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INDUSTRIAL COMMISSION
OF
WISCONSIN

Proceedings of Conference Concerning

EFFECTS OF DUSTS UPON THE RESPIRATORY SYSTEM

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

CONTENTS

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. C. 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. Farrell 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,

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.

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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

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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.

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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

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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

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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. 182 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

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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,

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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

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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-

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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.

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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	3.80

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-

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	0.84
Water and Loss	2.49	3.30

Heffernan, Journal of Industrial Hygiene, November, 1926.

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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

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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

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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.

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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.

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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.

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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

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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.

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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

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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.

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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.
Utjen, 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.	
Schlomovist, W. Benj. H.	210 Empire Bldg., Milwaukee, Wis.	Internist	
Serton, 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.
Hahbe, 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.
Milosiavich, 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.