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"The man who says 'It can't be done' is liable to be interrupted by somebody doing it."

Silicosis and Asbestosis.

A Joint Meeting of the Refractories Association of Great Britain, with the Yorkshire Section of the Society of Chemical Industry.

A JOINT Meeting of The Refractories Association of Great Britain, with the Yorkshire Section of the Society of Chemical Industry, was held at the Grand Hotel, Sheffield, on Friday, 13th April, 1934, at 7 p.m. when a General Discussion was held on "Silicosis and Asbestosis."

In view of the new ideas with regard to the causation of Silicosis, the meeting was particularly well attended.

The discussion was opened by the following three short papers :—

"The Normal and Silicotic Lung."
F. S. Fowweather, M.D., M.R.C.P., D.P.H., M.Sc., F.I.C.

"Silicosis : The Minerals which cause it."
Dr. W. R. Jones, D.I.C., F.G.S., M.I.M.M.

and

"Some Chemical aspects of Silicosis."
Dr. F. V. Tideswell.

Mr. W. Bain (Vice-Chairman of the Yorkshire Section of the Society of Chemical Industry), (British Belting & Asbestos Ltd., Scandinavia Works, Cleckheaton, Yorks.) presided along with **Mr. Alex Lomas** (representing **Mr. Wm. Lomas, J.P.,** President of The Refractories Association).

Mr. W. Bain. We have a long programme in front of us to-night. Unfortunately, Professor N. M. Comber, D.Sc., A.R.C.S., F.I.C., Chairman of the Yorkshire Section, is unable to be present. He has been called to London.

I have also an apology from Professor M. J. Stewart, M.B., Ch.B., F.R.C.P. who was to have given a paper on "The Morbid Anatomy of Silicosis." He also has gone to London as he had some examinations to conduct.

To-night we have before us a subject of extraordinary interest as it touches so many different trades and is becoming a subject of vast interest to technical workers and laymen. Many new ideas and authorities have recently come to light and I am sure that, to-night, we are going to have a real feast of information on this important subject, "Silicosis and Asbestosis" actually touches miners, stone-masons, asbestos workers, grinders and pottery workers as well as those engaged in the Refractories Industry.

In Sheffield, you have been faced with the problem for many years, but workers in the Asbestos Industry have only been faced with it during the past few years, and new developments have now taken place to safeguard the worker by ventilation. Many experiments have been carried out, and I, personally, have had the opportunity of coming in contact with the excellent work carried out by Professor Stewart and his colleagues at Leeds.

You will have the opportunity of joining in the discussion after all the speakers have given their papers. If there are any points you wish to raise after the lecture, please make a note of them.

THE SILICA CONTENT OF NORMAL AND SILICOTIC LUNGS AND ITS BEARING ON THE PROBLEM OF SILICOSIS.

By F. S. Fowweather, M.D., M.R.C.P., D.P.H., *M.Sc., F.I.C.*

FOR over two years Professor Stewart and I have been collecting and examining the lungs of miners, asbestos workers, sandblasters and others who have worked in an industry believed to carry a risk of silicosis. Determinations have been made of the ash and the silica content of these lungs, as well as microscopic and naked-eye examination for evidence of silicotic fibrosis, and of associated diseases—tuberculosis, bronchiectasis, pneumonia, etc.

In the present communication I propose to deal mainly with the chemical findings, though some reference to the pathological evidence must also be included. Though the work is yet far from complete I shall attempt to draw some conclusions from the results so far obtained.

Normal controls have been examined, to obtain evidence as to the amount of ash and of silica that are present in lungs which have not been exposed to dangerous dusts. Of five adults who from this point of view were considered normal, and whose ages lay between 23 and 65, the lungs showed ash from 3.95 per cent. to 4.66 per cent. of the dry lung tissue, with an average of 4.42 per cent. The silica content was from 0.12 per cent. to 0.20 per cent. with an average of 0.163 per cent. It is interesting to note that a railwayman who, on account of marked anthracosis, was not considered a "normal" from our point of view, gave figures of 4.06 per cent. for ash and 0.17 per cent. for silica; *i.e.*, in spite of the anthracosis the lung ash and silica fell within normal limits.

