FILE NAME: Saranac 7th Symposium (SSY)

DATE: 1952

DOC#: SSY013

DOCUMENT DESCRIPTION: Transcript of Presentation by Dr. Fletcher with Discussion

TAKEN FROM PAGE 207.

BY DOCTOR [redacted]:

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

little about the geography of our country as I know about yours, I'm going to put a map up to show just where the coal fields are in Great Britain. We have, first, here, is the country.

The coal deposits in Great Britain are here in South Wales with little fields neighboring which are very unimportant in size. Then there is a very small field hidden at the moment, just coming out there, in Kent, some little fields in the Midlands, a very big field in Yorkshire and another one in Lancashire, a big one up in Durham, going out into the sea, another little one in Cumberland going out into the sea here, and other ones in Scotland.

And, where our work has been concentrated is in Wales, but we have also investigated the one up here in Lancashire and the one in Cumberland. By and large, the greater part of the coal in Great Britain here is bituminous, but in Wales -- next slide -- the coal grades are -- here is the coal field in detail, from anthracite over here through semi-bituminous or what we call steam coal out here, with bituminous coal out in the west. There is a complete spectrum of coals in South Wales from anthracite to bituminous.

Now, the disease of coal miners pneumoconiosis that I'm going to talk about was very fully described, I should

say pathologically described, over a hundred years ago in Scotland, and the disease then appears in British history to have disappeared. The experts said that there was no such thing now in about 1910, as the old miners asthma, the black lung of miners; it had disappeared and that was attributed to improved working conditions in the mines.

But then, as radiography began to be more widely applied in Great Britain, it was found that miners showed X-rays that looked very like silicosis and then the pathologists got interested again, and particularly in South Wales, described the sort of pathological picture that Doctor Vorwald has described to you, and in 1929, miners were admitted to Workmen's Compensation for the first time and some official figures as to the prevalence of this condition became available.

And here we start actually going through various legal delays, the first cases certified were in 1931. These are years along here (indicating on slide) and the height of the column represents numbers of men officially certified with the disease in each year from 1931 to 1947. There is a gap there because the legislation changed, and then there is '48, '49, '50. This is South Wales where about just over a hundred thousand miners work, and this is the whole rest of Great Britain where seventy thousand -- six to seven hundred thousand, I should say, work; seven times

the number of men here, as in Wales. These cases after this gap here are not comparable strictly, because the legislation changed slightly and the standards of diagnosis changed slightly but not very greatly.

You see the steady increase throughout the 1930's and then the very abrupt increase through the war. I'm not going into the reasons in detail; it was partly due to the increase of dust during the pre-war years, due to mechanization, also due to change of legislation during this time, and also due to the fact that during the war, miners could not get out of the mines except on medical grounds.

Wages went up in many industries and many men found pneumoconiosis a way to get lighter but equally arduous work, and so legislative, social, I think generally, epidemiological reasons for this tremendous increase. I just want to draw your attention to the increase of the size of the problem in South Wales.

These workers increasing during this time, over twenty thousand men were certified with this disease and removed from the industry. In 1945, over one in three of the underground workers of two mines were removed from the industry for pneumoconiosis.

In great contrast to this tremendous prevalence in South Wales, by certified figures, we have the very much smaller prevalence in the rest of Great Britain. The level

seems to be easing off in South Wales; it appears to be still rising in the rest of the country.

Well, now, in South Wales itself, there are also quite important changes in the certification prevalence as we look across the country. Over in the anthracite area here, this is a map in which little dots imply coal mines employing more than a hundred men, and these are contour drawings on mines with the same prevalence of certified disease over this period '40 to '45. The blacker they are, the worse they are.

The black here is over seventy per thousand per annum, and mostly the anthracite, with a tongue out here in the bituminous area, and the surrounding bituminous areas were very much lower prevalence.

Well, now, the Medical Research Council did a big investigation of this problem in 1938 and with Doctor Hart and Doctor Haslett and a lot of other people, and they confirmed, from survey work, this distribution of the disease in South Wales, the higher the rank of coal, the more the disease. They failed to show any direct relationship to any particular component of the coal such as the free silica or the ash. There was a general relationship to the total amount of dust. The disease, they pointed out, was quite distinct from classical silicosis and from their work, this rank of coal hypothesis, that high rank coals, anthracite

coals are more harmful than the bituminous coals, the soft coals, that came into being and has had wide currency, and that is Although, as I say, there was no direct evidence from their work, for this hypothesis because the anthracite mines were dustier than the steam coal, which, in turn, were dustier than the bituminous.

Well, now, to go on, owing to the very grievous situation in South Wales, the Medical Research Council set up a special unit in 1945, which I have had the pleasure and honor of being associated with since that time, and we have done a good deal of work into the epidemiological side - the epidemiological features of this disease.

The work has been done by my colleague, Doctor Cochran, and I really feel rather shy coming here and talking to you about work he has done. All that I am going to tell you about has been work for which he has been responsible and the credit goes to him and his team.

The objective of this epidemiological work has been, first, to try and see if we could get any explanation for these epidemiological differences as shown by certification figures, and secondly, to see if we could guide dust suppression by establishing safe limits of dustiness.

Now, the method -- next slide -- has been to study men and their dust exposure. Now, what sort of men

did we study? The sort of groups that have been used for epidemiological work have been the following: Miners attending hospital. I'm ashamed almost to put that on a slide, but I wouldn't do it except that quite prominent papers have been published on miners attending hospital, and clearly they are -- even a highly selected group, even if the doctor himself doesn't select his patients -- they have been selected for him on a very curious and unpredictable basis.

A lot of papers have been published on applicants or receipts for compensation. All the figures I have shown you so far as we have gone, are based on that sort of population, but again many-fold motives effect a man's application for compensation, and it's difficult to use that type of population for active work.

You then have more carefully selected populations. The ideal population would be all the men who have ever worked in an industry, whatever they're now doing, but clearly, that's impossible. What we can do is take selected cases, all the cases in a mine and selected occupations, all the underground workers, all the underground workers and surface workers, because miners tend to go from underground to surface from time to time, or complete mining communities. We have adopted, at various times, the populations three to six.

Now, the next important thing is to insure that you get all the population you're after. Here is, just to emphasize this point, the prevalence of disease according to our categories, 0, 1, 2, 3, simple pneumoconiosis, massive fibrosis, to a hundred consecutive cases attending the certifying panel at Collier. You see the first two are not represented, miners and ex-miners admitted to our ward, high proportion have exited; ex-miners in a town, this distribution; surface workers, and the designed underground population, and you will see that these three all agree fairly well, but the certified cases and the hospital cases show a gross distortion of the distribution of disease, although all these cases come from the same geographical area.

