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PNEUMOCONIOSIS IN COAL MINERS

Chairman: PHILIP DRINKER, Sc. D.

Industrial Hygiene Studies of Coal Miners in Two Geographical Areas in the United States

E. D. J. Urban

Discussion

Radiological Classification

Charles M. Fletcher, M. D.

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Arthur J. Vorwald, M. D.

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Epidemiology of Coal Workers' Pneumoconiosis in Wales

Charles M. Fletcher, M. D.

Discussion

BY DOCTOR VORWALD:

It's past our time to begin and there are a few announcements which I should like to make before the formal opening of the morning session.

Again, to remind you, those of you who have not registered, please do so at the John Black Room. Also, tomorrow night, on Wednesday, at eight o'clock, there will be a demonstration of colored photography by the Ansco Company in the main ballroom of the Saranac Lake Hotel. Those of you who are interested in color photography, I know, will be interested in what they have to present. Also, there are a number of you who are Rotarians and today being Tuesday, the Rotarians will meet this evening at the Fish and Game Club at six o'clock. The Fish and Game Club is on the outskirts of Saranac Lake and should you inquire, I'm certain that you will find your way there. It sounds very interesting when the Rotarians meet at the Fish and Game Club instead of at noon time at the hotel, because it always means something extraordinary in food and other things, so if you wish, you're welcome to go there, to make up your Rotary.

The banquet on Thursday night, as you will note from your agenda, there is to be a banquet on Thursday night, and I wish to announce that ladies are cordially invited to the banquet. There will be a good many there, so those of you who are accompanied by a lady, please bring them.

With that then, we will enter our - or begin our sessions for this morning, and our Chairman certainly needs no introduction to you -- Professor Drinker, who is Professor of Industrial Hygiene at Harvard. Doctor Drinker.

BY DOCTOR DRINKER:

Doctor Vorwald, Ladies and Gentlemen: I'd like to make it clear to those whom I don't happen to know personally that I am not a physician and if questions should, by any chance, come my way which are medical, I take second place to no one in the matter of evasion and I will evade them and pass them along to somebody else.

The first paper this morning is by Mr. Urban of the Laboratory here, on Industrial Hygiene Studies in Coal Miners in Two Geographical Areas in the United States.

BY MR. URBAN:

Coal is a sedimentary rock which, because of its high energy value, has been and is of outstanding importance to man. Coals occur in all geologiacl ages since the Devonian period about three hundred million years ago. World-wide distributions of coal originated in the and in the periods and in the later tertiary period, most of the lignites or low ranking coals of the world were formed.

The extensive distribution of the individual coal seams implies swamp accumulation on broad deltas, coastal plains and broad interior low lands where shallow waters rest throughout the year. All ordinary coals are of vegetable origin. In the sedimentary cycle of coal, the immediate source of materials were the prolific plant tree growths and the carbon dioxide which was drawn from the surrounding atmosphere and surface waters.

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The trees and plants which fell into the swamp waters underwent partial decay and the residue accumulated on the bottom of the swamps to form peat, which is the first stage of coal, all coals. It has been estimated that from 125 to 150 years of time of accumulation were required to produce the equivalent of one foot of bituminous coal. From 175 to 200 years were required to produce one foot of anthracite coal. An essential to coal formations is the arrest of bacterial decay in the submerged vegetable matter before complete destruction t akes place, so that there is some residue to accumulate. This is brought about from the decay-promoting bacteria to render the water toxic to themselves.

The type of coal eventually formed depends on the environment, the kind of plants and particularly on the duration of the bacteria decomposition. May we have the first slide, please?

In non-technical terminology, four main types of coal are recognized, anthracite or hard coal, bituminous or soft coal, canel, which is actually a special type of bituminous coal, and lignite. These types may be further subdivided into ranks. Each rank designates coal of specific energy values and chemical and physical properties, semi-anthracite, intermediate coal between anthracite and bituminous; there are actually five different kinds of bituminous -- sub-bituminous and brown coal are intermediate between bituminous and lignite, andhracite being termed as the coal of the highest type and lignite the lowest type.

Peat is a fuel. It's not a coal, but a mixture of partly decomposed vegetable matter in which bacterial action, for the most part, has been interrupted. Subsequent physical and chemical changes produce coal of the above ranks. Where these changes have reached completeness, graphite is the result.

Physically -- rather, chemically, coals contain hydrogen, carbon, oxygen and impurities either in simple elements or in complex combinations. The chemical changes which occur when the higher rank coals are formed from peat are the progressive elimination of water, oxygen and bituminous - either the solid and semi-solid hydrocarbons, the increase in carbon as fixed carbon in volatile matter, the consolidation of hydrogen development, heavy hydrocarbons, and an increased resistance to solvents, oxidation and heat.

Some of the physical changes which may occur in coal formed from peat, in the highest ranks, concern the by-products of those coals of the higher ranks which contain lignose, gums, waxes, oils, resins and fats. Those resins and waxes which are less assailable by bacterial action are present in most coals.

Coal beds occur within so-called coal regions which consist of alternating beds varying in thickness, of sandstone, shale, clay, almost -- mostly of fresh water origin. A coal measure generally contains several coal seams separated by sedimentary depositions of these various minerals.

In Pennsylvania, for example, there are 29 coal seams aggregating 106 feet of coal with beds varying in thickness from a mere film up to 100 feet. The famous Maimot seam is fifty to sixty feet thick. In Alabama, there are fifty-five coal seams. In England, the aggregate thickness of coal is 85 feet and in Germany 120 feet.

Now, during the past several years, the Saranac Laboratory has conducted industrial hygiene surveys in a number of underground coal mines located in various coal fields throughout the eastern section of this country. Data from such - from six such surveys has been selected for presentation. Each of these mines extracted a bituminous or soft coal from a different coal seam, with the exception of two mines, five and six which I'll show you later which worked in opposite ends of the same seam.

They ranked in size from employment of about 300 to 1,000 men. The deepest mine was about 600 feet below surface. All of the mines were entered either through vertical shafts or inclined slopes. Modern methods of mechanized mining were followed in each case, utilizing both track and trackless type of equipment. A limited amount of hand work, hand shovelling into conveyor pans was also done.

All the mines were generally regarded as gaseous.

Considerable attention in each case was, therefore, given to safety, to ventilation and dust control. Now, for these reasons, data from the - from these six mines can be compared.

It is our further belief that some of the conclusions derived from these studies can be employed also through other coal mines, even coal mintes of the anthracite type. Our primary concern in these surveys was for hazards of a respiratory significance. The nature and effect upon man of the gasses commonly encountered in bituminous coal mines, gasses like methane, carbon dioxide, carbon monoxide and oxygen-deficient air are rather well established. Our investigations were directed mainly, therefore, to a study of the air-borne dust, to which the workers were exposed.

May we have the first slide, please? Now, early in our investigations, it became apparent that a number of different minerals would be present in varying proportions throughout the same coal seam. A method was developed by us for the collection of air-borne dusts for chemical analyses. It consisted of a small one half horsepower United States Bureau of Mines approved suction motor set, connected to a paper filter by a two-inch diameter smooth ball hose. The paper was enclosed in a holder which was elevated to breathing level height by a tripod. The apparatus was completely portable and was used wherever electric power was available.

In operation, dust-laden air is drawn into the filter. The dust is retained and the clean air passed on through to the fan. This is your fan motor set; this is your hose; this is your filter paper holder. We just set it on an ordinary engineer's tripod so that we can carry it around at the same time keep it up in the air, and the filter paper holder section is here. It was a simple metal affair. It had a screw cap on it and the filter paper was held in position there tightly.

Now, we're not sure about the efficiency of collection of this device, although we know it's very high. Through experience, we have learned, for example, that we can run this thing all day in a coal mine, collection a -- collect a deposition on the filter which would be on this side, heavy enough to completely fill up all the cleats, and yet when we take the filter paper off, the back of the filter - the color of the back of the filter hasn't changed at all, that is if any particles did pass through, there were very few of them, so that the paper must be an efficient collecting device.

The device was employed for collecting the air-borne dusts from coal face operations, from workings in rock and during the coal train haulage. Figure two illustrates a typical section through a coal formation of two of the mines surveyed. This particular bed was the deepest of three coal seams which were being mined at the time in the same coal field or coal measure. In other words, three operators, three other beds had different elevations in the same geographical area and were being mined; this just happened to be the deepest.

The roof above the coal bed was a mass of sandstone deposits separated from the coal by a narrow layer of top slate. Lying beneath the coal were deposits of other sediments as coal slate, fire clay and sandstone. In addition the coal bed itself contained several other sedimentary depositions, termed locally as binder seams. This is your main sandstone area roof. We have a top slate layer here, some mines this wasn't present and others it was present. Some places, geographically, we called it - in some places it was called broceli.

This is your coal face or coal bed. Here are other sedimentary depositions within the coal face itself or rather inclusions of sedimentary materials. Actually, while we consider that this is mined as a single coal bed, actually, we have one, two, three, four coal beds representing different sedimentary cycles of coal formation, because each one of these binder seams was lain down after the lower coal seam was deposited.

In the other coal mine study, the respective coal seams were similarly enclosed by sedimentary deposits of varying thickness and minerological composition. In some coal beds, the binder seams were less prominent; in others, however, the coal seams included, in addition, deposits of Golden Bright, again these local terms, and rock layers as much as eight layers in thickness.

In the mine represented by this coal face, bulk samples of each of the various rock horizons, both enclosing the coal and included in the seam, were taken for chemical analyses. All contained free silica.

