know that this was going to be taken as actual evidence in the case.

MR. TARRELL: Before I consent to have your statement I want to ask some more questions.

MR. DOE: I thought this might be of great interest to have him tell us.

MR. TARRELL: I thought I heard Dr. Willis say this morning, or two of the other doctors, that from X-ray alone you could not reach a conclusion. Now, I understand you propose to submit an X-ray and have him reach a conclusion on that.

MR. DOE: No, I haven't asked him anything about any films yet. Now, with reference to this man, Bruno Rhode, his age was 42, his exposure was six years, the silica content of the abrasive wheels which he used as a grinder was 1.5% and the concentration was 1.1 millions; this man undoubtedly has an active pulmonary tuberculosis. Now, have you anything to say whether there is any evidence on that picture of dust inhalation? That is one of the series which cover an interval of roughly one year—if you care to you may look at the entire series.

DR. WILLIS: In the first place, there is quite a good deal of diffuse fibrosis throughout the lung. There is evidence of spotty infiltration in several places with questionable cavitation which would certainly indicate a tuberculosis. These diffuse fibroses and shadows are not the common finding in an ordinary tuberculosis. How old is the man?

MR. DOE: Forty-two.

DR. WILLIS: He has been occupied at the job six years?

MR. DOE: As a grinder.

DR. WILLIS: Before one could pass on that one would certainly want to know a good deal more about the man's previous occupation and previous history, that would all come in. The fact is as Dr. Russell brought out, from the film alone you cannot arrive at an ultimate conclusion, because it is one of several bits of evidence, but it looks like there is tuberculosis there. There is this interesting fact too, that these shadows here in the course of a year haven't changed as much as you might have anticipated if that were tuberculosis superimposed on a silicosis. The

two films are not quite comparable and that brings up also the question of absolute reliability of films.

MR. DOE: I show you another film, Dr. Willis, this was taken on March 10, 1932.

MR. TARRELL: Which case is that, Mr. Doe?

MR. DOE: This is the Prahl case.

DR. WILLIS: In this first film there is very definite evidence of tuberculous infiltration with cavity in the left upper and with quite a bit of diffuse areas of presumably tuberculosis. In a film like this here there might have been at an earlier time, some other markings, but the tuberculosis has supervened. It is awfully difficult to pass on the other markings and I wouldn't want to make an absolute statement as to whether all this below and on the other side is tuberculosis or is an occupational infiltration.

MR. DOE: That is all.

MR. TARRELL: No questions.

MR. DOE: I would like to ask Dr. Gardner some more questions. I would like to ask you, Dr. Gardner, whether in the case of inhalation of dust containing less than two per cent silica, do you believe you can assist us any on the question of whether there is any necrosis such as described in the silicotic nodules?

DR. GARDNER: In none of our studies has there been any necrosis except in the case of free silica.

MR. DOE: Does that have any bearing on the conclusion of the relationship between non-silicotic dusts and tuberculosis in your opinion?

DR. GARDNER: It has always been my belief that the necrosis produced by silica was responsible for the susceptibility to tuberculosis. That is at the present time a hypothesis, however, and one on which we are working to attempt to prove this association.

MR. DOE: That is all.

FRED M. WILCOX, *Chairman*, Industrial Commission of Wisconsin: To have had an opportunity to give intensive and sustained consideration to a subject of this kind as we have done is just another demonstration of how much better it is than to have to submit these issues in court to a jury made up of the butcher, the baker and the candlestick maker. I need not say more. I urge upon you who represent industry, you who represent insurance carriers and all those who are interested in the administration of workmen's compensation to keep in touch with the men who have been here, and to remember the counsel of Mr. Cummings and Mr. Bloomfield and the doctors as to the many things we ought to have in mind when we make our studies. When we do undertake a study let's tabulate everything that is available.

And there is another thing that I wish to impress upon you because of the delicacy of the situation. Families of deceased workmen do not want post mortem examinations. After all when death has overtaken an employe who has been exposed to a condition which may or may not produce silicosis, or perhaps a superimposed tuberculosis, we should do our best to dissuade the family from the feeling that they have and prevail upon them in the interests of a better understanding of this whole subject to consent to a limited post mortem examination,—at least of the lungs. Too often when these matters are in issue we play on the feelings of the families to induce a settlement of a case by compromise. Rather than go through the post they settle. It should be approached from an entirely different point of view,—the interest of all in a better understanding of the effects of our employment. We have just got to take a firm, yet sympathetic stand.

Thanks again for your attendance and your contributions.