The results so far as the remaining cases are concerned (*i.e.*, cases of actual silicosis, or cases who may have been exposed to some risk of silicosis) have been arranged in two ways, namely, according to the amount of silica found in the lungs and according to the industry in which the patient had been engaged. I propose to consider mainly the arrangement according to amount of silica in the lung ; I shall not consider all the cases, but only those that appear to be of

special interest. The amount of fibrosis noted on microscopic examination has been indicated by Professor Stewart in three grades, which I shall call 1, 2 and 3, in order of increasing severity.

The first case in the list shows 6.25 per cent. of silica in the lungs. It is the case of a coal miner who died at the age of 62 from chronic empyema with a terminal acute pericarditis. The lungs showed considerable patchy fibrosis; microscopically the fibrosis was of grade 2. This man was a miner for 20 years, and then a farmer for 30 years. He must, therefore, have accumulated his 6.25 per cent. of silica thirty years before he died. In sharp contrast with this case is that of a sandblaster who died at the age of 27 of silicosis after working at this job for 13 years. He was only away from his work two months before he died. While the silica in his lungs amounted to 4.18 per cent., *i.e.* only two-thirds of that of the previous case; the fibrosis present was of grade 3. Another sandblaster died at the age of 26, with 4.00 per cent. of silica in his lungs and a fibrosis of grade 3.

Next in the list comes a South Wales coal miner, aged 53, who had 3.95 per cent. of silica in one part of the lungs and 2.69 per cent. in another. He died of silico-tuberculosis, with a grade 2 fibrosis ; he had been a miner for 32 years. The next case, whose occupation we have not yet ascertained, died at 69, with 3.92 per cent. of silica in the lungs. The degree of fibrosis in this was only grade 1. An iron ore miner, aged 42, who had followed this occupation for 24 years, had nearly 1 per cent. of silica less than the previous case, viz., 2.96 per cent., but died of pulmonary fibrosis and tuberculosis, with a grade 3 fibrosis. The next case, a tin miner of 65, had a grade 3 fibrosis with 2.84 per cent. of silica in the upper part of the lungs, while the lower part was practically normal, having only 0.33 per cent. of silica.

Of considerable interest is the case of a stoker who died at the age of 55 of cancer of the stomach. He had 2.00 per cent. of silica in the lungs, and though there was considerable anthracosis there was no evidence of silicosis and microscopic fibrosis is reported as below grade 1. In a miner of 66, with 1.83 per cent. of silica, the lungs had also a negligible amount of fibrosis; yet an asbestos worker of 40, whose lungs contained 1.54 per cent. of silica, died after 11 years' work in an asbestos factory of pulmonary asbestosis six years after he had left this work. His lungs showed a grade 3 fibrosis.

Next come two sharply contrasting cases of miners. The first, aged 55, had been a miner for 41 years. He had 1.40 per cent. silica in one part of the lungs and 1.12 per cent. in another, with a grade 3 fibrosis ; the second, aged 53, had 1.15 per cent. of silica in the lungs, with a negligible fibrosis. He died of cancer of the lung. Then we have another sandblaster, also young. He died at the age of 24 after having been a sandblaster for 7 years. He had 1.14 per cent. of silica in one part of the lungs with a grade 3 fibrosis and 0.28 per cent. in another. Yet another sandblaster, aged 58, after 15 years in this industry had 1.03 per cent. of silica, and a grade 2 fibrosis. Tuberculosis was also present.

A Group of Four Cases.

Of special interest is a group of four cases all showing the same lung silica content, viz., 0.89 per cent. The first, a woman of 26, had been engaged in the making of an abrasive soap for three years. She died of acute silicosis, with a grade 3 fibrosis. The second, a man of 44, had been a grinder for 28 years. He had a moderately advanced chronic silicosis, with a grade 2 fibrosis. The third, a man of 65, had been a cuphandler for three years and had been engaged "in rock and jet for 30 years before that." He died from malignant disease and his lungs showed only a grade 1 fibrosis. The last, a miner of 61, who also died of malignant disease, had negligible fibrosis. Notable among the next cases in this arrangement are a stonemason of 52, who had worked all his life at this occupation and who had 0.76 per cent. of lung silica, and a woman of 31 who had worked for 11 years in an asbestos factory, and had 0.53 per cent. of lung silica. In both a grade 3 fibrosis was present. Following these cases there are none with grade 3 fibrosis, but a stonemason aged 61 with 0.43 per cent. silica, another aged 52 with 0.33 per cent. silica, a sandblaster of 42 with 0.33 per cent. silica and an asbestos worker, aged 45, with 0.28 per cent. silica, all showed a grade 2 fibrosis. Still lower in the list are an asbestos worker of 37 with 0.24 per cent. silica and a sandblaster of 50 with the same amount and both show a grade 1 fibrosis.