In the sandstone, the analysis, the average analysis of several samples showed it contained sixty-seven percent free silica, the top slate twenty-one and a half, the binder material which again was a sedimentary deposit, probably a shale material, contained forty percent free silica. The coal itself was taken very - quite a number of samples - and the average of these samples was only three tenths of one percent free silica. Coal slate and slate - the two in this particular case were so intermixed that we couldn't separate them and the combined analysis, the combined analysis showed thirty-one point one percent, the fire clay 39.2 percent silica, the sandstone similar to that, so all of these sedimentary depositions other than coal contained appreciable amounts of free silica.

Chanel samples were obtained from across the entire coal face from the roof to bottom and showed free silica samples ranking from 1.5 to 7.2 percent. The average of thirty samples was 8.3 percent silica. The air-borne dust created by all operations during the complete day's shift was collected on filter paper. The free silica values of these samples, however, ranged from three tenths of one percent to seven tenths of one percent with an average of five tenths percent.

The chanel sample was simply a run or cut of the roof of uniform size directly across from the top to the bottom and collecting all the droppings that came out of that room and analyzing those for silica. These results indicate that although silicious materials were associated with the coal bed, most of the dusts created by the coal face operations originated from the coal itself.

- We can see that pretty well even though we do have the silica-bearing materials here, the air-borne dust only contains half of one percent free silica.

May I have the next slide, please? To illustrate this point, still further, table Two shows a comparison of the petrographic analyses of three different types of sampling obtained from the same coal face. A quartz content of the coal only in this particular case was a hundred to one percent. A chanel sample from the entire coal face showed three percent silica and a filter paper collection of air-borne dust from all coal operations revealed but three tenths of one percent silica. When this first sample was obtained, we instructed the man that was doing the drilling with a drill, to drill into as clean a coal area as he possibly could and then we collected all the dust from the drilling and the quartz content of that dust was a hundred and one percent, -- rather, one hundredth of one percent.

Undoubtedly, for the proper evaluation of the silica hazard in coal mining, analyses should be made of the air-borne dusts rather than of the parent materials.

Dust conditions to which men are exposed for coal mining can be divided for study into three general categories: The dust divisions related to operations of the coal face, those existing in coal working sections which are adjacent to the coal face and those present in other areas of the mine, through which men must travel or in which general work is done.

May we have the next slide, please? Table 1 shows the average concentration of dustiness associated with common coal face operations in six bituminous mines. 250 samples are summarized in this table. One of the first standards for permissible levels of dustiness for silica, applied in this country, judged that if in any given dust concentration less than five million particles per cubic foot of silica were present, the condition was considered safe. Recently, the United States Conference of Industrial Hygienists have proposed that no permissible concentration of any dust should be denser than fifty million particles per cubic foot. Following this, the United States Bureau of Mines suggested a maximum weighted average dust level for coal mines of twenty million particles per cubic foot based on a work shift exposure.

Now, the first standard has some experimental basis. The other two, in part, are attempts to establish standards of good practice. In mines No. 1, 2, 3 and 4, all the coal face operations where wet methods were used, the dust conditions were probably with one exception, complied with, or they complied with all of the above standards. That exception is this mine - this 94.0 figure. These dust counts are all in millions of particles with the particles being all less than ten micron per cubic foot, so this 94.0 figure, if it was properly weighted by the time required to drill the five or six holes in the face, which ordinarily was about a half an hour, it was probably weighted against the number of times that the man had to drill these holes in an eight-hour shift, would probably be less than fifty million particles per cubic foot, probably closer to that.

No air-borne dust samples were collected in these first four mines. We collected bulk samples but not air-borne samples. From other observations, however, from a comparison of bulk samples from one, two, three and four mines, with five and six mines where we had air-borne sampling, the silica content was probably but slightly higher than that reported in five or found in five and six.

Now, dusty conditions were experienced in Mine Number Five and still higher dust conditions were found in Mine Six, even when using water. Based on silica content, however, the highest count taken was 3.3 million particles per cubic foot of quartz, that is even this figure of 672.3 million, with cutting dry, Mine Number six, that's total dust - the free silica count in that was only 3.3 million particles per cubic foot.

In Mine five, and particularly Mine Six, the coal was much more gracile, it was much more dry and gave rise to more dust than did the coals of any of the other mines studied. These results indicate that in most bituminous coal mines, dust conditions of good order can be maintained. In exceptional cases, however, additional precautions are required to prevent dust levels from approaching a silica hazard.

You can see in this dust count, this dry operation of cutting went up two hundred million particles higher, that we would approach an equivalent of fifty - of five million particles of silica present. We would be introducing a silica hazard simply from the silica which was present even though at low concentration.

Table three, please. The next slide, please. Levels of dustiness at coal section operations other than those at the coal face are shown in Table 3. In Mines 1, 2 and 3 and 4, in this case also Mine 5, conditions were maintained in good order;although there were - they were moderately high in Mine 6, the silica count was low.

By shunting car cycle, we mean that we went - we travelled with the operator of this particular car. The car itself represents a - looks like a large shallow pan on four wheels, and he travels from the loading station in the coal face to the dumping station into the mine cars through the - through the coal section itself, and our sample was taken during the course of this complete cycle. When the shuttle car arrives at the dumping station into the coal cars, the coal was unloaded.

This particular operation gave rise to, in some instances, to more dust than in other parts of the operation of the shuttle car cycle. You can see here 127.8 for that particular operation alone; that 127.8 is part of this 163.5. The general air in the roadways was pretty clean although in Number 6 mine, conditions were ordinarily dusty throughout, and also dusty in the roadways.

The next slide, please. Dust counts taken in intake gas supplies to the six mines showed consistently low values. It is in some of these airways that men are transported to and from their work places and where coal car haulage is done. In some of the mines, sand was used on the tracks to give the locomotives additional traction.

Several air-borne dust samples were collected on filter paper in these mines, from the position of the motor man during the course of an entire shift's operation. The levels of total dust and of silica in each instance were within permissible limits.

This (next slide) represents the area in the mine frequently travelled, where car haulage is done and the area of the mine where the work - where the workers spend possibly an hour or an hour and a half of his day just in the course of transportation.

The next slide, please. Table five is a summary obtained by the United - obtained by the U. S. Bureau of Mines in sampling the roof blocks common to fifty-one coal mines in nine eastern states. It was presented by James Westfield at the American Mining Congress meeting in 1951. It shows that more than ninety percent of the roof blocks in these mines were shale and sandstone, averaging 26.5 and 55 percent free silica respectively. These values are in accord with the findings of our survey.

Unquestionably, a definite silica hazard exists in coal mines when work is done in rocks other than coal. Shafts or inclined slopes are driven at times which required penetration in the rock. In some mines, rock falls and falls of roof rock must be removed in the course of operation. Roof folding is a new practice which necessitates the drilling of deep holes in rock. When such work is done dry, resulting dust levels are high.

It has been demonstrated conclusively, however, in other rock mines and in tunnel work, that levels of permissible dustiness can be maintained by the proper use of water and ventilation. Some of the coal mine surveys follow this practice. Other mines, although they drill dry and work in rock dry, provide the men with filter respiratory equipment.

In conclusion, it must be remembered that these methods are in respect to modern day practices of mining. It can be applied only as changes and practices remain essentially unchanged. In any occupation, the occupational dust conditions to the exposed worker, the history of the mine as well as the history of the man must be taken into consideration. Disease conditions may well be the end results of dust exposures which he has experienced previously. Thank you. (Applause).

BY DOCTOR DRINKER:

We will try to limit the discussion on this paper to about five minutes, because we're a little bit behind time. I'd like to ask Mr. Urban one question. Are you bothered by the bits of paper interfering with those samples in petrographic analysis when you use that completed filter?

BY MR. URBAN:

No, we have pretty good success with it and Doctor Durkan is here, I think, and can tell you about it. We have analyzed very small papers successfully.

BY DOCTOR DRINKER:

Are those the filters that are used in the Chemical Warfare Service Mass classification, you get it from the M. S. A.?

BY MR. URBAN:

I'm not sure about that, get it from M. S. A.

BY DOCTOR DRINKER:

That's the type we use.

BY MR. URBAN:

The only difficulty we had was trying to sample fields that were a little high in relative humidity, the paper getting wet, but where the humidity was less than say ninety percent, we didn't have too much trouble.

BY DOCTOR DRINKER:

Doctor Fletcher?

BY DOCTOR FLETCHER:

I'd just like to ask one or two questions, make one or two comments on this most interesting paper. First, as to the method you use for free silica analysis. We have recently in Great Britain, circulated a sample of dust around about six laboratories, and using three methods of free silica analysis, and the answers ranged on the free samples of dust from about one percent to ten percent on the same sample of dust sent around to different laboratories, so I think the free silica figures are very significant in relation to the method used, and also even to the individual using the method.

Secondly, I notice you did petrographic analysis of air-borne dust. Now, that has eluded us so far. We have not been able to study inhalable dust under five microns in diameter by petrographic analysis so far. The petrologist will go down to about ten, fifteen microns; below that, he says, 'Sorry, can't help you'. We hope we're getting somewhere by a new method in which the particles are crushed under the microscope, into a thin sheet and we may be able to identify them petrographically that way; we hope so.

Thirdly, I notice you took samples on filter paper; you apparently dispersed them to get your counts. Of course, the man doesn't do that. He breathes the dust in the air, and his own system and his respiratory tracts takes the particles, and our view at the moment is that he doesn't inhale anything over a particle size with a terminal velocity equivalent to a particle of - a spherical particle of unit density about five microns diameter.

