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BY DOCTOR SANDER:

Doctor Vorwald, Members of the Symposium: I'm one who has been teaching students for the last fifteen

years, that pneumoconiosis is an overall term covering all inhaled dusts. It simply means dust in the lungs, and to me, limiting the term to those dusts which cause disease or cause symptoms, is like saying it suits our convenience now to say that the world is flat. We know Christopher Columbo said the world was round and they thought he was crazy at that time. We finally have accepted that. Well, now, it suits our convenience, maybe because of our legal friends, to say that the world now is flat again. To me, it's just as ridiculous to limit the term 'pneumocconiosis' to those conditions which cause symptoms.

Dust in the lungs -- we all have some pneumoconiosis, every one of us, those who live in the city or live in cities, even the farmers have some pneumocconiosis, because they inhale organic dusts. It's just as simple as that to me. Let's assume everyone who comes to your office has pneumoconiosis. Then, let's be specific after that. What is it due to?

Speaking of dictionary definitions, Doctor Johnston sent me this, and I think it's too good to pass up. I don't believe in dictionaries, and here is the latest definition, found in Blackstone's New Gold Medical Dictionary, the latest edition. Grinder's Asthma is an interstitial pneumonia due to inhalation of fine particles set free in grinding steel, and then it says, See Fibroid Phthisis, and

then you look up fibroid phthisis, on Page 770, and it says: Chronic slowly progressive pulmonary tuberculosis with extensive fibrosis and mild symptoms. That's the latest edition.

The only textbook I know which has defined 'pneumoconiosis' as an overall term, generic term, meaning any dust in the lung is Doctor Johnston's book. Now, you look up any medical textbook today, and it will say pneumoconiosis is fibrosis due to the inhalation of dust. We have the evidence of dusts which cause no fibrosis, and it's perfectly clear evidence.

We have it, first of all, with the iron oxide, and we have it with the tin oxide. We have it with the barium oxide and there are others. We certainly have it with aluminum oxide. Aluminum oxide doesn't show, because it's not as radiopaque, but it's deposited in the same way. We have it with carbon pigmentation, and in carbon pigmentation the pathologist says, there it is, the paro-vascular lymphatics around blood vessels. It looks like nodular silicosis, doesn't it? It's carbon pigmentation. If that carbon were radiopaque as is iron, tin and barium, we all would have nodulation by X-ray, every one of us. Then we'd agree that we all had pneumoconiosis, but because our chest films are read as healthy lungs, we can't believe that we have pneumoconiosis, but we do. We're all carrying

plenty of material in our paro-vascular lymphatics, and it's not doing any harm, not doing any more harm than these little carbon particles are doing in these paro-vascular lymphatics.

There is just a little difference in the coal pigmentation and coal reaction than you get with the simple soot in the atmosphere. Coal, being a hydrocarbon, causes irritation, undoubtedly, in the bronchials and there is an added factor of obstruction, which undoubtedly we'll hear about from our English friends and Doctor Orenstein, but soot in graphite and non-irritating materials, like iron is non-irritating. You can inject it under the skin and it is the same thing as a tattoo mark. There is no fibrosis. There is no keloid. There is no reaction.

I think we might look at these slides. Many of you have seen these welders sections, but I thought it would be quite important to review it again, because it is basic. I hope you can see the absence of any reaction whatever. Three out of four here have seen these, but probably they're worth seeing again.

This is the welder who worked inside of a tank for ten or eleven years. There was no attempt at ventilation so he had heavy welding fume exposure. Welding fume, you know, is ninety-nine percent plus, iron oxide, very fine dust. He had no symptoms. He had this nodulation,

which when we saw the film in 1935, we scratched our heads and said, he probably has silicosis. He has no silica exposure, and we wondered, is there possibly a free silica liberated in the carbon arc from the coating of the welding rod. After all, there is sodium silicate binder in the coating, and we investigated that and there was no evidence of any SiO_2. Well, this fellow, unfortunately, died a year later, and we were able to get his lungs.

May we see the next, please? And, this is the low part view, showing the deposits of pigment in the paravascular lymphatics. The next is a high power view. Note the absence of fibrosis. Here is a high power view, showing the extreme amount of pigment deposit in the paravascular lymphatics. That pigment is inside the paravasculars. Grossly, that lung looked just like an anthrapigmentation, which you see in most adults living in cities, but with some ferrous cyanide stain, we were able to prove this was iron oxide from his welding fumes. Note the absence of fibrosis, and in the next slide, is the one for connective tissue, showing complete absence of fibrosis, no fibrous tissue reaction, no intelligible virus.

We have the additional evidence from the tin cases. We have had - there have been two post mortem studies, one by Bartuk in Czechoslovakia, and the other by Doctor Hughes and both showed a complete absence of fibrosis in these tin

deposits. Unfortunately, I didn't bring my slides, but you all have seen the extreme pigmentation with the tin oxide, and we have two good post mortem cases, which show a complete absence of fibrosis, and just an inert deposition.