These are the collierys we have studied which have been in South Wales. Some of the collierys already have been studied by the Medical Research Council in 1938. We have re-read the films in our classification and used their figures. We have also, particularly carefully, studied some steam coal mines, this group here in South Wales. Steam coal is our semi-bituminous, and an anthracite mine with very little disease in it. This is another steam coal mine, and two mines in England with rather low concentrations of dust, two mines in England in Lancashire and Cumberland with very high dust concentrations reputedly, from

all we could hear, but very low certified incidence.

We hoped there to get a real contrast between the conditions in bituminous and anthracite mines in South Wales and the general relationship between dustiness and rank outside South Wales, to get mines which were really dusty but had low rank coals, and those are the two.

Now, this slide is attributed to Doctor Cochran, the percentage of the underground-surface populations which he has managed to X-ray in each of those mines are given there, and the only two cases, in this mine here and here, in which he was not fully in charge of the survey, there were early surveys, is the proportion below ninety-eight percent.

The important aspect of that is shown in two figures -- next slide. At these two collierys where we didn't get ninety-eight percent of the population, you will see that the proportion varies with age. The statisticians found difficulty in the casuals young, and in the equally old. The middle-age you get easier. If you don't get ninety-five percent you get seventy-five percent; both of these collierys you got over seventy-five percent of the population, but you've got a distorted sample. It is essential to get a hundred percent population.

The next slide -- and one colliery where he got a hundred percent, Doctor Cochran analyzed the amount of

disease in relation to pneumoconiosis, inactive and clinically significant tuberculosis, on the radiograph and the first two hundred men came along willingly for examination. The second two hundred came along after some argument, and the third two hundred had to be frog-marched in, and you will see that people with pneumoconiosis come forth readily, the people with tuberculosis lag behind, so if you only get sixty percent, you have missed quite a lot of your disease. You get adversely distorted samples in relation to disease, a problem which is quite often omitted in reference to this point, and which is commonly omitted in survey work.

Well, now, some results -- next slide. Here are just the crude prevalences of these twelve collierys I have shown you, just the sort of thing you get from X-rays of these populations. Here is an anthracite mine, prevalence Category 1, 2, 3: Massive Fibrosis, PMF is short for that, quite a big range of these prevalences; these two English collierys with the low prevalence and you will see that this does, by and large, conform what is shown by certification figures, that if we're interested in dust exposure, we can't use this crude prevalence, and the question is what dust exposure should we examine in relation to this disease.

This slide shows figures from all these populations in relation to age, years spent underground and years

spent on the coal face, all lumped together, the whole lot, and you will see that in relation to age, there is a tapering off after the age of forty-five; there is no increase in the prevalence of the disease. This is Category 1. You take underground work, there is a smaller tapering off; when you take miners on the coal face, then there is a steady increase with increasing years on the coal face, and it was that argument and various other arguments that led us to concentrate our attention in relation to dust exposure to the coal face worker and to dust at the coal face.

And, what we have done is this: For our relationship between dust and disease, technique of field surveys, we have the unit, an X-ray team and a dust team. The X-ray team takes the X-rays and takes industrial histories. The X-rays are read by the duplicate reading technique I spoke to you about this morning. The industrial histories are carefully screened and from it we select pure face workers. They are defined as men who, during the ten years before the survey, have worked on the coal face for more than one year and worked in no other dust occupation for more than one year. We limit ourselves to ten years because our dust exposure may occur today, but the disease is produced in the past. We have, therefore, to multiply by a factor produced in the dust history in order to make the dust computation relative to the dust we have observed, and we do not

think we can do that over a greater period than ten years, so we restrict ourselves to pure face workers over ten years.

The dust team goes to the coal faces, all the coal faces in the mine using the precipitator, and they take dust measurements in terms of particles, c. c. or more strictly speaking, and I apologize, per millimeter, twenty-five microns, and we have now adopted a technique whereby the dust is not taken at fixed positions on the coal face, but is taken at the working places of colliers selected on a random basis, so that the dust measurements do represent the true measure of dust exposure of those men.

If the man selected is an absentee, well, no dust is collected that day and naught is contributed to the average, because the man wasn't exposed to dust that day. We introduced that relationship to the actual exposure of the man as closely as we can.

Then, we take the history of the mine as closely as we can in terms of production, ventilation, all the rest of it, in order to see how relevant this sample here is to the past and introduce a multiplication factor where necessary, but at the same time, through the filters, we take the analysis and we get an index of total dust dose expressed in particles per c. c. times years, that is the

concentration of dust multiplied by the number of years the man has been exposed to it, and on the other side, by multiplying that by the proportion of free silica in the dust, we get a total of silica dust.

Now, then, this technique of relating dust dosage to amount of disease is a standard pharmacological one, but I want to pay tribute to my colleague, Mr. Roach, who has done all our dust measurements, for the fact that he pointed out this particular method of expression to use, Doctors, who ought to have known better. Previously we were just taking an average dust concentration times an average years of all the men, but ignoring the great differences in the distribution of period of exposure between the different mines. What Mr. Roach suggested, we should do, is to take for each man the amount of time he had spent on the coal face, multiply that by the number of years and get, for each man, a dust exposure in terms of particle years, then group the man according to different groups of dust exposure and see the percentage in those groups showing radiological change and this is the result in all the pure face workers at eight of those twelve British pits where we had the dust information. Four of them we didn't do dust samples. Quite a nice sort of steep curve with the only dip here.

Well, now, we were interested in the difference

between the English mines and the Welch mines, so let's see what happens when we separate them. The dip comes right out and we find beautiful smooth curve, and this curve is fitted by less squares to these figures here which is quite different from the English.

These six Welch pits are all mutually consistent statistically, and statistically quite different from the English here. Here is the dust dosage, and they do show a quite clear difference in the relationship of radiological abnormality and dust exposure.

Now, then, why this difference? Well, it's natural that we should say 'Hurrah'; it is obvious that this is silicosis, anthraco-silicosis; you've only got to look at the American-European literature to see that, and it is the responsibility of silica, and so we take these five mines, the five not six, and we look to see. The range in ash content, nothing much in it; the high content is inone of the English mines; total silica, nothing in it; free silica, this should be 0.4 -- I'm sorry that's a misprint -- and you see again that the English mines fall in the same sort of range as the Welch Mines; calcium; albumen; and the only striking thing is the volatile matter. These Welch mines happen to have this range where both the English mines have the low volatile. We haven't yet managed to study low volatile Welch mines, so we don't know how

important this is, but it's the obvious point at the moment.

Well, now, let's just look at the free silica dosage against dust. That's in the six Welch pits, and I want you to remember that other slide, that beautiful steep curve, and the very visible sigmoid adopted from it, and it's very clear that the silica is not relating to the abnormalities as well as the total dust.