The first definite conclusion which arises from a consideration of these results is that there is no obvious connection between the amount of silica in the lungs and the amount of fibrosis which is present. Thus a stonemason with 0.33 per cent. of silica in his lungs died at 52, and a worker in asbestos after only six years' exposure to the dust and then a fifteen years' interval died at 45 with 0.28 per cent. of lung silica, while a miner, after accumulating 6.25 per cent. of silica, lived until the age of 62, and all three had approximately the same degree of pulmonary fibrosis. The amount of silica which accumulates in the lungs is therefore not the sole factor on which depends the resultant degree of fibrosis.

When we try to find the connection between the silica in the dust inhaled and the degree of fibrosis the conclusions are not so obvious. The time during which the patient has been exposed must be taken into account and whether exposure has been continuous during the working period, or intermittent. Thus we cannot say because two persons have accumulated the same amount of silica in the lungs that they had, therefore, been inhaling dust of equal silica content. Nevertheless, a careful examination of the list does seem to offer some evidence on

"Civility costs nothing, and buys everything."—M. Wortley Montague.

this point. We have, for example, a stoker for 14 years who had accumulated 2.00 per cent. of silica, while a sandblaster for 15 years had only accumulated 1.03 per cent. Yet the former had a negligible fibrosis, while the latter had a very definite fibrosis of grade 2. Without knowing more precise details as to hours and conditions of work, we cannot say that because the stoker had accumulated twice the amount of silica that the sandblaster had, that the dust he has inhaled had a silica content equal to twice that of the sandblaster's dust. But, after making considerable allowances for different conditions, it does not seem to be overstretching the probabilities if we say that the stoker's dust had at least as much silica as that of the sandblaster. If that is true, then it follows that the degree of silicosis does not depend solely on the silica content of the dust inhaled.

Again, a stonemason aged 52, who had been a stonemason all his working life, *i.e.*, for 30 years or more, had accumulated only 0.76 per cent. of silica while a sandblaster for 14 years (aged 43) had accumulated 2.46 per cent., *i.e.*, about three times as much in roughly half the time. Yet in the former there was a grade 3 fibrosis, and in the latter, grade 2.

Seed and Soil.

Many similar contrasting pairs can be picked out, strengthening the view that the relationship between silica content of dust and degree of silicosis produced are not closely related. If then silicosis does not depend solely on the amount of silica in the dust nor the amount of silica in the lungs, what are the real determining factors? It seems to me that there are only two possible answers; these are :—

(*i*) that silicosis is determined not by the amount of the silica but by its nature; *i.e.*, silica in some forms is a much more potent agent for producing silicosis than in other forms.

(*ii*) that silicosis is determined not solely by the amount of silica but by certain conditions present in the body of the person into whose lungs the silica gains access.

We have here, then, to consider, as in practically all diseases, the nature of the causative agent and of the organism on which it operates. It is similar in many respects to the old question of the comparative importance of seed and soil. There is certain evidence in favour of each factor being of considerable importance, and as in so many other conditions, it is very probable that in many cases, both are in operation together. Silicosis is most likely to develop when silica in certain forms operates in persons who, for some reason or another, are more than usually susceptible to its action.

Let us consider first the question of the soil—the nature of the individual concerned.