Appendix A

BIOGRAPHICAL SKETCHES

Dr. Leroy U. Gardner was graduated from Yale College and from the Yale School of Medicine, receiving degrees of B. A. and M. D. in the years 1912 and 1914 respectively. He spent three years in post-graduate study of pathology under Dr. F. B. Mallory at the Boston City Hospital, and during the last two years of this period he was also ranking instructor in pathology at the Harvard Medical School. He then accepted a position as assistant professor of pathology at the Yale School of Medicine, when it was being reorganized by Dr. M. C. Winternitz. After a brief period of teaching there, it was necessary for him to come to Saranac Lake. As soon as his health would permit, he became associated with the Saranac Laboratory for the Study of Tuberculosis. In 1919 they started experimental investigation of the study of the inhalation of dust as a predisposing factor in the production of tuberculosis. These studies have been in progress since that time. In 1928 he was made Director of the Saranac Laboratory, and since that time they have expanded their work in the field of pneumoconiosis, associating themselves with others in various parts of the country who have been similarly interested. Today the whole Laboratory is organized for the investigation of this problem and they are devoting the major portion of their time to it.

Mr. Donald E. Cummings was graduated from the U. S. Military Academy in 1920 and entered a School for the Instruction of Officers, as a first lieutenant, immediately following his graduation. He remained in this school for one year, at the end of which time he resigned and entered the Massachusetts Institute of Technology. He was graduated from this institution in 1923 with the degree of B.S. in chemical engineering. He then accepted a position as instructor in the Massachusetts Institute of Technology under Dr. Warren K. Lewis. It was then necessary for him to come to Saranac Lake and after recovering his health he became an instructor in physics and chemistry

in the high school at Saranac Lake for two years in order to assure his complete recovery. Early in the year 1928 he became associated with Dr. Gardner in research work, dealing with pneumoconiosis in the Saranac Laboratory. He has been made Assistant Director of the Saranac Laboratory and has continued research work in pneumoconiosis until the present time.

He has been a Consultant in the U. S. Public Health Service under Dr. Leake, and at present, an instructor in the Trudeau School of Tuberculosis. He is a member of the National Tuberculosis Association, American Chemical Society, and a special committee of the American Society for Testing Materials. He has had an opportunity to make several field investigations in industries having a known dust hazard.

Dr. H. S. Willis graduated from the University of North Carolina in 1914, obtained his M.D. at Johns Hopkins University in 1919, and his M.A. at the same University in 1920. From 1919 to 1922 he was Assistant and Instructor in Medicine; from 1922 to 1928 was Associate in Medicine; from 1928 to 1929 was Associate in Clinical Medicine; from 1929 to 1930 was a lecturer in Clinical Medicine at Johns Hopkins University. In 1929 Dr. Willis was placed in charge of Dows Tuberculosis Laboratory of the Johns Hopkins Hospital, and during the period of his connection with the University was Assistant Visiting Physician and Dispensary Physician at the Johns Hopkins Hospital. From 1928 to 1930 he also conducted a general practice in the City of Baltimore. In 1930 he came with the Wm. H. Maybury Sanatorium, which is the Detroit Municipal Tuberculosis Sanatorium, at Northville, Michigan, as a pathologist. He has done a volume of clinical work in internal medicine and tuberculosis, and several years of experimental work on tuberculosis and pneumoconiosis. He has made numerous publications on these subjects in the American Review of Tuberculosis and in the magazine Medicine. At the present time he retains his connection with the Maybury Sanatorium, where he is still pursuing clinical work in pneumoconiosis in conjunction with his other duties.

Dr. W. Irving Clark received his medical education at Columbia University, New York City, where he received the degrees A.B. and M.D.

Following this he served as interne at the Roosevelt Hospital, New York City, from 1904 to 1906.

Moving to Worcester, he became connected with the Worcester City Hospital, where he worked on tuberculosis in the out-patient department and later became the Secretary of the Worcester Tuberculosis Relief Association which carried on active field work among the tubercular patients in Worcester.

He was later appointed assistant surgeon at the Memorial Hospital, Worcester, and in his private practice he did considerable chest work, dividing his time between this and general surgery.

During the war he served overseas with the American Red Cross and later as Captain of the Medical Corps of the United States Army.

In 1911 he was appointed in charge of medical service of the Norton Company, Worcester, a large manufactory of artificial abrasives, grinding wheels and grinding machines. He has been connected steadily with this institution until the present time and has made a special study of the effects of the inhalation of abrasive dusts on the lungs of the workmen. These studies have been published in four papers on the Dust Hazard in the Abrasive Industry.

Dr. Clark has also written an article on the effect of inhaling artificial abrasive dusts for Occupation and Health Encyclopedia of Hygiene, published with the International Labor Office, Geneva, Switzerland. He has also published an article on Industrial Medicine in the Oxford Medicine, and a similar article for the Nelson Loose Leafe Medicine.