Well, now, what can this mean, this difference between the Welch and the English mines? I think that in looking for the silica difference, we're really barking up the wrong tree altogether, and for this reason: That in talking about active and inactive dusts the other day, I think I suggested to you, or perhaps I didn't, but it's in my written paper, that you might define an inactive dust as one in which there is a lot of dust in the lung in relation to the amount of reaction to it, but as an actuve dust is one in which there is relatively little dust in the lung in relation to the amount of reaction.

Cork - quite a little cork, produced fibrosis. In coal, in iron, you get a large amount of dust with relatively little fibrosis, but if this difference between the English and Welch mines, were between the bituminous and the anthracite and the steam coal mines, if that were due to differences in the fibrogenic properties of these dusts, we would expect, pathologically, to find a greater reaction

in relation to the amount of dust in the Welch mines than we find in the English mines. Now, hitherto, the amount of material we've got from English mines is relatively scanty, but we have quite a bit and we don't see any such difference. If you look at an English bituminous miner's lung, Professor Goffe will say, 'I can see no difference in this lung from a Welch mine - miner's lung, except there seems to be less dust in it'. The amount of reaction in relation to the amount of dust is the same, but there does seem to be less dust in it, and that at present is our thinking, that for some reason or other the bituminous coal dust either doesn't get into the lung so easily or having got in, is easy meat to the phagocytes and they just walk straight out with it again, a very difficult hypothesis to test.

We're trying to do that with animal experiments, but it's going to be quite an elaborate statistical problem to get proof of that hypothesis, but that's where we're sitting at the moment. We think there must be some difference of some kind in the retainability of these dusts. I can say straightaway that it doesn't lie in the size distribution of dusts.

Well, now, that is as far as we have got in relation to these epidemiological differences. There is this difficulty. We can't yet explain it, and at the time, we

have set for the future, a plan to carry out simultaneous dust and radiological surveys at twenty mines scattered throughout the whole of Great Britain, which the coal board are organizing in collaboration with us, and we hope to overcome the inaccuracies of the historical factor in this way, and from follow-up surveys of this kind, to get really good evidence, more precise than we've got at the moment which, associated with and in addition to, animal experiments, may help us to solve the why's and wherefore's in this difference we have found.

Now, what about safe limits? Next slide. Here we have, for the Welch pits, where our working data is much more complete, these dose response curves for Category 1, and for Category 2, and more and Cateogry 3, and here we have the dust dost. These are sigmoid curves, and are, of course, sound distribution curves. That is to say this curve here represents an ordinary - an ordinary normal distribution curve, and the fifty percent point is, of course, the most accurate point to take the relevant point on it from, and I have mentioned in my paper that our ratio between our categories is roughly speaking, but only roughly, linear, is about half as much dust again, at the fifty percent point here; two and three, you can see it isn't quite as accurate as it should be, but it's not bad, and this is our evidence that our categories are related to dust exposure.

Now, then when I putblished this in the Screed, I shall leave out these figures here, and I hope you will forget them, because obviously, this is quite a dangerous graph, because you can read up from here, if a man is exposed to five thousand particles per c. c., by the by, a thousand, approximately, particles is, I think I'm right, aren't I, Professor Drinker in saying it's approximately twenty-five to thirty million particles per cubic foot? I know you talk about these different things, but perhaps you better leave it untranslated at the moment.

Anyway, a man is translated to a thousand particles per c. c., which is about the official approved levels in Great Britain, for five years, fifty percent of the men will have Category 1 in five years. Ten years, fifty percent will have Category 2. Does it matter? I think we'll leave that discussion until Doctor Hugh-Jones has told you something about the disability.

Clearly, from this sort of chart, you can read off what will happen to men if they're exposed to these particular conditions for various periods of time, and obviously, I suppose to some of us, should forget the political consequences and just publish his results and let the workers and the employers fight it out, but I think that it's better at the present, to publish that graph without the figures on it for the future, so I hope you'll try to

understand why I omit the figures from the publication. It's also a matter of discretion, because in these figures we have got our historical factor which is a little bit of guess work, and these figures here are only relevant to the particular range of dust concentrations that we have observed.

We have found, for instance, that if you separately analyze years and particles, that for the same -- sorry, that for - yes, the same period of time, twice the exposure produces more than twice the amount of disease. That is to say concentration is more important than time and these figures just represent an even weight of concentration and time. So much about safe conditions; that is our approach; I recommend it to you as a very valuable method of expressing this relationship.

I now want to turn to discuss what we refer to, conversationally, as the two-disease hypothesis which Doctor Vorwald has already raised with you. We find, in simple pneumoconiosis, we call it, these discreet capacities throughout the chest, and we find these massive lesions. Are the massive lesions just due to large quantity of dust in the lung or are they due to the action of some other agent and dust?

Doctor Goffe's view is that these lesions are tuberculous, as Doctor Vorwald said. He finds tubercle

bacilla in fifty percent of them at post mortem, but in the other sixty percent he doesn't find them. He says that the histology looks to him like tuberculosis or the same. Doctor Vorwald won't support him there, and I can't enter into that, but we have got this large number of cases without definite overt evidence of tuberculosis at death. The explanation on the tuberculous hypothesis is that the tuberculosis has died out leaving its scar behind it, but I don't like that very much, because we do see cases that progress rapidly right up to death, and even in those cases that have progressed right up to death, we have two in which we failed to find any definite evidence of tuberculosis. It is possible that the tubercle bacillus initiates some process which then becomes self-propagating. We know that the dust in the lung is mobile, even in these little foca, because a man with simple pneumoconiosis, when he gets bronchitis, will cough up dust in his sputum many years after he leaves the mine, and it may be that once an infection starts in the lung, the rest of the lung tends to move up in that focus and then starts a propagating process that is due to the dust. That is a possible way of explaining these progressing lesions after tubercle has died out.

But we still believe that there is a lot of evidence for the tuberculous process and certainly for there

being two different disease processes involved and the evidence we have, I want to just discuss with you now briefly.

First, the Europeans say that these massive lesions are due to the action of silica. If so, we would -- sorry, will you just skip the next four slides; they're just pictures that Doctor Vorwald has already covered on the pathology. I think I can skip that, so come on to the next table. I just put this in in case Doctor Vorwald hadn't covered it.

Here we come to cases that Professor King in London and Nagelschmidt have done analyses on, on the whole lung in relation to the pathological group, normal, slight simple, marked simple, early coalescent nodulation and massive fibrosis, and you will notice there is no difference in these groups in proportion to the amount of free silica anywhere in that table.

If these massive lesions were due to those cases with a lot of silica in them, then we ought to find a higher proportion of free silica in these massive cases, than in the simple cases and you will see nine, ten, eleven, so we don't think that it's due to silica.