Now, of course, if you collect dust on a filter and then disperse it, you break up all the aggregates and the aggregates are, of course, the big factor in the actual inhalable dust concentration in any coal mine, and I wondered what your views were on that point.

And, lastly, I see you quoted average dust concentrations without mentioning any standard areas. We find that the co-efficient variation of precipitate amounts, I quote from memory, it's of the order of fifty percent. There is a collossal variation from moment to moment, and just an average dust concentration taken regardless, to the moment at which the man is working and without reference to the various - the variability we feel may be highly misleading, and I wondered what your views were on that question.

BY MR. URBAN:

I'm sorry I didn't make myself clear. The dust counts that we made, the dust counts that we made were not on filter paper but we made them with the standard United States Bureau of Mines Light Field count. That is we actually sampled atmospheres and counted particles less than ten microns. The filter paper collection was simply to collect air-borne dust.

Now, it's true that, petrographically, we can not see, that is we can not analyze petrographically, particles lower than five microns. In our case at the laboratory, we employed three men. We used topography, chemistry and index, and X-ray defraction combined to give us some idea of the mineralogical components of the dust. We had done the air-borne samples by petrography, so we could get our other mineralogical components, and their action, like calcium, magnesium, iron. The quarts in that table was done chemically.

Now, the reported average counts for several of these: One was a very practical one. If we put the ranges on that table, there just would be so many figures we coudl not recognize the table, and certainly we did find variations as much as fifty percent from one count to another in the series, but we did report the average because we studied the series. The average looked fair and we reported the average.

BY DOCTOR DRINKER:

We're doing petrographic analysis, Doctor Fletcher, on particles that are well under five microns. The only one you've got is past history. It seems to me it's perfectly analogous to what you demand in radiological analysis. You won't give a positive diagnosis of silicosis unless you know the man's history.

Well, I don't see - this seems to me perfectly analogous, if you know the past history of the dust and know it accurately, you can do things petrographically that you can't possibly do if you come into an unknown, and following that argument out, it's common knowledge now that a number of our laboratories in the United States do do petrographic analysis on dust well below five microns. They wouldn't teach you that in a course in petrography, but it's being done.

I asked him that designedly, that question about the interference of the fibers on the filter, because Briscoe and Matthews and your other pals have said that you can't do it with those. My point is that the numbers of those fibers are so trivial that they're not of any consequence, but I'm sure that the Americans Don't profess that their accuracy in these determinations in any way is remarkable, and we have just as bad troubles on the big ranges of concentration as you do.

The next paper is by Doctor Fletcher on Radiological Classifications.

BY DOCTOR FLETCHER:

Mr. Chairman and Members of the Symposium: I'm sorry that I should appear again so shortly. I hope that there are not too many radiologists in the Symposium this morning, and although I should like there to be in some ways,

TAKEN FROM PAGE 156.

BY DOCTOR FLETCHER:

Mr. Chairman and Members of the Symposium: I'm sorry that I should appear again so shortly. I hope that there are not too many radiologists in the Symposium this morning, and although I should like there to be in some ways, and that is that I find that radiologists known as little about radiographs of pneumoconiosis as general physicians know about the clinical and physiological picture of pneumoconiosis.

Seven years ago, when I started my work in relation to pneumoconiosis in South Wales, I consulted a very eminent radiologist in London and he told me that the radiological side of the pneumoconioses had been completely worked out and there was no further cause to do any work in that subject at all. And when I got down to South Wales, I started studying literature.

Could I have the first slide, please? And this is a brief summary of the sort of thing I found about the systems of classification that had been widely used in relation to coal miners, particularly in various countries. There was the original ILO classification with the three stages which were partly radiological and partly clinical; there was the Miners South African Classification that some authors had attempted to apply to coal miners with all these elaborate classifications here, mostly based on the size of the operation; the American Public Health Service classification used in those excellent reports in the hard and soft coal miners, again with various elaborate subdivisions.

Now, these ones here, and this side of the diagram, I know the real equivalents in these classifications, because we've done actual readings of the same sets of films in the different classifications with the people using them, and we know something about the equivalents.

Over here, the equivalents are a bit different. We have the French one, which is normal with a little sub-group of normal, the 'pathologique normale' which is a rather nice bit of French compromise, the little sub-division of normal, and the fibrotic F here, 'Fibros modulaire', and 'supernaturale', and these were based on the size of shadows.

The Germans, in deference to them, I have simply - I have simplified what they actually do. They actually put in the intermediate classifications of one and two, two to three, between two and three, and they even have a sub-division here, naught to one to one, and naught to one to one in between these two, and then the Silicosis Boards in England have modified this original one here. Genhardt, who wrote those, did the original survey work . In South Wales they had done an original classification of their own, using a term reticulation, which I regard as an abomination, because it is being used by different authors to indicate anything from a normal film up to the most advanced nodulation and then that was the situation, and we eventually worked out a new system of our own which I have indicated roughly here.

Now, the three reasons why we wanted a new system. First of all, none of the existing systems recognized the basic distinction we wanted to make between radiographs that showed only generally discreet nodular shadows and those that had, in addition, local, coalescent or massive shadows.

Our reason for wanting to make this distinction was largely based on Professor Goffe's work, as he made a very clear pathological distinction between what he called simple pneumoconiosis and massive fibrosis, and I'll show that in a moment.

The second thing was that in our follow-up studies of radiographs of men progressing, we found that the progression of the disease in those with generalized discreet shadows, was manifested always by an increase in the number and profusion of shadows and only in proportion by an increase in their size.

Now, all the other classifications indicated progression by increase in size, ignoring increase in profusion.

And, the third reason was that we wanted to make our classification purely radiological. We didn't want to have any confusion with clinical symptoms which had appeared in some of the others.

The next slide, please, shows this basic distinction -- the radiology -- between coal deposits in the lung --

next slide -- and those with, in addition, large massive shadows, and that is the basic distinction that we make in our classification.

Could I, just for one moment, have the first slide back again. I'm sorry to go out of order, but it just would help if we could have it back. Well, now, we devised a system which has been published and which some of you know about and we advocated a similar system at the ILO Conference at -- I say we did -- I advocated a similar system at the ILO Conference in Sydney in 1950, and somehow or other, they suggested a system of classification that bears rather a resemblance to the one that we have been using.

Later on, we thought that the just mere announcement, by an International Conference of a system of classification that was a good one wouldn't do much to increase international understanding. In any case, the ILO have not yet published the report of this Conference. Things go slowly in Geneva. The Conference was over two years ago, but nothing has appeared yet about it, and we wanted to be able to understand epidemiological results that were being obtained by workers in coal fields in the north of France and in the Ruhr in Germany, and we therefore, sent them sets of films from our mines and asked them to send us sets of films from their mines.

We carried out readings in their classification and our classification, and we undertook meetings to discuss this and eventually, both in France and Germany, they have agreed to use this type of classification for their work, but have introduced a small modification that they want to distinguish in the groups of simple pneumoconiosis, that here the type of films with very fine capacities, moderate size and larger size, so as to retain something of the original indication of size, but in a secondary role.

Well, now, the actual classification used, consists of a normal group, three subdivisions of discreet shadows. We originally had four; we have dropped the fourth now. Three are quite sufficient , and then they have four divisions of cases with massive shadows.

Now, the next slide. Here is a radiograph. It's almost impossible to demonstrate these things with lantern slides, but I have got quite a collection of films here if anybody would like to come and see some of the original films afterwards, but in classification 1, we demand presence of minute opacities, usually one millimeter to 1.5 millimeter diameter, in little clusters in at least two rib spaces, each area occupying an area of one square centimeter.

In Classification 2 -- next slide -- we require that these opacities be distributed throughout both lung fields, but they are usually sparce at the periphery and up at the apex.

By Category 3, they haveto become generalized by all lung fields and are now profuse and extend right out to the periphery.

That is the basic classification of the simple pneumoconiosis, but in addition, we do recognize these different types, that is the mixed type which we call mixed, or 'Micron Nodulaire' which also begins with 'M', and the Germans call it , but unfortunately, we're prepared to call it some other word which I forget at the moment which does begin with an 'M', but -- next slide -- there is a common type occupying about ten percent opacity in simple pneumoconiosis, in which these little minute opacities occur in pure culture which we call the pinhead type; the French have had the courtesy to call it 'Pinhead Fleshaire'. The Germans - the Germans have insisted on calling it , which is a little different, but anyway it indicates this pinhead period.

On the next slide, we have, on the other hand, the nodular type of film,in -- fortunately, 'Nodulaire' in French, and 'Nodular' in German, and we indicate that with an 'N', following the number indicating the category of the simple pneumoconiosis, and this, of course, the nodular appearance tends to occur in men with a heavy rock exposure who have actually got classical silicosis, together with coal deposits, the only condition which justifys the term 'Anthraco-silicosis'.

Well, now, coming on from that -- next slide - we come to the beginning of what we call progressive, massive fibrosis, PMF, which the ILO calls pneumoconiosis, with coalescent massive shadows where, in addition to the general condition, you have an area with larger opacities, amounting to rather more in diameter than appear, and although, in many films, the radiograph also shows a definite mass. That is the beginning which we call PMF, to distinguish the categories from the numbers we use in simple pneumoconiosis.