In the first place, we cannot deny that there must be a considerable variation in the powers of resistance of different individuals all presumably healthy, to the harmful action of silica. It is true of all other disease-producing agents and there is no reason to doubt that it is true of silica. Again we should expect that in the presence of some pre-existing weakness or disease of the lungs, the harmful effect of silica would be greater and more rapid than when the lungs are originally in a sound, healthy condition. No doubt it is generally true that the diseases found commonly to be associated with silicosis are secondary to, or their production and development are favoured by, the damage produced by silicosis. But this may not be true of each individual case. It is probable that of the cases we have examined a few at any rate may have had some lung disease at the time the silica began its harmful operations. Moreover, there is the question of the occurrence of disease after silica has already gained access to the lung.

It is possible that what, in normal lungs, would be a minor and temporary affection, may be in lungs already containing silica, or the seat of an incipient silicosis, a stimulus to the development and acceleration of the silicotic process. The recent work of Kettle (E. H. Kettle, Journ. Path. and Bact., XXXVIII., 201, 1934) in which the combined effect of dust and an infective agent has been studied, is of considerable importance. The infection itself need not be a virulent one; in fact low grade infections are believed to be of special importance in this connection. Without going into the details of his work I will quote from his conclusions :—

"It is generally acknowledged that even a serious degree of pulmonary fibrosis may be caused by the inhalation of silica without any accessory factor, but this pure or simple silicosis is rarely of clinical significance; nearly always silicosis is associated with tuberculosis and my experience of silicosis in this country leads me to place increasing importance on the infective factor. I rarely see a case of simple silicosis, and even in those in which tuberculosis appeared to have been definitely grafted on to a silicotic lung, there has often seemed to me strong evidence that the apparently pneumoconiotic lesions were really infective from the beginning. These experiments support this view for they show that lesions can be rapidly produced in the lungs of guinea-pigs when a dust is combined with an infective process, whereas lesions can only be produced with the greatest difficulty, if at all, by the dust alone."

These conclusions imply that the presence of an infective process is of paramount importance in the production of silicosis, but in considering the experimental results it should be borne in mind that :—

(*a*) The test animal was the guinea-pig and not man.

(*b*) The siliceous material was administered by intratracheal injection and not by inhalation.

"We make our future by the best use of the present."

"*Great works are performed, not by strength, but by perseverance.*"

Thus neither the subject nor the method is quite the same as in naturally occurring industrial silicosis.

With regard to the relative frequency of pure or simple silicosis, and silicosis associated with tuberculosis or other infective processes, our own cases show about 50 per cent. with tuberculosis, while in about 10 per cent. not only was there no tuberculosis but no evidence of bronchiectasis, bronchitis, pneumonia, abscess or gangrene. Of this 10 per cent., death was due in certain cases to disease unrelated to the silicosis (*e.g.*, malignancy, outside the lungs). Hence, not all of these cases were affected clinically by their silicosis. Thus it is true that cases of pure silicosis, especially with clinical evidence of the condition, form only a very small portion of all cases of silicosis, but we hesitate to describe the condition as "rarely of clinical significance."

But when due allowance is made for all these considerations, it must be admitted that Kettle's work does indicate that the infective factor is an important one in the production of silicosis.

Now let us turn to a consideration of the evidence in favour of the nature of the silica as being of importance. I do not propose to deal with Dr. Jones' work, since he is here to do that for himself. Our own work, however, has some bearing on this subject. An examination of the classification of our cases that I have already referred to, shows that at the head of the table coal miners figure very prominently, whereas at the foot of the table we find sandblasters, stonemasons and asbestos workers. That is, in general, miners who have silicosis have relatively a large amount of silica in their lungs while sandblasters, stonemasons and asbestos workers have silicosis in the presence of much smaller amounts of silica. In general terms the dusts inhaled in some industries are more potent, as silicosis producers, than those of other industries; the nature of the inhaled silica has something to do with the onset and development of the disease. There is also the possibility, however, that accompanying non-siliceous dusts may exert a modifying effect on the action of the siliceous material inhaled. Some modifying effect of coal dust, for example, may account for the difference observed between the silicosis of coal miners and that of workers in industries in which coal dust is absent.

I propose now to consider some important facts to be drawn from the second classification of our cases, namely, according to occupation. Of special interest is the group of asbestos workers.