Now, evidence then, for their being two processes in the progression of the disease. We have followed cases over various periods of time, using other people's previous X-rays in this group, and our own X-rays here, and in simple

pneumoconiosis, with dust exposure, without dust exposure, and with dust exposure, we had eighty-one cases Category 1 to three without dust exposure. None progressed; two hundred sixty cases with dust exposure and eighteen percent progressed, so in smaller interval, our own X-rays, dividing no dust exposure, minimal, engine drivers underground, and under control code, none progressed without dust exposure; 21. -- 2.1 with minimal and 7 percent with dust exposure over this short period. Those are published already, so that pneumoconiosis progresses as duch only with dust exposure.

Now, you go on to massive fibrosis; there are two features, one the attack of massive fibrosis on simple pneumoconiosis, and secondly the progression of it once it starts. Here again, small groups of cases, on here, forty in each group, but with over five years dust exposure and no dust exposure. This represents the attach, the number of cases attached by massive fibrosis in that period, rather more in the no dust exposure group than the dust exposure. There is not a significant difference.

When you come to progression again, no difference, significant difference in the amount of progression or the attack rate of massive fibrosis in relation to dust exposure, so in relation to dust exposure, the two types of appearance, radiological appearance, behave differently.

Well, now, the way in which we've tried to further our knowledge of this question is an ingenious and courageous plan put forward by Doctor Cochran. We can't produce simple pneumoconiosis in animals and we don't think, therefore, we can really get relevant information about the tuberculous nature of massive fibrosis from animal work. Well, then, he said we'll have to use men and he took a complete population of a mining valley, the Little Rhondda, which is the Rhondda Fach, he took the complete population, thirty thousand and decided he would X-ray them all and he would then discover all the cases of tuberculosis in that valley, open tuberculosis, with the help of the regional hospital board, he would get those cases into hospitals and have sputum control of the majority - the remainder, rather, and by one-two testing, see whether he could reduce the rate of tuberculous infection or infectivity in that valley.

He would then compare the attack rate of massive fibrosis in that valley with that pertaining in a neighboring valley where no special measures had been taken.

This shows his success in X-raying that valley. I do want to pay tribute to Doctor Cochran for this remarkable performance. These are age groups along here, women here, men here, and this is the proportion of the total population established by private census at the time of the

X-ray survey, and the ground is well over one hundred per-cent. Where it gets over sixty, where it's difficult to get some of the old men and old women out, the survey was done in winter which is a mistake, because it's difficult to get them out and convince them their X-rays have any relevance to the health of the community, so you just compare this with the business figures published in this country, where a careful census has been taken according to county, and considerably lower figures, and this interesting drop in the young, the men presumably too busy, and the women with too many children to be able to leave and come to the X-ray.

But anyway, that remarkable achievement there, I think, shows that he has succeeded in getting nearly all the cases of tuberculosis, except some of these here, on X-ray. Well, now, the results of his experiment won't be available, of course, for five or ten years. But meanwhile, there are some interesting points from the prevalence point of view, which are relevant to our problem. This shows the numbers actually X-rayed, miners, ex-miners and non-miners, the adult females and the school children. The infants in the school areas were not examined, twenty-one thousand more.

In the course of this survey, Doctor Cochran and his team have - his team of four, in six months, they had

no less than twenty-thousand personal home visits to poor people in the valley.

This rather depressing picture shows the prevalence of pneumoconiosis in the ex-miners and miners in the valley. It shows there is quite a lot of pneumoconiosis, study of simple pneumoconiosis here without fibrosis in which the study of the attack rate and also quite a lot of massive fibrosis to study too, enormous population there, and the total numbers in this whole group are about nine thousand.

Now, this is quite a complicated one. I do apologize. The first point is that there is a logarithmic scale, and here I considered cases of infectious tuberculosis, let's say, with a positive smear on culture, cases of inactive tuberculosis, diagnosed on the radiograph as held inactive tuberculosis, and cases diagnosed as clinically active tuberculosis, let's say, cases requiring supervision, or in the cases where there is pneumoconiosis or massive fibrosis, and we found none in Category 1 in which the shadow looked like pneumoconiosis, but in many cases, on the others, it was awfully difficult to call the thing tuberculosis or massive fibrosis, so we put them here.

We have in category, infectious cases less than one percent, inactive tuberculosis and clinically significant cases. Coming into Category 1, there is practically

no infectious tuberculosis, and indeed in a survey of these, all these men and the neighboring valley, this one man is the only case out of four thousand miners in Category 1 pneumoconiosis, we had had a positive sputum on. There is decrease, insignificant decrease in the amount of clinically significant, but a rise in the prevalence of inactive.

Might we suppose that a little bit of coal dust in the lung increases the fibrogenic action of tuberculosis so that there is a tendency for a healed active scar and a reduction in the tendency for open tuberculosis, might that explain the facts? We then get a bit more coal dust in the lung, Category 2. Fibrosis is further stimulated. Massive fibrosis begins to appear. The inactive cases drop off as they're going slowly over there, but now infectious cases begin to appear when we get up to Category 3, when it goes right up to sixty percent.

Thus, in the case of massive fibrosis, of course, the Category is read on the background and your - it means unreadable, where it's that you simply can't read simple pneumoconiosis in the background . What's interesting is this reappearance of infectious cases and quite a portion or proportion of about one percent of the cases of massive fibrosis do develop positive sputum so that even though they are definitely these highly fibrotic lesions on our tubercular hypothesis, some of them may, for some reason or other,

and it's almost entirely in the age group over forty-five, break down and develop active tuberculosis, spreading tuberculosis.

It's perhaps of some interest that it's at that age that the mortality of tuberculosis rises in the general population. Perhaps it's something to do with resistance.

But, now, I can't speculate any further. Next slide -- and I must just point to the future for what is going to happen in this valley. And this is a map of the valley as it was in the first of September, 1950, with all the positive cases marked by pins on the map here. There is all the cases, in or out of hospitals. This is the same date, with the cases who were in hospital removed, and I think that it expresses the inadequacy of our present tuberculosis services, that is the very small impression that they make on the amount of infectious tuberculosis in a mining community, but the Regional Hospital Board said for this special purpose, they would open particular wards to which these men could be admitted and that was the position the 1st of October, 1951, the 31st of October, 1951, a year after the survey started.

This is relatively small, only about thirty-nine out of the original one hundred twenty-two are still in the valley, four months later only thirty-eight. The others are still in hospital or in their graves and the remainder

here are being carefully visited every month by a health advisor to insure they are really looking after their sputum, not going out to the pubs, and generally behaving themselves.

And what will be fascinating in the future will be to see what effect this has on the none too positive rate in the children, and then what effect it may have on the attack rate in massive fibrosis in those who already have simple pneumoconiosis in that valley, when compared with the neighboring valley in which this special procedure has not been followed, and I hope perhaps at your next Symposium, Doctor Cochran may be able to come himself, Doctor Vorwald, to tell you what he has found.