The next slide. These cases have the peculiar property that they tend to progress whether or not the man is exposed to further dust inhalation. Our simple pneumoconiosis, we see progression from Category One to Two or Two to Three only under conditions of dust exposure where presumably further coal dusts are being laid down in the lung, but here is a radiograph of a man who leaves with a shadow, leaving the mine in 1941.

Next slide. By 1943, he has now a sizeable mass of it here and another one beginning there -- (next slide) -- and in 1947, he has a very definite mass here. Note the next one. The next one -- may we have the next one -- this is 1947 with now a large mass in both sides of the lung field, and we indicate these decreasing size of masses with the letters B and C, B for shadows that extend only over three anterior rib spaces, C if they extend over more than the three anterior rib spaces, just to give an indication of their size. That's not a very important distinction, so far as we know.

And, lastly, we use the Category D -- next slide -- for those which, in addition to massive shadows, have gross distortion of the diaphragms, the mediastina here, and with emphysema at the basis, which, as this radiograph shows, can be quite extreme in many of these cases.

So, in summary now, -- next slide -- we have the classification slide which I have used both in France and Germany; that's why it's international - in which we have the normal films with naught and the French again wanted to retain some vestige of their pathologique which we call X, which is a film which you can not put your hand on our heart and say it's normal because there is something that suggests pneumoconiosis to you, because it might well be something else, some sort of non-specific fibrosis, emphysema or what have you, bronchitis; and so, if you like, you can call it X. We regard it as pretty bad form in the pneumoconioses to use X; we only do it when we're absolutely forced to, because we just can't make up our minds.

Then, we come on to simple pneumoconiosis, which is three categories depending chiefly on the diffusion of the opacities, on their diffusion up to the rib area, with the pinhead, micro-nodular, mostly put in for the consideration of the French, and for description, but we don't regard these categories as having any great significance. Natural history, and the prognosis and everything else, of the pinhead, micro-nodular and nodular is similar.

Then, we come to progressive fibrosis, A, B, C and D, depending on the size of the shadows, and eventually D, which you can't see on the table there which has distortion.

Well, now, that is the classification, and now I want to say one or two words to try and persuade you that it may be quite a good one. First of all, it is a purely radiological classification. It is used by looking at the radiograph and without any further information at all, which is what a radiological classification should be.

Secondly, the categories are based on studies of the progression of the disease, so that the earlier categories represent earlier stages in the natural development of the disease, than do the later categories.

Thirdly, in justification of our categories of simple pneumoconiosis, we have found evidence which I shall show in my next talk on the epidemiological side, evidence that there is a fairly direct quantitative relationship between the amount of dust exposure required to produce these three categories. The ratio of dust exposure required to produce Category Two is about twice that required to produce

Category One, and to produce Category Three is half as much again. The actual ratios are about six to ten to fifteen. So that these categories do represent a fairly direct quantitative relationship to the amount of dust inhaled. We want to confirm that by chemical analyses of the amount of dust in the lungs in these categories but we haven't yet had the results of that completed.

With regard to the relationship to the underlying pathology, there is difficulty. We, as radiologists, on the radiological side, think the difficulty lies on the pathological side and the difficulty I referred to yesterday, and that is the difficulty of getting the pathologist to be quantitative and to say just how much abnormality there is in different lungs, but we have got Professor Goffe and his colleague, Doctor James, to classify a large number of lung sections done by his technique, which we had radiographed too under three systems, one according to the profusion of the opacities, and also according to the severity of the focal emphysema.

There is no relationship between our categories and the severity of focal emphysema, none. The profusion of the opacities is not effected by the development of focal emphysema, but there is a relationship, a definitely rough relationship between our categories and the dust foci, which is some indication of the amount of coal in the lung.

Lastly, the relationship to disability is of great interest, but as Doctor Hugh-Jones is devoting his talk to that subject, I will leave it to him, and I will only say here that there is little relationship to disability here, but a very great justification of this distinction between simple and massive fibrosis, on disability grounds.

Now, one point, I don't expect any of you could see any abnormality in that film I showed you of Category 1. Well, now, quite experienced radiologists in England and in this country, are having the privilege of discussing some films of ours with Doctor Eugene Pendergrass in Philadelphia, and he expressed a firm idea that films of Category 1 and 2 were absolutely normal and he could parallel them with films of any man walking the streets of Philadelphia.

I tried to get him to look at these specific ocpacities which I thought justified the diagnosis. He said he was looking at them but even so we couldn't agree and so either we were wrong or Philadelphia was much smokier and dustier than it looked when I was there, or he was wrong.

Well, I was very worried about this and I was worried, too, about radiologists who said that they couldn't see any abnormality in our Category 1 and, as this Category is one which we have used extensively in our epidemiological work, it was of vital importance to see whether it was true or not, so what we did was to collect 125 films of non-miners, men who had had no significant dust exposure. Some of them were members of our own unit, but the majority were men working in a power station on non-dusty occupations where we had a survey, so we got good films there. We mixed them up and ran them out with films of seventy-five miners of neighboring mines which had previously been read by readers A and B on survey, fifty of them being normal and twenty-five of them being category 1.

Well, now, we thought we'd be clever and not only read category naught, one, two, but we thought we'd put in an extra category naught, with a cross in it, to indicate that although we thought it wasn't quite category one, to indicate that the film showed as if it was a miner's film, because we not only believed we could read category one, but something earlier. So, that's what we asked the readers to do.

The films were read, interestingly, by four readers, A, B, C and D, and then read together by A and B, and by C and D, and finally those films, on which we differed, were read in consultation by all four together, and here are the results for the non-miners and the miners.

In the non-miners, A and B -- miners, first of all -- were rather cautious under these experimental conditions. They didn't want to show they had been reading one wrongly and you will see of the twenty-five films, they read as one - they read ten as one; now, I pushed them down to normal miner and non-miner. In so doing, they didn't read any pneumoconiosis in the non-miners and only read one case as showing, looking like a miner, but not being category 1. C and D was myself and Doctor Gilson. We were more courageous and we read up a little bit; we read thirty-eight out of these films as showing Category 1, one as 2, and about the same number, about thirty-six coming in the normal group, but in so doing, we had read four of the non-miners as having Category 1, four of the 125, and nine as being suspicious.

When the four of us met together to try and straighten this out, we pretty well hit off the original level of reading the miners' films and we never once agreed that there was evidence of pneumoconiosis. I asked that A and B try and re-read these films using their normal survey technique. Here they were put off by the danger of reading things that they were frightened about and they did try and read them exactly as they read the survey films, and they very nearly hit off the same level as on the survey and they never once read Category 1 in a non-miner. They weren't attempting, in that case, to read the suspicious.

Well, I conclude from that, that at any rate, we can distinguish our Category 1 and that it does occur only very rarely in non-miners, and there only when we're over-reading. When we're reading as we normally do, a Category 1 is a thing which is sufficiently rare in a normal not to have occurred in this group of 125 non-miners. Just the same, the sort of danger of less specific reading, I did send these films to two Germans who agreed strongly on this abnormality and you will see they read on the miners, and no less than twenty and twenty-two in the non-miners, as beginning silicosis.

So I think this type of experiment, mixing up completely normal films with films of men exposed to dust, is a completely salutory experiment. I recommend it to anybody who is engaged in this sort of work.

One small point of interest, what with these cases that C and D, Doctor Gilson and I, read as showing some abnormality. I was interested to know if it had any relation to Age. So I have just plotted it out in relation to Age. You see that there is a very clear relationship between age and this abnormality, and I believe that it is possible that these older men aged forty-five to fifty had, in fact, been exposed to a certain amount of dust and smoke in and around Cardiff, and that what we were reading as early pneumoconiosis, may have been the earliest signs of dust accumulating in their lungs with age.

What is most important is that even our overreading of Category 1 never occurred in a man under thirty, and most of our active epidemiological work is concerned with men in that age group, so that I am quite satisfied that our Category 1 is an abnormality attributable to the inhalation of dust in the - at least in the younger age group, and nearly always so even in the older age groups.

Next, I just want to talk very quickly about the question of observation which has been pioneered in this country in relation to tuberculosis, but I think we have pioneered it in our country in relation to pneumoconiosis. The first experiment we did was to get 102 films of miners ranging from normal to nodulation in the original classification. We asked ten experts to classify them into normal, early, definite, certifiable pneumoconiosis, and nodulation.

We found that, of these ten experienced observers, in thirty films, opinions ranged from one observer to the other, from one to four or two to five, that is one quoted normal, another certifiable pneumocóniosis, another nodulation. For shattering results, I took that up to a very eminent radiologist in London. He said none of them were radiologists. He said he and another radiologist would agree a hundred percent on these films.

The silly ass tried it, and the difference between them was greater than between all ten, so -- this is very important -- the number related as showing certifiable pneumoconiosis by these various observers, varied. One showed that four of 102 films showed enough pneumoconiosis for certification; another said that 29 were -- I mean normal. The number certifiable ranged from 9 to 53. These were experienced observers. The average consistency-each observer read the film twice -- was sixty percent; sixty percent of the films the observer did give the same opinion on the two occasions; the record was eighty-four.

The other thing was under-exposed films. Some of the films were classified higher than the hard films. Well, now, we have done a great deal of work on this subject and we have attempted to improve this situation, first of all, by accurate definitions of the categories in words; secondly, by setting up standard films exemplifying the categories, so instead of just putting a film up and saying, well, that looks like Category 2, you put up another film alongside which has been agreed to be Category 2 and see if it looks the same and, thirdly, we have adopted the method of reading shown to be so valuable by Duorshonn and his colleagues, of duplicate reading. Every film on our survey is read on two different occasions without any knowledge on the two occasions what the previous reading was and any film on which two opinions differ is given a third casting vote to get the final category.