We have so far complete data of five cases; four others are awaiting analysis. One of the five cases, on account of freedom from other complications, gives data of special importance. If from the actual amount of ash and silica found in this case are subtracted the amounts found in normal lungs, the differences are approximately the same, *i.e.*, the amount of ash in these lungs in excess of what would be present in normal lungs is wholly accounted for as silica. Yet the material inhaled in this case, viz. asbestos, was not free silica, but a complex silicate containing considerable amounts of other oxides besides that of silicon. One cannot avoid the conclusion that the asbestos has undergone decomposition in the lung, with deposition of SiO_2 and removal of the metallic oxides. Herein is possibly the clue to the production of silicosis; namely that it is due to the chemical action of silica, either inhaled in a chemically reactive form, or liberated *in situ* from inhaled silicates capable of relatively easy decomposition within the lungs. It is notable that in this case there was an interval of six years between the man ceasing work and his dying of asbestosis, and apparently these six years have sufficed for the complete decomposition of the asbestos present in his lungs when he ceased work. No doubt considerable decomposition had already occurred when he gave up this work, since he had been employed in it for a total period of 11 years.

The presence of other disease resulting in the formation of pus, inflammatory exudates, caseous material, etc., all of which would add to the non-siliceous ash of the lungs, prevents our dealing with the analytical data of the other cases in the same way. Three cases in this group, however, are noteworthy in that, following relatively short exposures to risk (4 years, 6 years, 1½ years) and considerable intervals away from the employment (15 years, 15 years, 25 years) death has occurred and evidence of considerable pulmonary asbestosis been obtained. This strengthens the previous conclusion, that once asbestos is present, slow decomposition with the production of active silicosis-producing material, *i.e.*, free SiO_2 in a chemically active form is continually occurring, so that in spite of the abandonment of the occupation, the disease slowly progresses.

It is known, of course, that silica is transported to some extent from the lungs and carried to neighbouring lymph glands. It is possible that some may be excreted from the body altogether. It is very probable that silica deposited in the lungs as a result of decomposition of silicates is in a form which will allow of more rapid transportation and possibly excretion, than silica originally deposited as such in relatively large, inert particles. Hence it seems very probable that the silica actually found in these cases, in which long intervals have elapsed since exposure to asbestos, is a less correct indication of the actual amount of silica which has been in the lungs than in certain of the other industrial conditions we have mentioned.

Time does not permit me to call attention to the results in other industrial groups, *e.g.*, stonemasons, sandblasters, coalminers, etc., but there is one other case to which attention must be drawn, that is the abrasive soap worker previously mentioned, who died at the age of 26 of acute silicosis. She had worked in this occupation for three years, after which the

illness apparently incapacitated her, as she is reported to have been ill for the three years which intervened between ceasing work and death. There is no evidence of tuberculosis here.

I have at present no information as to the nature of the abrasive used, but it seems to me that the unusually severe silicosis may be connected with the simultaneous inhalation of siliceous material and soap powder, which would give an alkaline reaction. This reaction would, of course, be specially favourable to the decomposition of a silicate or the solution of free silica.

Attention has previously been drawn by Heffernan to the severe and rapid silicosis of workers handling siliceous soap powders and he too ascribes the results to the effect of the products resulting from chemical action between the siliceous material and the alkaline soap powder.

The evidence of this case, therefore, reinforces that of the case of asbestosis I have just mentioned, in indicating that conditions favouring the deposition of silica in a chemically reactive form are those which are most likely to induce rapid silicosis.

The blood plasma has a pH. of 7.3 to 7.5, *i.e.*, is very slightly alkaline and tissue fluids are similarly alkaline. Infective conditions, by promoting inflammatory reactions and therefore an increased supply of blood and tissue fluids to the affected parts, may play their part in determining the progress of silicosis by thus causing an increased supply of slightly alkaline fluid to come into contact with the siliceous material already present in the lungs.

If these views are correct, the materials which we should expect to be most potent as silicosis-producers would be the soluble alkali silicates, and it would therefore be of extreme interest to test these views by finding out experimentally what reactions would be obtained in animals which were given these silicates, either by inhalation or intratracheal injection.

SILICOSIS: THE MINERALS WHICH CAUSE IT.

By Dr. W. R. Jones, D.I.C., F.G.S., M.I.M.M., (Geological Dept., Royal School of Mines), (Imperial College of Science and Technology).