Well, now, gentlemen, I have spoken chiefly, on - about methods, just - and there is just time to refer to this chart. This is - I just have this one. It's a very provisional chart, this is the first of one year, but it's the mortality in age groups during the first year after the survey according to X-ray category and here is massive fibrosis, simple pneumoconiosis, Category 1 and 2, and that is the figure for the whole of England and Wales for the year.

Simple pneumoconiosis is just lined up completely with the normal population, normal male population; massive fibrosis is up, but I don't want to lay too much emphasis

on this. It's only one year and this particular kick here, which looks so impressive was due to four cases dying and two of them had carcinoma of the lung, so I think there is very possibly a significant difference up there.

But our point, I chiefly have spoken to you about methods and I'm afraid I have given you very few answers. I have, I hope, perhaps convinced you that there is some subtle difference between, at any rate, Welch and - Welch anthracite and steam coal dust and English bituminous dust. No same safe level would be applicable to these two mines.

I have touched on this two-disease hypothesis which we regard as very interesting and which we can not solve our problems at the moment, but we believe it to be perhaps more important than the problem of safe dust conditions and of simple pneumoconiosis. Doctor Hugh-Jones will show you this afternoon that disability in this disease is very largely attributable to massive fibrosis. We have some preliminary evidence that mortality is related to massive fibrosis. If simple pneumoconiosis is really not much more than an abnormal radiograph, then it's simple pneumoconiosis, that's caused by dust and if it is true that massive fibrosis is due to an additional factor, perhaps tuberculosis, maybe that we oughtn't to worry so much about dust concentrations which cause the simple pneumoconiosis, because the abnormal radiographs with little

disability and little mortality and what really we ought to do in our country is to remove tuberculous infection from the mining ideas and if from the mining area, why not from the rest of the country.

I don't say that is necessarily the answer, but it's the way we're thinking at the moment. I think that it's going to be exceedingly difficult to lower our dust concentrations in British coal mines, the level of which no man will develop the radiographic abnormalities of simple pneumoconiosis. It may not be necessary to exert that close - that collossal an effort with all its economic consequencex if we can protect the population from tuberculous infection, but first we've got to prove our point.

(Applause).

BY DOCTOR DRINKER:

Anyone wish to question Doctor Fletcher?

BY MR. URBAN:

I wonder if Doctor Abbott would like to comment - Doctor Hammond, would he like to comment on what he did along this line in Michigan?

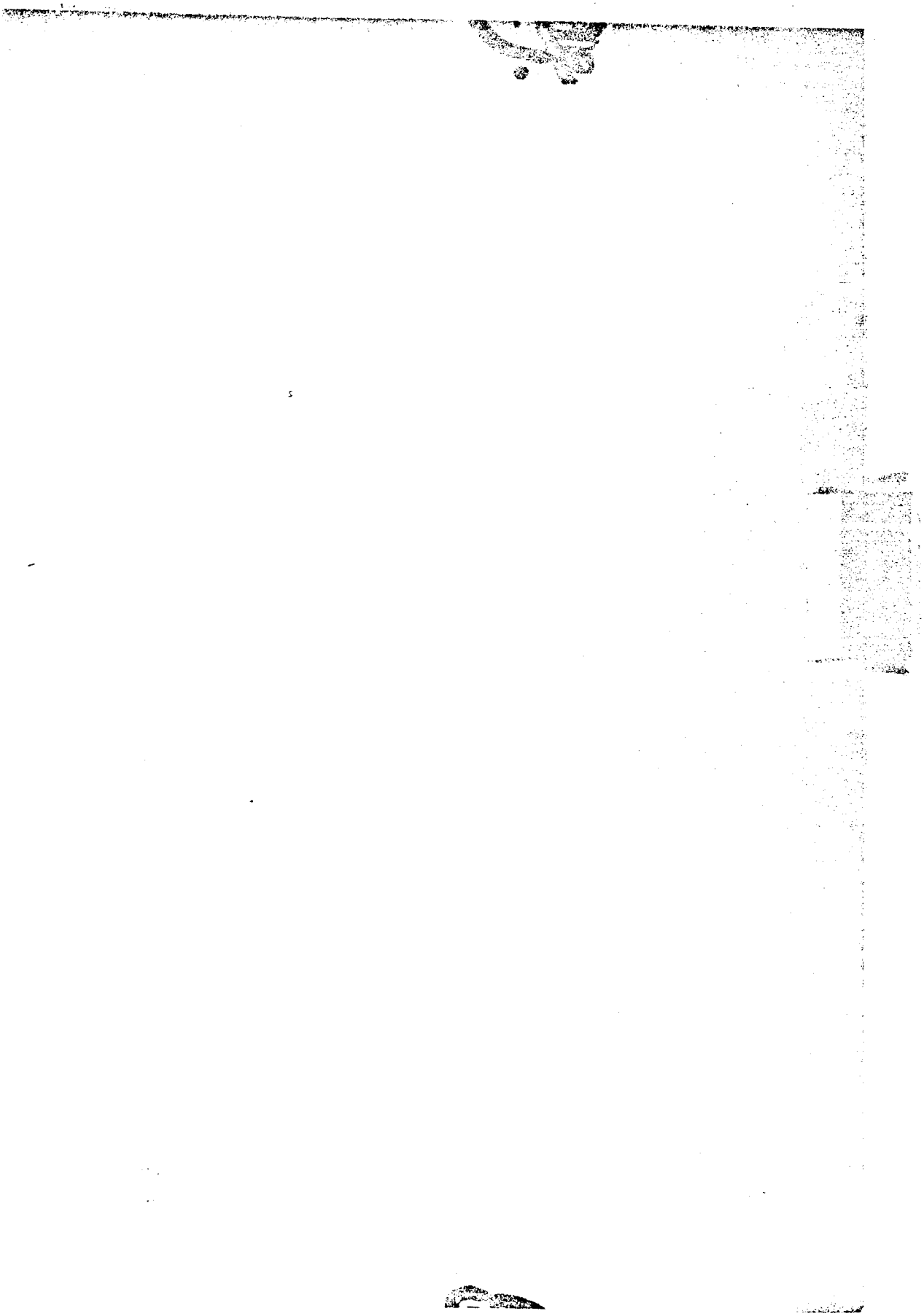
BY DOCTOR DRINKER:

I couldn't hear that.

BY MR. URBAN:

Is Doctor Hammond here?

BY DOCTOR HAMMOND:



Well, I haven't been in contact with the iron mines for a long time, Doctor Urban, and I don't know that I have anything to add to what Doctor Fletcher said in that line, but I think it's - his experience parallels our own pretty well. As I listened to him, I thought that we were getting right back to what we here in Saranac Lake were taught a good many years ago -- when I say a good many years ago, I mean ten or fifteen -- that tuberculosis was the important thing, perhaps in these massive fibrotic lesions. We felt up there that every shadow we saw in an X-ray at that time, in our own bailiwick there, would probably have to be interpreted in the light of being tuberculous.