The next slide just snows -- no, we'll skip this one, I think this is just showing the errors in relation to the diagnosis of pure math in this international set-up. We'llskip that one. Now, this is our latest result of our readers A and B who do all our survey reading showing that first reading of five hundred films taken at random from a large survey of five thousand and their second reading here, and any film which was read normal on the two occasions never goes to a third reading, but if no two agree on two readings we have a third deciding reading, and you can note the lack of divergence of opinion, which is relativelysmall.

Lastly, on the question of understanding, I have told you that we have been taking films around in France and Germany to get them read and here is at leastthe result of 150 films which we sent out, which we classified as thirty in Category 1; thirty-two; thirty - three, and we sent them to them and put them into the international classification, and into the German classification as presently used, and you will see that while there is difference in the minds of the observers here, there is, on the whole, agreement. If they were to have classified that set of films and given us a prevalence of pneumoconiosis based on that classification, it would have been very similar to the prevalence that we would have found if we had read those films. We could understand their statistics, but if they did it in their own classification, there was some agreement between the categories here. You will see that our Category B films are pretty evenly distributed, and what we call Category A were distributed through the whole range. It would have been very difficult for us to have any accurate translation of that sort of classification into ours, even if we had known what the results of this experiment were going to be.

At the present, the existence of different sets or systems of classification in the different countires of the world, and the different centers, militates very seriously against scientific understanding of epidemiological work. This system of classification has now been adopted pretty well uniformly throughout Great Britain. As I said, it is being adopted in France and in Germany. A conference is being held in Paris to further its adoption in France, next - the end of this week, on this Saturday, and I think the time has come for some international body, the ILO, to start being active in this field and carry on this sort of work we have been doing on our own, by getting sets of films sent around to the various centers where work of this kind is being done so that we can see what the relationship between different forms of classification is, and if possible, obtain the consent of the different centers in this part of the globe, to use a classification similar to that which we we are using, not identical, because I have no doubt that you will have a great contribution to make in suggesting valuable modifications, but at least that we should adhere to using a uniform system so that we can know what you're talking about and you can understand what we're talking about in our radiological work. (Applause).

BY DOCTOR DRINKER:

Any discussion of this paper? Doctor Richards?

BY DOCTOR RICHARDS:

I don't think we're too far apart. I think I could understand Doctor Fletcher as he went along with this, very well, and I don't think I have anything particularly to add.

BY DOCTOR ORENSTEIN:

Mr. Chairman, I would just like to pay a tribute to Doctor Fletcher's work from the point of view of the tactical worker in his field. To many of you, this may seem to be a supererogation of trying to establish a whole lot of categories, but it is a matter of the greatest importance that there shouldbe an understanding in this matter, not only from the point of view of epidemiological survey but from the point of view of such countries where compensation is based on stages of the disease and is not true to form simply because a man has pneumoconiosis. He receives a certain amount if he has pneumoxoniosis in a certain stage, and a certain amount for a larger stage and so on, and because of that point of view and because of the great damage that was done to the compensation question, the certification question by the 1930 adoption of that one, two and three which you saw on the right-hand side of the scale, when an attempt was made to correlate the capacity, that is to say compensation, to stages of radiographic appearance, now, a lot of good has been done by Doctor Fletcher in his endeavors in this way in particular, where he demonstrated this at great length and I'll never forget the night we spent in a small office somewhere up on the top floor of a Sydney building, threshing out this question.

I also would like to pay a tribute to the objective work which he carried out in trying to discover the errors of his own interpretation and of others' interpretations. A lot has been said by radiologists, if we have long enough experience and enough correlation with post mortem findings, we can classify with great ease. That's quite untrue. They can not, and a lot more work will have to be done to make that clear.

I merely want to say this, Mr. Chairman so that somebody who has to work in this field would speak for the great benefit this work has conferred on the knowledge of pneumoconiosis.

BY DOCTOR DRINKER:

Thank you, Doctor.

BY DOCTOR VORWALD:

Mr. Chairman, I should like to join Doctor Orenstein in his tribute to Doctor Fletcher and we here are indeed fortunate to have him with us and to hear what he has to say. I don't think that we're too far apart. Of course, there is divergence of opinion with respect to Category 1 and Category 2. That is where we find the greatest difference.

Doctor Fletcher, I know, is keenly aware of the technical qualities of the film. He hasn't mentioned it; perhaps he will, the necessity for having good films of good technical quality in order to classify these films. Then, I should also like to ask Doctor Fletcher one question, does his classification pertain only to the coal miners' lung, to the coal worker, or does his classification pertain to all of pneumoconiosis?

BY DOCTOR DRINKER:

Will you answer that, Doctor Fletcher?

BY DOCTOR FLETCHER:

Thank you very much indeed, Doctor Vorwald, for raising these two points. I am afraid I was getting behind time and I didn't have time but, of course, the technique of the radiograph is of the greatest importance. We have shown that the average of category given by ten observers to one film, or films of the same man taken on the same day, under-exposed and over-exposed, the average opinion may shift by one and a half categories easily, and it is absolutely essential to have really good technique and we have devoted a great deal of attention to that point in our own radiographic work.

We have now, in our mobile unit, not only an automatic exposure control mechanism which Seaman's in Germany have devised for their automatic, which is quite remarkable in the way in which it enables uniform exposure to be obtained of men of various chest thicknesses and with variations in main supply, but we now have also an automatic processing unit which puts the films through at exactly the right time and temperature and in that way, we have been able to reduce the number or percentage of films that we have to reject on our survey as technically satisfactory from about forty percent right down to two percent.

About the width of application of this classification it has been worked out on coal miners and this work has been done on coal miners, which is the big subject problem we have in Great Britain, but I think that in principal, it is applicable to all the world's pneumoconioses which manifest themselves in discreet capacities going throughout the channels, that is to say coal pneumoconiosis, what Doctor Sander was calling sidero-silicosis, silicosis and so on.

I think the same principals can be applied.

The members of the Silicosis Boards in Great Britain who see men from all industries, tell me that they find this method of classification valid for at least ninety-five percent of all the cases they see. There are the odd cases which you'll say, I'm sorry we just can't fit this in. You've got to put it down as unclassified.

BY DOCTOR RICHARDS:

Mr. Chairman, I think while we're discussing this problem of terminology, I'd like you to call on Doctor Johnston, from a clinical standpoint to say a word to you.

BY DOCTOR JOHNSTON:

I have nothing to add. I just think that Doctor Fletcher, in all the arguments yesterday, Doctor Fletcher has probably established Doctor Sander's point that everybody has pneumoconiosis, the classifications one or two; even the farmers I'm certain we'll see, that in farmers who live in impure air all their life, so everybody must have pneumoconiosis.

BY DOCTOR DRINKER:

There are a fair number of persons in this room who are not good Roentgenologists then, and there is one definitely of that number I'd like to ask a layman's kind of a question. I have been steeped in the doctrine started, Pankos and Sampson and right on down to the present experts, that these radiological pictures differ enormously with the geology to which these men are exposed and presumably, Doctor Fletcher's interests are largely in the Welch coal fields. How does this classification apply to say our hard rock miners in the Missouri area, Pendergrass certainly must have brought that up in talking with you.

BY DOCTOR FLETCHER:

I think that the classification would apply completely, but that our category 1 and 2 are a little earlier than Doctor Pendergrass would recognize. One of the difficulties is with these very early abnormalities, you have got to study the film extremely closely. We say ten inches is the right distance from the observer to the film. A greater distance than that you just can't see these early changes and I put up that film, that slide of the results of distinguishing normals from these category 1, to show that if you do that, you can distinguish and all I can say to Doctor Johnston is that if you will get these films of these farmers and send us a group, we will mix them up with some miners and get some readers who don't know what we've done to read them, and I'll bet he'll find that the miners have got more dust in their lungs and have got a higher proportion of Category 1 abnormality than have his farmers.

If, on the other hand, his farmers have got a great deal of Category 1 abnormality, all right, I agree with him, they must have some pneumoconiosis, and Doctor Lanza, I think, would agree too that it might be called occupational.

BY DOCTOR VORWALD:

Well, Doctor Fletcher, there is no way in which Doctor Johnston can win, is there?

BY DOCTOR FLETCHER:

Yes, yes, he can, because we can get a third group of real -- in our country, there is very little dust in farming; in fact, none at all, and we could get a group such as these power station workers who also are not perhaps a dusty occupation as your farmers. I don't know. I have heard about the Dust Bowl, but I don't know how dusty this is, this occupation of farming over here, but we could certainly get a group of true normals in agricultural workers in our country or in office workers in New York; looked to me as if the atmosphere was pretty clear there, and we could say these are normals. If we diagnose abnormality in them, then clearly this Category 1 we're talking about is absolute nonsense in terms of coal workers pneumoconiosis.

BY DOCTOR JOHNSTON:

Phil, I don't want to labor this point; I happen to have a daughter who has lived in California all her life. She has never been on a farm, never done any work of any kind, there is no bituminous coal in California. I can vouch for that. She does have, however, some markedness that

DR. SANDER: I don't say that everybody has pneumoconiosis which is visualized by the X-ray. We all have some pneumoconiosis, by strict definition, but not something visualized by X-ray. One can have a lot of anthracotic pigmentation and still have a perfectly normal chest film. We are deeply indebted to Dr. Fletcher for this precise classification and I see no reason why we can't adopt it for almost every industry in this coutry. Even the diatomaceous earth cases can go into categories A, B, and C; they miss categories 1, 2, and 3 because the shadows are never nodular. I see no reason why we can't use the classification for almost any dusty industry in this country, with the possible exception of category 1.