Bartuk did complete function studies on his cases and found no decrease in pulmonary function. The barium cases of Doctor Prendergast have no symptoms of any kind. There are not too many barium cases. There hasn't been that much barium oxide exposure, but there again, we have this marked deposition of inert material, which causes no fibrosis.

Therefore, I say there is such a thing as inactive dust and no matter how much iron oxide, no matter how much tin oxide, no matter how much barium oxide you inhale, you are not going to get symptoms. We have yet to see a case of tin oxide with symptoms. They haven't seen any in Cleveland where they have uncovered a good many. Bartuk had no case with symptoms, in spite of the extreme X-ray evidence of it. So there is such a thing, in my opinion, of inactive dust, and I don't like the term benign pneumoconiosis any more than you do.

I wish Doctor Gene Prendergast were here to defend himself. He coined the term, you know, about ten years ago. Benign implies that the other pneumoconioses are malignant. I don't like it for that reason because

they're not all malignant either.

I think inert pneumoconiosis would be a perfectly proper term for the pure iron oxide, the pure tin oxide, and the pure barium oxide deposits. I think it would be perfectly proper because we have the pathological evidence that it does not cause reaction, that it doesn't cause symptoms.

As for the retention or discard of such terms as anthraco-silicosis or sidero-silicosis, I think it would be perfectly proper to retain sidero-silicosis where you know that there has been both silica and iron exposure. Every iron miner, no matter where he works, who has any exposure to silica, and most of them do have exposure to silica, we have been looking for iron miners who have never had exposure to silica, had only hematite exposure, and we haven't found one so far. Every miner has a sidaro-silicosis if he develops anything at all. We know he has hematite in his lungs, and I firmly believe that some day we're going to be able to show that this hematite itself, in itself, is adding to the nodulation which we see by X-ray.

We have seen it in a few post mortems in iron miners, where there was considerable iron pigmentation, which undoubtedly helped produce the nodulation. I have yet to find, however, a miner who had only iron pigmentation, that is that he had a nodular pattern in his X-ray

film where the pattern was due only to iron pigmentation. We haven't found such a case and we may not, because all of the miners in the past, the older men who have nodulation, by X-rays, always helped drill the shafts, and the shafts, as you know, are in silica, in quartz. They're usually quartz veins, so even the mucker, who has only ore dust exposure, has had silica exposure in the past, because he usually helped in the drilling of the shafts.

I believe, firmly believe, that it is perfectly proper to retain the term sidero-silicosis. As for the coal miners pneumoconiosis, I'm definitely in favor of throwing out the term anthraco-silicosis. We know that there is a pathology, in the absence of any significant amount of free silica with coal dust. Doctor Fletcher and his coal workers have shown that very clearly, and you will hear more about that later. I do not believe that that term is proper in the majority of coal miners pneumoconiosis cases.

Now, as for pneumoconiosis itself, I'm in favor of retaining it, and keeping it as an overall term, and always driving home to whomever you're talking to, whenever you're thinking about it, that you and I, all of us have some pneumoconiosis.

Thank you.

TAKEN FROM PAGE 37.

BY DOCTOR VORWALD:

You have heard the representative from the legal

profession, and I think it is evident that that profession is placing upon the medical group, the task, and pleading for clarification and definitions of terms by the medical profession, for use and application by the legal group. They look upon the medical profession for those definitions and that clarification.

Our next member on the panel comes to us from England. Doctor Fletcher is Director of the Pneumoconiosis Research Unit of the Medical Research Council in Cardiff, Wales, and has had much experience with coal miners in that area, and he is also a lecturer of the Post Graduate School of Medicine, University of London, London, England, and I'm happy to introduce to you Doctor Fletcher.

BY DOCTOR FLETCHER:

Mr. Chairman and Gentlemen: I'm in slight difficulties, since this is the first occasion on which I've attempted to take part in a panel of this kind and I don't know to what extent I'm expected to answer the questions that Doctor Vorwald fired at me across the Atlantic, or to what extent I can throw brickbats at the - some of the remarks which I regard as highly debatable, which have been made already this morning. I will attempt to combine the two.

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pneumoconiosis proposed by the International Labor Office Conference in Sidney two years ago, that pneumoconiosis is a diagnosable disease of the lungs produced by the inhalation of dust, the term dust being understood to refer to particulate matter in the solid phase but excluding living organisms.

The term diagnosable is later amplified in the report as meaning the presence of signs or symptoms, with radiological abnormality as being a sign, but not always loss of function. To me, the word disease implies any abnormal condition of the lungs.