Now, I think that we're probably getting away from that a little bit, but I still think that it is perhaps the important factor, and I know that up there, we very definitely felt that if we could protect these people from tuberculosis, then we had a much better chance of getting disability from silicosis in that field. We had a situation there which was perhaps somewhat unique in this country, as far as the problems of prevalence of tuberculosis were concerned. We had some family histories that were, well, to say the least, they were unusual, but by intermarriage, younger people become diseased with tuberculosis, the parents are then going into the mines and contracting

tuberculosis at an early date, sometimes dying with their boots on. Those days now seem to have disappeared pretty well since we have better methods of exhaust ventilation in the mines, but those of us who worked in that area, and there are a number here, I think were pretty well convinced that in the majority of cases if we could control that, we could prevent disability from these cases.

BY DOCTOR MOTLEY:

I'd like to ask Doctor Fletcher, if in the epidemiological study they have made, any correlation with the study of the man? We have occasions to study buddies who were in the war together, in some cases even brothers, and one man may become severely disabled, where one man may show only slight disability. The factors we have thought of is sinus infection, mouth breathing. I know Doctor Schepper, when he visited my laboratory from South Africa, made much of the physical examination they gave the white men there before they permitted them to work in the gold mines. They gave them a very rigid physical examination and in correlating dust count, it seems to me there might be some correlation with the rapidity with which they develop changes, and it's a physical finding and I wondered if they had made any attempt to correlate that.

BY DOCTOR FLETCHER:

One man being taken and the other left. It's like

the Bible call, the last Judgment. We have got that too; one has massive fibrosis and the other hasn't. Two men work the same place, one man gets a huge mass of shadow and the other doesn't, one is disabled and the other isn't. You could say one has tuberculosis and the other hasn't. Whether in two men exposed to the same dust conditions, one will develop radiological abnormality and the other will not is a problem we discuss at great length.

We have, at the moment, got a group of miners who have worked on dusty coal faces for more than twenty-five years, in whom we can find no radiological abnormality, we hope to trace those up to see what happened and we hope to chase them into Professor Goffe's department to see if our X-ray is wrong or whether they can cope with disease so efficiently they don't take it, and I personally believe that some men have such competent phagocytes or something of that sort that they cope with dust that many others will be susceptible to.

My colleague works with animals, and he says that one will be exposed to dust and will cope with it, and the others may be susceptible, and he says that animals and men are the same in regard to dust. He may be wrong and I'm not.

In regard to South African experience, examination, prophylactics, Doctor Cochran has done a great deal

of investigation as to radiological activity and body type. By evidence, he has been able to sort his men into tall, thin, short, squat, and so forth, and there is no relation at all between these anthropological measurements and pneumoconiosis, simple pneumoconiosis; on the other hand, there is a relation between the tall-thin type and massive fibrosis. Either the tall, thin type are more prevalent to get massive fibrosis and tuberculosis, or when a man gets massive fibrosis and tuberculosis, he becomes thin and tall.

BY DOCTOR DRINKER:

Doctor Sander?

BY DOCTOR SANDER:

I'd like to ask, do you have a correlation, or have you made a correlation with emphysema in the various categories of simple pneumoconiosis, clinically significant emphysema, radiologically and by lung function studies?

BY DOCTOR FLETCHER:

As far as lung function studies are concerned, I'll leave that to Doctor Hugh-Jones. As far as radiological emphysema, I'm afraid we've been so far disappointed to read emphysema repeatedly on X-rays, that we have decided that a radiological reading of emphysema is worth about as much as the ink, the ink you bother to write it down with. But we do know our radiological categories are

unrelated to professor Goffe's focal emphysema. All we can say in the very advanced focal emphysema, Professor Goffe has got two or three cases, one of which published in his paper in the Faculty Radiologist, where there is gross focal emphysema, and the radiograph does show a kind of honey-comb pattern, so when we see a marked honey-comb pattern in a miner, we think perhaps he has got focal emphysema, and we have got about three X-rays of that kind, and the man has either died or we haven't got them. So I don't think I can say that; all I can say is where massive fibrosis develops in the last stage, there you can see the big bully and you can be pretty confident.

BY DOCTOR SANDER:

Did you feel that your first film on the left, Category 1 has emphysema?

BY DOCTOR FLETCHER:

I feel it has, but I'm not sure, I'm not definitely positive.

BY DOCTOR SANDER:

You feel, logically, there is emphysema.

BY DOCTOR FLETCHER:

I feel there is, but if you haven't done any careful repeat reading of X-rays to classify them as emphysema and correlate them with the physiological findings, but I would only say that where the emphysema is gross, and

I think that one of these films fell into that type or category, then I would be jolly cross with the physiologist if he didn't find some evidence.

BY DOCTOR MAYER:

Now, we can discard completely the term anthraco-silicosis here, and to consider this as a form of anthracosis with infection. If so, it's going to effect our attitude toward compensation because our laws in New York State compensate the silicotic. Now, would you have us discard that term anthraco-silicosis?

BY DOCTOR FLETCHER:

Well, if it's going to deprive coal miners of compensation, I would say you must go on calling it silicosis, but I hope you'll be putting inverted commas on each side of the word, because I just don't think there is any evidence that the very small silica content, you may have noticed how very small the silica content of this coal dust is, I don't think there is any evidence that this silica content is responsible for this silicosis, even when Doctor Vorwald points to the diagram and says, here we have the characteristic response to silica, in the middle of the thing. Well, I am reminded that Doctor Hebbleston maintains that reaction to coal dust, with age, collagen may develop and it's common for any scar, so I'm told by the pathologists, for collagen to develop, and it may be a

reaction to aging as much as a reaction to silica, and I just simply say that the anthraco-silicosis implies to me that this is caused by silica, and I don't know anything in the world literature to support that hypothesis.

BY DOCTOR McCORMICK:

I would like to ask Doctor Fletcher whether he would care to comment about the range of dust concentrations that you may find in British mines, and where he has been able to set up any sort of defens e marks below which his early pneumoconiosis does not occur.

BY DOCTOR FLETCHER:

The range is very great indeed, and due to a variety of reasons, the anthracite mines that have been very dusty in the past for various reasons. First of all, in anthracite mines, there is no danger of coal dust explosions. Therefore, ventilation is not quite such an urgent safety measure, and in general, anthracite mines have been characteristically, have had sluggish ventilation in the past. There is much more short-firing anthracite mines and they have been very dusty. Bituminous mines have to be very vigorously ventilated because of the danger of coal gas and coal dust explosions, and by and large, the dust production from the tibuminous coal doesn't seem to be so great.