DR. FLETCHER: I wish to make it clear that normal individuals in Great Britain whom we have examined do not show the abnormality we classify as category 1, except perhaps 1 or 2 per cent of persons older than 50 years. I am quite convinced that this abnormality which we are classifying is a specific change produced by dust. I won't say that the odd case of sarcoidosis or non-specific fibrosis can't mimic it but that is unimportant from the standpoint of industrial medicine. We needn't worry if we make a mistake about the odd disease which affects one in a million, but this condition is specifically associated with the development of dust retention in the lungs, as is shown by the coal miners.

I'm interested in Dr. Sander's statement that some persons may skip categories 1, 2, and 3. I believe that happens to pottery workers in our country and I have seen films supporting this belief.

DR. WORMALD: If you classify in category 1 an individual who has had some respiratory difficulty, are you ready to compensate him for the inhalation of dust?

might be classified under two of your films.

Now, I don't know how, what we do about such people. We can get into a hobby classification or a work classification and still they show this little stuff.

Now, I'm still getting back that we must adopt a point if we're going to go ahead with your classification, to get back to Doctor Emmett Sander here that everybody has pneumoconiosis.

BY DOCTOR DRINKER:

Doctor Sander?

BY DOCTOR SANDER:

I'd like to clarify that. I don't say that everyone has pneumoconiosis which is visualized by X-ray. We all have some pneumoconiosis by strict definition, but not visualized by X-ray. You can have, I'm satisfied, you can have a lot of anthracotic pigmentation and still have a perfectly normal chest film. I think we owe Doctor Fletcher a great debt of gratitude for this precise classification. I see no reason why we can't adopt it for almost every industry in this country. Even the diathermacious earth can go into Category A, B and C; they miss Categories 1, 2 and 3, because they never are nodular, and I don't see any reason why we can't use it for almost any and every dustty industry in this country, — with the possible exception of category II.

BY DOCTOR DRINKER:

Doctor Fletcher?

BY DOCTOR FLETCHER:

I do want to be absolutely clear about one point and that is that normal individuals in Great Britain, that we have examined, do not show the abnormality which we classify as Category 1, except perhaps in one or two percent of those over the age of fifty.

Now, I want to make that absolutely clear. I am quite convinced that this abnormality that we're classifying here as a specific change produced by dust. I won't say that the odd case of sarcoidosis or non-specific fibrosis, dermatosis, God knows what, can't mimic it, but that's absolutely important from the point of view of industrial medicine. The odd disease which effects one in a million of the population, we needn't worry about if we make mistakes there, but this condition is specifically for - as I'm convinced by our experiments, for the development of dust retention in the lungs as shown by coal miners.

I'm very interested in Doctor Sander saying that some people may skip Category 1, 2 or 3. I think that happens to the pottery workers in our country. I have seen films there, which show a shadow in which the background showed no pneumoconiosis at all, and I think that can happen, but I think the principals can be applied, and I do want to make it quite clear, and I think perhaps some of the slides I shall show you on the epidemiology of the disease, will persuade you that this condition does develop in relation to dust exposure.

BY DOCTOR VORWALD:

I should like merely to ask one more question and that is, Doctor Fletcher, you have classified now an individual as belonging to Category 1, had he had some respiratory disability. Are you ready to compensate him for inhalation of dust?

BY DOCTOR FLETCHER:

I have the good fortune to be a research worker not concerned with compensation, and it's entirely a question of the legislative definition. The practice of the medical boards in our country at the moment is not to recognize anything less than our Category 2. My own personal view is that if a man has worked in a coal mine and shows respiratory disability and has any evidence of dust retention, Category 1, it would be fairer to bias the scales in his favor and give him the benefit of the doubt and say that that disability could be due to pneumoconiosis, but I think that what Doctor Hugh-Jones will say this afternoon may throw a little further light on that problem.

BY DOCTOR JOHNSTON:

Doctor Fletcher, while you're still there, do you have any figures on your present employment classification?

BY DOCTOR FLETCHER:

I hang my head in shame and say that although we have been agitating in our units for information, for four or five years now, for pre-employment and regular examinations of coal miners, they have not yet taken place in Great Britain, but in those films I showed you of non-miners, some of those might have been applicants for coal miners and all I can say is we did not diagnose Category 1 in any man even when we were reading rather generally, we didn't diagnose it in any man under the age of thirty-five.

BY DOCTOR DRINKER:

The next paper is by Doctor Vorwald on the Pathology of Coal Miners Pneumoconiosis. Doctor Vorwald.

BY DOCTOR VORWALD:

Mr. Chairman, Ladies and Gentlemen, at the onset, I wish to say that we, in planning for this day's session, made every effort to bring Doctor Goffe (Gough) from England. As you know, Doctor Goffe is Professor of Pathology and Bacteriology at the Welch National School of Medicine in Wales and he has had perhaps more experience with the pathology of pulmonary changes in coal workers than any man, and it is unfortunate that he could not be with us.

Now, in a general way, our concepts of the changes in the lungs of coal workers which have come to us here in the Saranac Laboratory for study, conform in general with

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Now, in a general way, our concepts of the changes in the lungs of coal workers which have come to us here in the Saranac Laboratory for study, conform in general with the concepts expressed by Doctor Goffe. In some instances, however, there is divergence of views which I hasten to say is occasioned perhaps by the fact that our study of lungs of coal workers has been limited to relatively few cases. In addition, it is possible that the conditions of exposure experienced by coal workers in this country are different from conditions in Wales.

Furthermore, the problems pertaining to the inhalation of coal dust are indeed complex. Now, that complexity may be more fully appreciated should an attempt be made to answer the question as to what is coal dust, and then may I have the first slide.

Now, this slide, taken from a publication by Parkers in Coal Science, by Doctor Banion, Director of Research Laboratories, the British Coal Research Association, here Doctor Banion has tabulated some of the various rare elements in coal ashes.

Now, the data, as we see them here, are expressed in grams per ton or parts per million. To give an approximate idea of the concentration factor, comparison may be made with the average amount of the same element found in the oridnary rocks of the earth's surface as we'll use this factor. As for Arsenic, As, arsenic is one thousand six hundred times - one thousand six hundred times more concentrated in the ash of certain coals than in the average rocks of the earth's surface, so we can also go through the listing of the other elements, beryllium, germanium, lead, nickel, platinum, zinc, zerconium, and you can multiply, you can see the factor, the concentratio n of those substances in coal ash.

Now, it is possible that some of these substances exist, per se, in coal in the dust breathed by these individuals and it is also known that some of these substances produce damage to the lungs.

In attempting to define what is coal dust -- next slide -- the problem becomes even more complicated should we examine the so-called coal tree, and here one visualizes the production obtained from coal by carbonization. Now, many of these products may exert their effect when coal dust becomes deposited in the lung. I am not too clear about this, but I merely present it to give exposé to the possibilities at least that some of the complicated features as we have seen them, pertaining to the inhalation, deposition of coal dust in the lung, may be related in some way to all these products which are derived from coal.

Now, what are the changes that occur in the lungs of some coal workers exposed to coal dust? This is the chest Roentgenogram of a 38-year old, I mean of a fifty-year old white male, who has worked for 38 years as a motorman in a bituminous mine. Now, his medical history was i irrelevant except for two years prior to death when this individual suffered from dyspnea, from exertion, I mean, expecially on exertion, and from productive cough. Furthermore, he had clinically manifest arterial sclerosis, which - and ultimately developed multiple infarcts in the lung and brain and he died in coma.

The X-rays, as you see here, the lung markings are slightly exaggerated. There is a primary complex, as you see here, in the right lung. There is no definite Roentgenographic evidence of change, comparable at least with a diagnosis of silicosis. The gross section of this lung exhibits diffuse pigmentation, slight focal emphysema, as you see, rather coarse in type, some focal emphysema in the lower lobe of this lung which is of the right side. The lymph nodes are small, but they are slightly pigmented, and this is the section of this lung and it happens to be the left upper lobe, and we find that there is focal deposition of dust scattered everywhere throughout the lung. Furthermore, there is also deposition along the vascular fronts which gives rise to what we call a linear deposition. Also one sees the dust deposited in the tissue immediately below the pleura. Furthermore, one can see at least the disporportion between the size of the alveolar spaces which we identify as the anatomical evidence of emphysema, also note the tracho-bronchial lymph nodes which are only slightly enlarged and pigmented.

This is from the right upper lung, right upper lobe. Now, in addition to having the focal deposition of dust, the linear deposition of dust along the vascular tracts, we also have a large area of deposition of dust and in addition, the emphysema in this instance is quite obvious, occupying one small portion of this lobe, but it is also apparent in the mid-portions of the lung and quite well scattered throughout the whole pulmonary tissue.

The next slide, this is from the right lower lobe. I am merely showing you the different sections from different portions of the lobes of the lung so that you might have opportunity to compare what we see in the actual tissue with the changes visualized in the Roentgenogram.

Again, this does not differ much from the sections of other lobes, focal deposition, linear deposition following the vascular tracts, evidence of anatomical forms of at least coarse emphysema.