The theory that silicosis cannot be contracted by the inhalation of any dust other than that of uncombined silica in the form of quartz, chert or flint, has been based entirely on inferential, and not direct, evidence. When that theory was formed, two facts of outstanding importance were not known to its advocates. The first of these is that the finest and hence most dangerous dust produced, for example, by the rocks worked in the gold mines of South Africa (where compensation for silicosis amounted, for the year 1933 alone, to £1,200,000) and in some of the coalmines of South Wales (where 385 cases of silicosis have been diagnosed by the Medical Board in the last 2½ years) contained proportionally very considerably less free silica in the form of quartz, and more sericite fibres, than the parent rock. The reason is that the sericite fibres, unlike the quartz, are already present in the rock before it is broken, of a size sufficiently small (one twelve-thousandth of an inch and less) to enter the recesses of the lung, once they are released into the atmosphere. Moreover, they are only loosely held together in the rock, and are freed readily when the rock is broken; and due to the smallness of their size and their form, they remain suspended in the air to be inhaled by the employees. The bulk of the quartz, on the contrary, is in much larger particles and settles quickly.

The second important fact which has not been realised is that the great majority of the mineral particles found in the silicotic lungs of the South African goldminers, South Wales coalminers, and British potteryworkers, consist not of quartz, but of minute fibres of sericite of the size and form they are found in the exploited rocks and in the "clay-body" used in the manufacture of pottery.

These two facts to which I have drawn attention are not based on any theory, but on data which can easily be tested. That with reference to the great increase in the ratio of sericite to quartz in the finest dust, was proved underground in the goldmines of South Africa during my recent visit there, and confirmed by chemical analysis and also by some of the leading experts of that country. The presence of innumerable fibres of sericite in microscopic sections of silicotic lungs, and in their mineral residues, has been confirmed recently by a number of geologists in this country, including Dr. H. H. Thomas, F.R.S., Government Petrologist to H.M. Geological Survey and mineralogical expert to the Home Office.

If these facts had been known at the International Conference on Silicosis in 1930, silicosis would, at the final meeting of that Conference, not have been defined as a "pathological condition of the lung due to the inhalation of silicon dioxide," and that to produce this pathological condition "silica must reach the lungs in a chemically uncombined state." Greater attention would have been paid at that Conference to the incrimination of silicate minerals, particularly those occurring in the rocks, in the dust, and in the silicotic lungs, as minute fibres. Indeed, some of the leading medical authorities who sat on that Conference, including its Chairman, have since agreed in their contributions to the discussion on certain papers I have given on the subject, that the important rôle in the causation of silicosis played by silicate minerals, particularly sericite fibres, had until very recently, been greatly underestimated.

The reason why these two facts were not then known is because, strange as it may seem, this finest dust and the mineral residues obtained from silicotic lungs, had never been examined by a geologist in this or any other country. The chemists who

"Clever business men learn as much from their mistakes as from their successes."

Fig. 1.

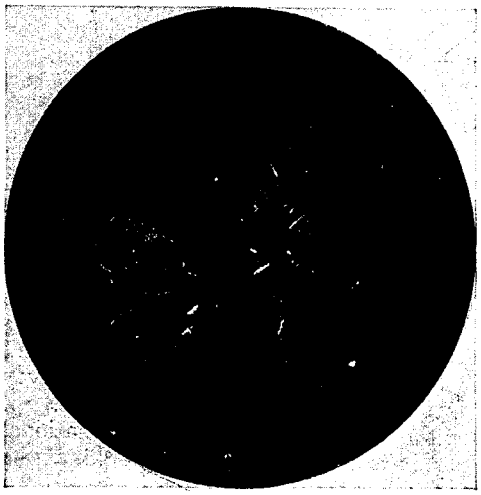

Fig. 2.

Fig. 1. Aggregates of the mineral residue from the silicotic lung of a collier. Taken under polarised light to show that the material is doubly refracting. × 30.

Fig. 2. Two of the aggregates, shown in Fig. 1, of the mineral residue from the silicotic lung of a collier. Taken under polarised light to show the form of the longest fibres of sericite. The great majority of the points of light are minute fibres just out of focus. × 500.

Fig. 3.

Fig. 4.