At the moment, in Great Britain, there is an official standard of 650 particles for anthracite, 850

particles per cubic millimeter as approved conditions. Nobody states how long those approved conditions have got to be prevailing. That is to say, if a coal face is approved, if the dust concentrations rise for five minutes above that level or the average for a week or what. We feel that the right figure is the average for a month, and Doctor Righter is busy designing, has already designed a dust sampling instrument which will give an integrated sample for a period of a week and just give you one dust count to do at the end of a period of a week and we think that will be a sound basis.

Well, now, on those figures I pointed to you on the board, men will develop simple pneumoconiosis under those approved conditions. There is no doubt about that, so that the level will have to be lower if our objective is going to be to prevent all radiological abnormality, but massive fibrosis doesn't develop until a man has got at least category 2 in our experience, so that perhaps that's the level we ought to aim at, and we may not have to drop this too low to achieve that. Certainly in bituminous mines, we may have to be more strict than anthracite and steam coal mines.

There is one other point. I showed you a fall in the amount of infectious tuberculosis and clinically significant tuberculosis with a rise in inactive tuberculosis

in Category 1. Maybe we ought to give all our coal miners clinically active pneumoconiosis to protect them from tuberculosis. It is interesting that mortality figures for Great Britain, and I think other countries too, Great Britain quite definitely, have shown a low tuberculous mortality for coal miners.

Now, in Great Britain as a whole, Category 1 pneumoconiosis is very prevalent and there is very little 2 or 3. Maybe that is a - it's an advantage to a coal miner to have just a little dust in design, enough to protect him from tuberculosis, but not enough to disable him.

BY DOCTOR WARING:

I'd like to ask Doctor Fletcher if he has made any studies of the conversion of the tuberculous ties in relation to the development of pulmonary - progressive pulmonary fibrosis, massive fibrosis?

BY DOCTOR FLETCHER:

Unfortunately, we can't do that, because our miners have such a high disability rate. At the age of fourteen, when leaving school in Rhondda Valley where we had this figure, I think it's sixty percent mine coal, but the earliest, at the age of eighteen, which is the earliest we got a mining population, it's ninety-five percent, so our chance of watching that are very slight. We have got two miners in Category 2 pneumoconiosis, positive, and we're watching

them like anything, but unfortunately, one got nephritis and died so we have only got one, but in the future in Rhondda Valley, we shall have opportunities for that sort of thing.

BY DOCTOR FRIEDMAN:

I'd like to comment on Doctor Fletcher's remark that we could detect some of the difference in the body in the people who have conglomerate lesions in the X-ray and the physical appearance. We're studying about two thousand soft coal miners in Alabama; we have failed to establish any correlation between body build and the appearance, whether it be a simple type of pneumoconiosis or whether it should be a fibrotic variety. However, we have observed that where there is a coalescent factor and the infection becomes overwhelming, in the event he dies in a state of malnutrition, it becomes evident.

Now, the second point Doctor McCormick raised about the dust levels. I feel, and I think it is true too, that regardless of the safe level of dust in which a man works -- and that is within reasonable limits of safety -- if he works long enough in that environment, he will obtain the same type of exposure, let's say, in twenty years in a safe environment that a man would obtain in an unsafe environment, in a lesser period of time.

I don't think we should place too much emphasis

over the long range period of time on safe levels of dust, because of the individual exposed long enough toa dust, he will get the same effects over a long period of time which he canin a short period of time in higher concentration.

BY DOCTOR BRODKIN:

My name is Brodkin and I was going to ask a simple question. The incidence of scars is frequent enough. Has anyone made any correlation between the tendency to keloid formation and the extent of fibrosis?

You take two individuals. Each has the same scar over an area; one will develop -- irrespective of any infectious bacteria, one will develop a large keloid, the other one will develop a fine hair-line scar. Has there been any correlation of that?

BY DOCTOR VORWALD:

Who is he asking that of?

BY DOCTOR DRINKER:

Were you asking that of anyone in particular?

BY DOCTOR BRODKIN:

Well, anyone.

BY DOCTOR VORWALD:

I have no evidence - I have no evidence to show that, for example, individuals or subjects from the faces who develop keloids very readily, that they respond more readily to the deposition of dust in the lungs with the

formation of collagen. I have no evidence, but we do know of course, that collagen does form in the Negroid race and the Caucasian, people from Japan and elsewhere. Perhaps Doctor Orenstein could tell us something about that. Doctor Orenstein, Doctor Orenstein, can you comment upon whether there is, in the Bantu, the colored individual, presumably a greater tendency to develop keloids, and if so whether he is more prone to develop collagen in the lung due to the deposition of dust, than is one from a race without or not prone to develop keloids.

BY DOCTOR ORENSTEIN:

Why, actually, it used to be one of the extraordinary museum specimens that we had and still have, of our Bantu lungs and spleens and livers with enormous things that you used to call tuberculoma, huge deposits, and nowadays you don't see it any more. The specimens are old. I don't know why, I have no explanation. It would be purposeless to detain you here with trying to explain differences between what you see in colliery workers in, say, Wales, to what you see in the workers in Johannesburg, where they work in a high percentage of silica, and historically, it's a very complicated thing, so I don't think you want to take up your time, because I can only say this, in the Bantu and in the white man, the type of pneumoconiosis which is se in the first two and a half decades of

this century and the type you have today are as differnet as anything can possibly be, pathologically, microscopically and clinically.

BY DOCTOR VORWALD:

Certainly we know from evidence there is a species difference, as for example, the white rat is much more prone to develop collagen in a shorter period of time than is the guinea pig or the rabbit, to the pulmonary deposition offree crystalline silica, but again, I certainly have no evidence that the colored race responds with more collagen under the same conditions of exposure than does an individual from the white race.

Now, perhaps Doctor Fletcher would like to comment. Fletch, Charles, do you want to say anything about that?

BY DOCTOR FLETCHER:

I have nothing to say about that; I would hate to be charged with designing experiments on this problem.

BY DOCTOR ABNER:

Abner, Buffalo, New York. Isn't, logically, a keloid the same as a fibrous tissue appearing in the lung following silica and if it is, should the response to X-ray therapy be the same.

BY DOCTOR VORWALD:

Well, histologically, the actual architecture of the keloid, histological architecture is different than the

architecture we see in the silicotic nodule, but basically it's collagen and I assume formed in a different way. I can't comment as to whether the collagen in the lung would be as amenable to change under the influences of X-ray therapy as might be the keloid.

BY DOCTOR DRINKER:

All right, Doctor Friedman.

BY DOCTOR FRIEDMAN:

Half of our population in the coal mines in Alabama is colored and the other half is white. We have not been able to detect any difference at the autopsy table or in the Roentgenogram on both. Now, on the other hand, we have an opportunity to examine one Negro who had massive keloids, but his bone specimen didn't show any inflection of what his skin showed in keloid formation. There was absolutely no correlation that we could detect in our study. You can find just as much fibrous tissue in the white man as you can in the colored man and I say, as Doctor Vorwald pointed out, there may be more of an individual difference than there is a difference between the races. We can't detect any racial different in response to the dust.