The next slide. Now, if we examine this lung microscopically, we find that many of the vascular trunks are surrounded by a small amount of connective tissue which is readily seen in sections named specifically for connective tissue. Here we find no evident deposition of dust around this vascular trunk. As you perhaps know, following the deposition of dust in the lung, that dust is taken up by phagocytes; it is mobilized. Those phagocytes, laden with dust, find their way ultimately, that some of those phagocytes, ultimately into the lymphatics about the vascular trunks, but this is a normal vascular trunk seen in the section of this lung.

The next slide. And then, we examine other vascular trunks and we find that in some instances, there is deposition of this black pigment, and there is a slight, very slight increase in the collagen that has been formed. You can see by this connecting tissue.

The next slide. And if we examine the lymph node of this man who, as you will recall, was fifty years of age, and worked for thirty-eight years as a motorman in a bituminous mine, we find dust which has been drained from the lung to the tracheobronchial lymph node, yet, there is no evident significant response to that dust which has become deposited in the node.

We thus - this case demonstrates that long occupation in a coal mine as a motorman does not demand the presence of large amounts of dust in the lung nor does that occupation demand the presence of significant pulmonary changes due to the inhalation of dust.

The next case, some cases exhibit changes as seen in this case, as seen cases with classical silicosis, complicated also by tuberculosis. This is the case, the chest

Reontgenogram of a fifty-year old colored man; occupational history, worked underground for twenty-eight years as a coal loader for eleven years, as a rock helper and rock helpman for thirteen years, as a truck helper for four years.

My X-ray on the right side, we see scattered throughout the entire lung field, areas of increased density, and there is also a fan-shaped area of consolidation, as you see in the upper third of the lung, within the region; that region there is also an area of relative rarifaction. On the left side, there are scattered throughout, small areas of increased density.

The next slide. The section of this lung, this happens to be the right upper lobe, exhibits scattered nodular lesions throughout all of the lung tissue. They are quite well defined, but yet not as defined as is the case with the classical nodular silicosis, in that the borders of these lesions are irregular, and if we examine many of them, we find that they are scattered in shape, with finger-like projections into the adjoining alveolar walls.

The next slide. And, if we examine many of these nodular lesions, we find that they demonstrate the deposition of collagen, arranged in typical fashion, which is classical for the reaction to free crystalline silica. In addition, we also find that the periphery of these nodules, is usually surrounded by a thick or thin collar, depending upon the circumstances of exposure, of deep pigment, of black pigment. Furthermore, this pigment, and its associated slight reaction, deposition of collagen, extends into the adjacent alveolar walls. Examination of other lesions, we could demonstrate fibrosis, actual fibrosis, of hazy fibrosis in this case, of this hyalinized islanized fibrous tissue, which is similar to the change of scene in silicotics, complicated by tuberculosis.

Thus, under certain conditions in coal mining operations, or certain coal mining operations are associated with the dissemination of dust, which, on inhalation into the lung, produce silicotic reaction, and some such reactions are also complicated by tuberculosis, as is present in this case. This complication is more evident in the next case.

Next slide. The gross section of this, the lung of a driller in a bituminous mine. He was a driller for nine years, been exposed to high levels of free crystalline silica. Now, we see, first the nodular lesions, scattered throughout the lung. We see that they are studded in shape. We see many of them on close inspection, that they project into the adjoining alveolar walls. We see the evident emphysema which is at best and coarse in type. We see larg lesions and we also see coalescence of lesions which has given rise to a large massive shadow in the Roentgenogram, and close inspection shows obvious tuberculous complication.

The next slide also manifests the tuberculous complication such as here, coalescence of lesions with a massive area of fibrosis, complicated by tuberculosis with the production of an excavation in which numerous tubercle bacilla were found.

Now, the lungs of some coal miners show also the complicated features which accompany other pneumocionioses. For example, the next case, that of a white male subject fifty-three years of age, who had been employed for many years in a coal mine, a bituminous coal mine. Now, I can not give you all the details of his occupational history, because this is a relatively recent case and we are still seeking them.

The medical history, the patient developed a spontaneous pneumothorax, and we can see it on this side. Here is the edge of the lung, along the lung after an attack of coughing following a cold. The X-ray at this time shows a spontaneous pneumothorax on the right, estimated degree of collapse about sixty percent. The impression is given that within the collapsed portion of this lung, there is definite nodulation. On the left, several areas of rarification are seen in the upper half of this line, with the loss of pulmonary markers which are usually present in a healthy lung. The impression is also gained here that nodulation is present.

The next slide, eight days later, now, a hydropneumothorax on the right has developed, a rubber catheter is inserted; there is a fluid line, and the area of rarifaction still exists on the left. The impression is gained that nodulation is present.

The next slide is a section from the right lower lobe in the area of consolidation seen Roentgenographically. It is a common fluid hypostatic pneumonic process. We will not spend much time on this particular feature of this case, but note also the scattered areas of focal pigmentation, and also the rather coarse emphysema.

The next slide, the right upper lobe section and also sections from the left lung, we see the isolated lesions which are not too numerous and here and here, stellar in shape, projecting along the alveolar walls. We see the emphysema, but what is more troublesome, in some of these cases of coal miners, and indeed it is not specific for the coal miner, we also see it in those individuals who have inhaled free crystalline silica, mixed with other components such as the iron miner, the magnatite miner. We see large areas of dust pigmentation presumably due to coalescence of the pigmentation, but we find areas of liquifaction necrosis within those lesions.

Now, this liquifaction necrosis is also very troublesome, although we believe that in the majority of instances where coalescence of lesions has occurred where large massive areas of shadows develop in the test Roentgen, that that coalescence is due to a complicated tuberculous infection. Yet, there are those cases, with necrosis, with liquifaction and indeed with excavation, where we have been unable, with all means at our command, to detect the presence of the tubercle bacillus or to see evidence of reaction which characterizes tuberculosis.

Lest we're led to believe that coalescence and necrosis and liquifaction may be due to agents or to factors other than infection, and we have advanced the view that perhaps high local concentrations of dust may be responsible for some of the necroses, or that the necrosis seen in these lungs were - with mixed exposure, where there is present carbonization material in large amounts, where there is present iron oxide in large amounts, that perhaps this necrosis is a specific action for compounds in the dust, other than the free crystalline silica. Is it possible that the carbonation component, that the iron component, may have a power of absorption or adsorption and so keep localized the toxic substances, perhaps liberated from the free crystalline silica particles and some dust irritating particles which certainly may produce this necrosis. Certainly infection is considered - and I mention that - infection other than may produce tuberculosis, is a possibility, and I submit that the demonstration of tuberculosis in some of these cases, can not be accomplished.

The next slide, if we take a micro-photograph of these areas of liquifaction, some of the things which I have said become more clear, as for example, here is a micro-photograph of that area of liquifaction. A connective tissue state, we see the typical collagen, sustained with connective tissue, as we see, which characterizes the response to free crystalline silica. We also see the heavy black pigment which has been inhaled and deposited in this coal miner's lungs and we see the area of necrosis and liquifaction. There is no evidence, histological evidence of tuberculosis infection. Then what is this due to? And I again submit the reasons for possible necrosis and liquifaction other than infection.

The next slide, and this is the character of the stellate lesion as we like to identify it, the irregular lesion which is not discredt, which is not the run or order of classical nodular silicosis or the reaction of the lung to pure free crystalline silica. We have here the whirling of fibrous tissue which characterizes the reaction to free crystalline silica and we have had the color of pigmented -- that collar of pigmented tissue, about the lesion which extends, as you see here, along the alveolar wall, and also the distortion of the adjacent alveolar spaces. This is an important feature which I will call your attention to shortly.

The next slide, in addition to the area of liquifaction necrosis without detectable infection, is the focal reaction present about small deposits of coal dust in the lung. Now, this focal reaction has been described by Doctor Goffe. It has been described by others. It poses a problem which is difficult to solve and perhaps has not been solved. The focal deposition of dust with its reaction, the focal emphysema, is troublesome because it is a large factor in the respiratory disability perhaps of these coal miners, and here we have a case, a gross section of the lung of a coal miner, in a bituminous mine. He was a machine man and a loader for seven years. Here we see the focal deposition of dust scattered throughout the lung, different portions of the lung. The lesions are stellate in shape and you can readily see the distortion, the increase in size of the alveolar spaces, adjacent to these focal deposits. That then is the focal emphysema which has been so clearly defined by Doctor Goffe, and which has been referred to by Doctor Fletcher and his co-workers.

The next slide, and we stain these lesions specifically for a certain - with certain special stains and we note the heavy deposit of coal dust and we also note a proliferation of collagen, which permeates the lesion, and we can see the irregularity, we can see the emphysema and also the projection into the adjoining alveolar space.

The next slide, another case -- no, this is the same slide, and here it is, showing you the infiltration of the adjacent alveolar walls with coal dust which has become deposited in the wall, the thickening of the wall, the relative or the degree of - the sparcity of the collagen, formation of collagen such as we would see if pure free crystalline silica were deposited in those walls and we see the large alveolar space, this is the anatomical form of emphysema as we see it.

The next slide. This emphysema is more pronounced and these, of course, are advanced cases and this - you can readily visualize, belongs to the - one of the stages so well described by Doctor Fletcher, of a bituminous miner for thirty-eight years. He was a mule driver for a number of years; he was a machine man and motorman; he was a coal loader for approximately twenty-four years, and he was a trackman.