Now, it's difficult to define where normality leaves off and where abnormality begins. You can't draw any sharp distinction between the accumulations of soot dust in a town dweller long in a smoky city such as Sheffield, andthe early stages of pneumoconiosis, and, of course Zenker and Schuller describe pneumoconioses in the town dwellers in the apex.

But, I should think here, we want to increase the acceptance within the framework of medical knowledge of the statistician and if we do that, possibly even the lawyers may follow us and recognize statistical concepts, and I regard as abnormal, something which is two standard deviations away from the normal, and where we can measure that, we should admit that as an abnormality or come to some

agreement as to what the number of standard deviations we can admit are.

We can do that by surveying the normal population and discovering the frequency of certain radiological pictures, comparing them with the occupation we're interested in, and we may even say the pathologists to start, can become quantitative in their work so that they can follow suit with the radiologists and the physiologists. The physicians -- well, perhaps later, they too.

With regard to the pathological definition, that, of course, is useless in life, and I do deplore, I must say, for legal purposes, a definition based on fibrosis which can not be diagnosed without lung biopsy, because much as I like my colleagues, the lung surgeons, I think they already have a tendency to be too enthusiastic.

For purposes of compensation, for scientific purposes, I think we must be quite strict and recognize a process in the lungs attributable to dust. Now, that is pneumoconiosis, whatever the lawyers want us to say. It is up to the lawyers to decide what they want to compensate. Do they want to compensate a man for having an abnormal radiograph or for being disabled? Let them say what it is they want to compensate. Then the doctor, in his term, can say how he will set so sort of limit, with, I plead, the help of the statistician.

One of the difficulties of the conflict between the compensatable pneumoconiosis and scientific pneumoconiosis is psychological. The man who has pneumoconiosis, diagnosed on a radiograph by one doctor, applies to compensation and is turned down and is then disgruntled, or in an area like South Wales, where there are hundreds - yes, tens of thousands - of disabled men with pneumoconiosis, he develops an anxietystate.

Well, I suggest that clever doctors needn't be baffled by this any more than they are by tuberculosis. If we diagnose, on a radiograph, a hemolytical scar, we don't tell the man he has got tuberculosis. We ignore it, but we make a record in our own notes that he has had tuberculosis; he has some sign of tubercular infection. The same sign can be used in pneumoconiosis, and we have not found it difficult to explain to miners that they have dust in their lungs, but that is not compensatable pneumoconiosis.

Most miners have signs of dust in their skin where they have wounded the skin or have got dust in the skin. We can explain it is not a dust disease; it is dust in the skin. They have dust in the lung and they will accept that explanation.

Moreover, we have found the miner much more willing to accept the fact that two doctors may disagree in their opinion about an X-ray film than our doctors in service.

Active and inactive dusts, I don't think you can make any distinction without so much specificationthat the distinction loses any value. In the case of silica, a clearly active dust, if given in high dose, the effect will be clearly shown, but the same quantity spread over a long period of time will have no effedt whatever on an animal's life. Active or inactive? It depends on the dose.

And, for that reason, I am very much afraid of Doctor Sander's statement about inactive dusts, that soot is inactive. Krause and Gatner, recently in Germany, have confirmed the observations of Locke, that men working exposed to high concentrations of pure soot, 99.9 percent soot, pure carbon, developed radiological abnormalities, and I have seen them myself, identical with coal gas. Is soot radiologically inactive? No, not if it's inhaled in so much quantity that it displaces air from the lungs; solid material up against air in the lungs will cause a radiological picture, so soot is as radiologically opaque as edema fluid when it replaces that in the lung, and you see the shadow, and I don't like the quotation of two or three cases of tin oxide, barium oxide lungs and iron in the lungs as evidence that no matter how much of these dusts these men inhale, they would suffer no damage.

I could match Doctor Sander's pathological pictures with coal miners lungs, with just about as much

dust, just about as much reaction. All right, would argue Doctor Sander, there is no such thing as a dangerous quantity of coal dust. Coal is an inactive dust. Let him come to South Wales and see what happens when the man inhales enormous amounts or quantities of coal over long periods of time. Then, indeed, there is a reaction.

And, I suspect that these diseases due to the so-called inert dusts have their character determined very largely by the quantity of dust that is inhaled, and that any inert dust or so-called inert dust, simply because it is not silica, as the usual meaning for the term, any such inert dust inhaled in sufficient quantities, will cause liability to disability, especially when complicated by tuberculous infection.

Doctor Vorwald asked me, whether silicosis was a disease or a condition. I'm afraid that these two words have very little differennce in their meaning in Great Britain. It's one of these meanings that are different, of course, because we think we talk English and you think we talk English and actually I think our languages are, in some respects, different. But I would simply value the distinction, if it is necessary, of classical nodular silicosis as one particular form of silicosis, with the particular process in the lungs strictly attributable to the inhalation of silica, by which I mean crystalline free