Since 1919 he has been connected with the Harvard School of Public Health, first as an instructor in Industrial Medicine, and for the last two years as assistant professor in the same subject.

Mr. Bloomfield was graduated from the University of New Hampshire in 1920 with the degree of Bachelor of Science in Engineering, and immediately after graduation

became affiliated with the United States Bureau of Mines at the Pittsburgh Experiment Station. During the three-year period at the Bureau of Mines, Mr. Bloomfield conducted extensive studies on problems related to toxic gases, fumes and dusts. A considerable portion of this time was devoted to the development of protective devices for use in contaminated atmospheres.

From April, 1923, to the present date, Mr. Bloomfield has been attached as Sanitary Engineer to the Office of Industrial Hygiene and Sanitation of the United States Public Health Service, devoting his entire time to problems dealing with the industrial environment as related to its effect on the health of the worker. The health hazards associated with radium dial painting, lead storage battery manufacture, the use of tetraethyl lead gasoline, chromium plating, and the pneumonia problem in the steel industry are a few of the problems which Mr. Bloomfield has been engaged on during the past ten years. The largest portion of his time, however, has been devoted to studies of the dust hazard in various industries. Mr. Bloomfield has conducted detailed studies of the dust exposure of workers in connection with every one of the many dust studies carried out by the United States Public Health Service, and is co-author of the Bulletins issued to date on the studies of the health of workers in the cement and granite cutting industries. He was also affiliated on the study of the dust hazard in the sandblasting industry, which was conducted jointly by the United States Public Health Service and the National Safety Council.

In addition to the studies concerned with the nature and concentration of dusts in industry, Mr. Bloomfield has also made many studies on the efficiency of dust removal devices and other equipment used for the protection of the worker.

Mr. Bloomfield is author or co-author of more than 30 papers on various subjects pertaining to health hazards in industry.

Following the graduation of Dr. Albert E. Russell from Medical School he became Resident Physician of the Waverly Hills Sanatorium at Louisville, Kentucky, in 1918,

which position he held until 1919. He then entered the employment of the United States Public Health Service as a member of the Staff of Service, Hospital No. 26, at Greenville, South Carolina, where he remained in service, specializing in the treatment of tuberculosis during 1920 and 1921. From 1922–23, inclusive, Dr. Russell acted as Assistant Chief of the Tuberculosis Hospital Section of the U. S. Veterans' Bureau. From 1924–30 he was in charge of field studies as to the health of workers in dusty trades for the United States Public Health Service. In 1930 he was appointed as official representative of the United States at the International Silicosis Conference at the League of Nations at Johannesburg, South Africa, of which he acted as Vice-Chairman. In 1930 he became Chief Surgeon of the U. S. Bureau of Mines.

Dr. Russell has made numerous contributions to medical magazines, and has written many reports for the U. S. Public Health Service, covering particularly the field of pulmonary diseases and more specifically those diseases resulting from the inhalation of dust.

Appendix B

LIST OF PERSONS WHO ATTENDED CONFERENCE CONCERNING EFFECTS OF DUSTS UPON THE RESPIRATORY SYSTEM HELD BEFORE THE INDUSTRIAL COMMISSION OF WISCONSIN AT CHICAGO, ILLINOIS, ON NOVEMBER 16-17, 1932.

Name	*Address*	*Profession*	*Business Connection*
Andrews, Asa A.	Chicago, Ill.		Lumbermens Mutual Casualty Co.
Atkinson, F.	Milwaukee, Wis.		Liberty Mutual Casualty
Augst, R. A.	Montreal, Wis.		Montreal Mining Co.
Banyai, Dr. A. L.	Milwaukee, Wis.	Physician	Clinic Director, Muirdale San.
Belknap, E. L. M. D.	231 W. Wis. Ave. Milwaukee, Wis.	Medical Director	Globe Union Mfg. Co.
Bellis, Dr. G. L.	Wauwatosa, Wis.		Supt. Muirdale San.
Berlin, Dr. D. S.	Chicago, Ill.		Augustine Hosp.
Biever, E. J.	Kohler, Wis.	Mech. Engineer	Kohler Company
Bloomfield, J. J.	Washington, D. C.		U. S. Public Health Service
Britton, Dr. J. A.	Chicago, Ill.	Medical Director	International Harvester Co.
Brown, Wm. E.	Milwaukee, Wis.	Lawyer	Allis-Chalmers Mfg. Co.
Clark, Dr. W. Irvine	Worcester, Mass.	Physician	Norton Company Harvard School
Cottingham, M. D.	Kohler, Wis.	Medical Director	Kohler Company
Cummings, D. E.	Saranac Lake, N. Y.	Ass't Director	Saranac Lab.
DeBlois, Lewis	New York, N. Y.		
Dickson, L. E.	Chicago, Ill.		Standard Equipment Co.
Dobbins, Dr. Thos.	Kenosha, Wis.		Nash Motors Co.
Doe, Arthur B.	825 N. Broadway, Milwaukee, Wis.	Lawyer	Employers Group
Earlywine, J. L.	Chicago, Ill.		
French, G. E.	Chicago, Ill.		Liberty Mut. Cas.
Ford, Dr. W. B.	Milwaukee, Wis.	Physician	Ass't Chief T. B. Div. Health Dept.