His symptoms began approximately six years prior to death and they were increasing respiratory and cardiac difficulty. The X-ray, in addition to these areas of massive shadows, shows scattered focal shadows throughout the lung. There is also marked prominence of the left ventricular cells and the transfer diameter of the cardiac silhouette is somewhat enlarged. The Roentgenographic interpreation was that in the presence of an adequate history of exposure to free crystalline silica, the changes visualized in the lungs are compatible with a Roentgenographic diagnosis of silicosis with conglomeration. Post morten showed advanced arterial sclerosis, coronary involvement, cardiac hypertrophy and dilation.

The next slide. The gross lung in this case is heavily pigmented as you see here with black pigment and, of course, one can also readily recognize the coarse emphysema present. The next slide, and the section of the right upper lobe, the emphysema certainly is now most pronounced and most emphatic, and now again, we see the focal irregular deposits of pigment extending into the adjacent alveolar spaces, the small portions of the lobules of the lung are emphasized about these, and if we pick many of them up, there are large or more coarse emphysematous areas. There is, near the hilar region, this area of massive pigmentation. The tracheobronchial lymph nodes are enlarged.

The next slide. That was from the right upper lobe. This is the left lower lobe. Again, the emphysema now is more pronounced. Of course, gross emphysema, and the massive area of pigmentation. Of course, this is now the emphysema beyond the stage which is so troublesome. course, this is the progression, no doubt of this small focal area, areas of emphysema which occur about the deposits of dust.

And now, the question arises, did the emphysema exist before the pulmonary deposition of dust? Would the emphysema have developed without deposition of dust or is the emphysema related to or caused by the deposition of dust in the lung? I am unable to answer the first two questions, namely, did emphysema exist before the pulmonary deposition of dust in this case, or would the emphysema have developed in these cases without deposition of dust? That demands a number of studies which I'm sure you will hear more about in subsequent days, but the question is, is the emphysema related or caused by the deposition of dust?

This picture of focal emphysema has been adequately described by Goffe and I should like to show some of his preparations which he has so kindly given to me.

This is the section from apex to base of a coal miner, age twenty, who worked on the coal face in a semi-bituminous mine. You will note that there are focal collections of dust in the lung and many of them. Some have coalesced to form larger collections. However, the point to be made is that there is little or no focal emphysema, associated with these deposits in this instance. Thus, the - not all cases showing focal deposition of coal pigment in the dust, in the lung, are associated with this focal emphysema, as seen when the specimen is met. That does not mean that it may not develop, but certainly a case in point, is that not all cases of dust are accompanied by focal emphysema.

Another section by Doctor Goffe shows the deposits of coal dust in the lung and now the focal emphysema, associated with every - practically every deposit of this dust in the lung.

The next slide. Four points of reference at this time, I should like - I present the normal lung, a section prepared by us in accordance with the Goffe theory, and we see one lobe and another lobe of the lung of that side, and note the absence of pigment, the black foci that you see are cross-sections of asperducts; note the uniformity in size of the alveolar spaces. Note no focal emphysema or gross emphysema.

The next slide. Let us focus our interest now on this focal emphysema which characterizes the reaction associated with the deposition of coal dust in the lung and which is responsible, I believe, for most of the emphysema, focal emphysema, and thus responsible in large measure, for the respiratory difficulty experienced by some coal workers.

Here we see the deposits of coal dust in the lung. We see that the alveolar walls are pigmented with this deposit, that some of the alveolar walls are apparently thickened and then ruptured with projections into the adjoining alveolar space.

The next slide. We'll go hurriedly now. The higher magnification shows the deposition of this coal pigment or coal dust in - again in both sides, sometimes about a vascular trunk, forming thick collars, but the point to be made here is that this pigment is not concentrated immediately about the wall of the vascular trunk, but it forms a thick wide collar, and as we look at it closely, we find also the deposition or formation of thin strands of collagen, showing that there is a reaction to this dust as deposited in the lung.

The next slide -- and a connective tissue stain of that lesion, again the vascular trunk, the - again the heavy deposit of black pigment which tends to obscure the collagen which is formed, and which makes histological study difficult, but note also the blue collagen which has responded to this deposition of coal dust.

Now, is the focal emphysema that we see associated with these focal deposits of dust, due to the collagen deposition and formation which we see here in these deposits of coal dust? Is it possible that these foci have reaction, that they act as anchors and so inhibit the natural function of the lung in these immediate areas, or is it because the changes -- the next slide -- which this merely shows the area of higher concentration due to the response of free crystalline silica, due to pigmentation -- or is the emphysema due to changes in the vascular trunks? Or the blood supply to certain areas, as for example, here we see, a large vascular trunk sustained with elastic tissue? We see the lumen of this vascular trunk which is considerably narrower and smaller than normal. We see the elastic membrane and we see the deposit of coal dust about this vascular trunk.

The next slide. And, in other instances, again the wall of a vascular trunk, the inner elastic tissue and here we see the deposit of coal dust, and here we see the deposition of collagen, Actually involving and impinging upon these vascular trunks.

The next slide. Or, in other instances, we see the vascular trunk in the center of an area of pigmentation, almost occluded. Is it then possible that this focal emphysema which we see, and the respiratory difficulty in these coal miners, may be due to these changes in the vascular trunk?

The next slide. Or, is the emphysema and the respiratory difficulty, more directly due to the involvement of the pulmonary tissue per se? For example, in this instance, with deposition of this coal dust in the alveolar wall and its associated inflammatory response, could it be due to that, and incidentally, we see here a body which is elongated with segments, and also with bulbous ends. They simulate the asbestos bodies which we see in asbestos workers. I have six cases in which we have discovered similar bodies. These have been reported by a good group, a large group, of other workers. Whether that asbestos body is due to really an asbestos body or whether it is a reaction to an asbestos fiber which has been inhaled incidentally, or an asbestos fiber from some fuses used, I am unable to say.

And, we see now, the stellate lesions, changes per se, extension along the collagen, extension along the alveolar walls, disportion of the alveolar walls, degeneration of the alveolar walls, loss of function of the alveolar walls and its accompanying distortion of the alveolar space, and emphysema. This slide again shows the thickening of the alveolar wall, obliteration of the architecture of that wall, loss of vascularity, the deposition of collagen.

The next slide -- and here is a sedtion of the lung of a coal miner in which we see almost complete obliteration of the alveolar wall structures, replacement by deposition of collagen. The next slide -- and elastic tissue stains. We see adjacent walls of the elastic fibers quite prominent, we study the pigmented wall. It is difficult to see the elastic fibers, but I have not detected any significant different in the character or amount of the elastic fibers which are present or absent in these areas of pigmentation.

The next slide. And we see here, complete obliteration of the architecture of the lung, by an involvement of the perimysium, pigmentation, hilization of the alveolar walls. The next slide -- And, is the emphysema and the respiratory difficulty due to obstruction of the major air spaces or passages? We do find areas of pigmentation, association of reaction, expansion of the alveolar wall, and repression of the small air space.

Next slide. In other instances, we find that the respiratory bronchiole is filled with mucous or with inflammatory obstruction. Is it possible then that the vascular changes, that the hilization of the alveolar walls, that the obstruction of the respiratory bronchiole, which we see in these cases, is responsible, as individual factors, for this emphysema and respiratory difficulty, or is any combination important?

Now, I should like to show you quickly a few other slides. The next slide. Although focal emphysema is discovered in the lungs of many coal workers, nevertheless, it is not unique for the deposition of coal dust in the lung. It accompanies also the pulmonary deposition of other types of dust inhaled by workers in other forms of occupation, as for example, and quickly, the lung of a granite cutter.

Again, we see these areas of reaction to the dust, and we see emphysema about these foci. Here and here. The next slide -- A molder, in an industry, and we see the deposition of dust with its reaction, associated reaction, but not again the focal emphysema. The next slide -- I'm sorry, let's go back to that slide. This is a diathermacious earth worker and we see the reaction, and the focal emphysema.

The next slide -- A molder, and we see the black pigmentation, focal emphysema localized about the pleura, about the areas of deposition of dust, and also the coarse emphysema. The next slide - a magnatite workers for thirty five years, again the focal emphysema and the areas of reaction to the free crystalline silica, which this worker has inhaled, with collars, irregular pigment extending into the alveolar walls, in this instance.

The next slide -- a hematite worker. Again, the emphysema which seems to be more prominent about the areas of dust deposition. The next slide, a graphite worker, again, the focal emphysema. The next slide - a carborundum worker; again, the emphysema which seems to be most prominent about the areas of deposition and about the areas of reaction. The next slide, a limestone worker, and again the focal emphysema.

So I then submit that focal emphysema, although it may be more prominent in the coal workers' lungs, and although it may assume a greater degree of - may become more manifest in the coal miners' lung and may be responsible for much of the respiratory difficulty, particularly should it progress, yet it is not unique for the coal miners lung. Thank you. (Applause).

BY DOCTOR DRINKER:

Any questions to ask Doctor Vorwald? (No response). I suggest we have a five-minute recess.

(Recess taken from 11:20 to 11:30 A.M.).

BY DOCTOR DRINKER:

The final paper this morning is by Doctor Fletcher on the Epidemiology of Coal Workers' Pneumoconiosis in Wales. Doctor Fletcher.

BY DOCTOR FLETCHER:

I'm afraid the title of this paper is a little bit wider than that. Strictly speaking, I'm going to talk about the Epidemiology in Great Britain, because some of the great interest in the epidemiological features of this disease in our country, is the difference in the prevalence of this disease in the different parts of the country.

So, first of all, for those of you who know as