However, only one thing, we should say in conditioning this statement, is the fact that the colored population in our community live in a little less pleasant

environment. They don't have quite the economic facility that some of the white people have and the mortality rate in our colored miners that work underground is greater.

BY DOCTOR BOETJER:

I wanted to ask Doctor Fletcher if there is any difference in the prevalence rate in the general community in the English mining area which you were showing us, compared with the Welch mining area?

BY DOCTOR FLETCHER:

There is an interesting point there. The male population, mortality rate, is very specific, White Haven Borough Council and the Rhondda Urban District Council, here is distinct lines of pneumoconiosis, here female rates are almost identical and the children mortality rates are almost identical, but it is interesting that the male mortality rate in the Rhondda shows a deficiency, particularly in the age groups.

Now, if you add in to the age mortality, the male pneumoconiosis mortality which is nearly all pneumoconiosis, massive fibrosis, then there is another argument here for supposing that the men dying here of massive fibrosis would have died of tuberculosis if they hadn't had their massive fibrosis.

BY DOCTOR DRINKER:

Can you people in the back of the room hear this?

Can you hear the speakers all right, because I must say I can't hear when they ask me, but it may be a reflection on me. Can you hear the papers all right, you in the back of the room?

(General response indicating "Yes").

BY DOCTOR MILLER:

I wish Doctor Vorwald would comment on the idea that concentration of exposure was more important, the duration of exposure was more important than the concentration.

BY DOCTOR FLETCHER:

Excuse me, the other way around.

BY DOCTOR MILLER:

No, I believe Doctor Friedman said that the duration of exposure to a noxious dust was more important than the concentration, maybe I misunderstood.

BY DOCTOR FRIEDMAN:

I said that the duration of exposure has to be taken into consideration, it was also a very important factor.

BY DOCTOR MILLER:

Oh, not the sole factor?

BY DOCTOR FRIEDMAN:

No, sir.

BY DOCTOR FLETCHER:

I might just answer that point. The evidence we

have got on that point is simply this, that if you - if you do your time, your response curve here, the miners who have been exposed to more than a thousand particles per c. c. and less than a thousand particles per c. c., we have got time along here and percentage effected here, then the people in the high -- that's concentration -- show a durve like that; the people in the lower dust concentration a curve like that, so that just for equivalent particle years, you have a smaller response if the actual concentration of dust was level, and that is purely provisional, and is not of statistical significance there, but it is a trend that makes us doubt the applicability of our figures, necessarily, to the whole range of concentrations.

BY DOCTOR VORWALD:

I should like to make one comment and that is with respect to the free crystalline silica content of the lungs. Doctor Fletcher pointed that out and he used the low free crystalline silica content of the lung as a basis for saying that probably free crystalline silica is not a factor in the reaction of the lung to the deposits of coal dust, correct, Doctor Fletcher?

BY DOCTOR FLETCHER:

No, there is no more free silica in massive fibrosis than simple, so the massive fibrosis isn't due to more silica in the lungs in the fibrosis cases than in

simple pneumoconiosis.

BY DOCTOR VORWALD:

Well, some of our experience would be different than that, in that we have analyzed some of the massive fibrosis lesions and some without, and we have found higher free crystalline silica substance in the higher massive fibrosis lesions. I might emphasize though that there are those where we have not found that.

I would like to say also, with regard to the free crystalline silica in the lung, we have examined many lungs of normal individuals, of individuals who have been exposed to free crystalline silica, but without silicosis, and of individuals exposed to free crystalline silica with silicosis.

Now, there is a wide range of values observed in the normal lung versus the lungs of subjects without silicosis, but exposed to free crystalline silica, and the lungs of subjects with manifest silicosis. There is a wide range, and this range overlaps, so that except for the extremes, the two extremes, we believe that there is relatively little value or one can give a little index to the amount of free crystalline silica in the lung as related to the degree or character and extent of pathology within the lung. There is no correlation.

BY DOCTOR GREENBURG:

How about the time factor, Doctor Vorwald, did you take that into account?

BY DOCTOR VORWALD:

Well, I can't specifically give those figures; it's over a long period of time. In other words, here are fifteen normal lungs which we analyzed and they all have free crystalline silica, a certain range from a low to - I forget what it was - twelve percent, Mr. Durkan, do you remember those values?

BY MR. DURKAN:

No.

BY DOCTOR VORWALD:

Then we have sixty or seventy-five lungs of individuals who have been exposed to silica by reason of their employment, yet without silicosis pathologically, and they have a range of free crystalline silica value which is broad that way.

Then we have forty-five or fifty silicotics, established silicosis of industrial workers, and they do have a large range, but this range overlaps so that except for the extremes, we can not place any correlation between the amount of free crystalline silica detected in the lung by our methods and the degree and character, extent of pathology, silicotic fibrosis in the lung.

BY DOCTOR GREENBURG:

Well, that is another way of saying what has so often been said, namely, that there is a high degree of persons susceptibility in individual variation too?

BY DOCTOR VORWALD:

Yes, but maybe Doctor Pratt wishes to comment on that.

BY DOCTOR PRATT:

I think there is one important thing more that should be added to that comment, that is that all that work was based on the percent of silica in the lung. We realize that the amount of silica in the lung ash correlates fairly with the amount of silica in the lung, but very recently using a simple technique to determine the amount of silica in the lung, that correlates a great deal better with the amount in animals and a number of animal experiments show nice correlation, but the amount of disease estimated in the sections and the amount of silica in the lung.

To quote one example of a case in which a lung showed fibrosis, another lung showed simple nodular silicosis. In each lung the percentage of silica was the same, but on the basis of total amount of silica, there was about one gram of silica in the whole lung, with silicosis, and one gram in the lung showing fibrosis, even though the percentage was the same.

Obviously, the difference is the ash from the

reaction dilutes the silica which is present, and I think Doctor Fletcher's figures could be a lot more convincing if they were based on the total amount of silica.

BY DOCTOR DRINKER:

I suggest we better adjourn and continue the discussion this afternoon. The next meeting is at 2:30 this afternoon.

-oOo-

(Adjournment taken from 1:10 to 2:35 P. M.)

PNEUMOCONIOSIS IN COAL MINERS (Continued) 2:30 to 5:30 PM Sept.23, 1952.

Chairman: O. A. Sander, M. D.

Pneumoconiosis in Coal Miners in Alabama

Louis Friedman, M. D.

Discussion

Pulmonary Function Studies of Coal Miners in Pennsylvania and West Virginia

Hurley L. Motley, M. D.

Pulmonary Function Studies of Coal Miners in Wales

Philip Hugh-Jones, M. D.

Discussion

BY DOCTOR SANDER:

Let's have the meeting come to order. We'll carry