Name	*Address*	*Profession*	*Business Connection*
Gandrey, Alfred R.	Milwaukee, Wis.	Lawyer	Ass't City Atty.
Nowak, C. A.	Chicago, Ill.		Ill. Indus. Com.
O'Malley, Dr. T. S.	238 W. Wis. Ave., Milwaukee, Wis.	Surgeon	
Ogden, Dr. C. H.	Chicago, Ill.		Ill. Steel Co.
Otjen, C. J.	Milwaukee, Wis.	Attorney	Liberty Mutual Ins. Co.
Parrish, L. J.	Milwaukee, Wis.		A. O. Smith Corp.
Pierport, Dr. D. C.	Ironwood, Mich.		Pickands, Mather Co.
Reid, M. A.	Hurley, Wis.		Montreal Mining Co. & Odanah Iron Co.
Reynolds, Paul F.	Cleveland, Ohio		Montreal Mining Co.
Ringo, Dr. H. F.	Montreal, Wis.		Montreal Mining Co.
Russell, Dr. A. E.	*Washington, D.C.*	*Surgeon*	*U. S. Public Health Service*
Sander, O. A.	800 Empire Bldg., Milwaukee, Wis.		Employers Mut.
Sappington, Dr. C. O.	Chicago, Ill.	Consultant Ind. Med.	
Schlomoviat, W. Benj. H.	210 Empire Bldg., Milwaukee, Wis.	Internist	
Sexton, James M.	Milwaukee, Wis.		Employers Mut.
Story, H. W.	Milwaukee, Wis.		Allis-Chalmers Mfg. Co.
Tarrell, L. A.	Milwaukee, Wis.	Attorney	
Tharinger, E. L.	Milwaukee, Wis.	Pathologist	
Warfield, Dr. Louis M.	Milwaukee, Wis.	Internist	
White, Wm. W.	Cleveland, Ohio		Montreal Mining Co.
Wilcox, F. M.	Madison, Wis.	Chairman	Wis. Indus. Com.
Willis, H. S.	Detroit, Mich.		Detroit Public Health Dept.
Goldschmidt, W. J.	Milwaukee, Wis.	Attorney	
Graves, Dr. S. S.	Chicago, Ill.	Medical Director	Ill. Indus. Com.
Gray, A. W.	324 E. Wis. Ave., Milwaukee, Wis.	Medical	Private Practice

Name	*Address*	*Profession*	*Business Connection*
Guillert, F.	Chicago, Ill.		Eye Shield Co.
Guire, Peter, Jr.	Chicago, Ill.	Commissioner	Ill. Indus. Com.
Habbe, Dr. J. E.	Milwaukee, Wis.	Roentgenologist	
Hensel, H. G.	Chicago, Ill.	Safety Director	Youngstown Sht. & Tube Co.
Huth, Gordon C.	Chicago, Ill.		Universal Atlas Cement Co.
Ireland, Walter	Kohler, Wis.	Employment Mgr.	Kohler Co.
Janzer, W. W.	Milwaukee, Wis.		Seaman Body Corp.
Knutson, R. G.	Madison, Wis.	Member	Wis. Indus. Com.
Kuechle, B. E.	Box 32, Wausau, Wis.	Insurance	Employers Mut.
Kuhn, Dr. Leroy P.	Chicago, Ill.		Lumbermens Mut. Cas. Co.
Lavich, J. L.	Chicago, Ill.		Lumbermens Mut. Cas. Co.
Lotz, Oscar	324 E. Wis. Ave., Milwaukee, Wis.	Medical	Private Practice
McIntyre, M. D.	Cleveland, Ohio		Pickands, Mather Co.
McLaren, Dr. J. B.	Appleton, Wis.	Physician	Chief Surgeon, Kimberly-Clark Corp.
Mellum, H. J.	Kenosha, Wis.		Nash Motors Co.
Miloshavich, Dr. Edw. L.	Milwaukee, Wis.	Pathologist	
Nelson, Harry A.	Madison, Wis.	Director, Workmen's Comps.	Wis. Indus. Com.
Wrabetz, Voyta	Madison, Wis.	Member	Wis. Indus. Com.
Wright, C. B.	Minneapolis, Minn.	Internist	
Zinn, R. P.	Ironwood, Mich.		Pickands, Mather Co.