Fig. 3. Finely powdered quartz taken under polarised light to show the form of the particles. The quartz, before powdering, was isolated from a sandstone in the anthracite district of South Wales which had given rise to many cases of silicosis. × 70.

Fig. 4. The large dark crystal occupying the centre of the field is felspar; the small fibrous aggregates inside the felspar crystal are sericite. Taken under polarised light to show a type of sericitisation of felspar. × 50.

carefully carried out the analyses of the dust and lung material, the geologists who were asked specifically to examine the rocks for their content of free silica, and free silica only, and the medical authorities who received these results, all worked independently and the final conclusions were written by medical authorities and published in medical journals which did not meet the eyes of the chemists and geologists. The result is that English and foreign literature on silicosis contains many erroneous statements about minerals and rocks, and the nature of their dust; and far-reaching and misleading deductions have been based on such statements. I have dealt with some of these mis-statements and deductions in a recent publication, and need not now repeat them.

The Normal and Silicotic Lung.

In the short time available, I shall show you some lantern slides to illustrate some of the points I have raised and, if time permits, I shall deal briefly with preventive methods against the incidence of this disease which is now known to be, individually, the most important of all specifically industrial diseases, with wide and insidious ramifications in many industries.

"Merry goes the time when the heart is young."

"The truest wisdom is a resolute determination."—Napoleon.

Fig. 5.

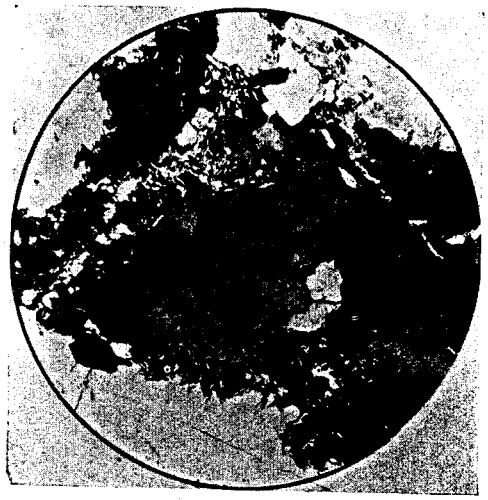

Fig. 6.

Fig. 5. Microscope section of the Transvaal "Banket" taken under polarised light to show the acicular aggregates of sericite between the quartz pebbles and grains. × 30.

Fig. 6. Microscope section of the Transvaal "Banket" (from a different part of the reef to that in Fig. 8) to show the acicular aggregates of sericite between the quartz pebbles and grains. × 30.

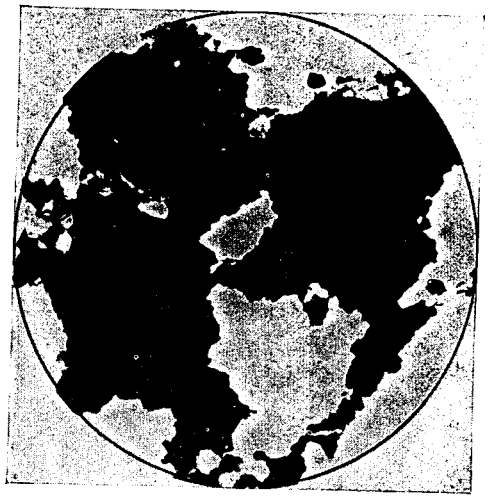

Fig. 7.

Fig 8.

Fig. 7. Microscope section of the Kolar (India) gold-bearing quartz, taken under polarised lights to show the absence of fibrous minerals between the interlocking grains of quartz. × 30.

Fig. 8. Microscope section, taken under polarised light, of a sandstone from a colliery in the anthracite district of South Wales which has given rise to dust that has caused many cases of silicosis. The clear and dark areas are quartz; numerous aggregates of sericite occur between the quartz grains. × 30.

The first five slides illustrate the progressive change in the mineral composition of the dust at different periods after blasting. The finest dust, that is the dust which enters the recesses of the lung, contains a far higher ratio of sericite fibres to quartz particles than does the coarser dust. These slides are based on the actual dust collected when I was recently in South Africa, in one of the most modern and best ventilated of the Rand goldmines.

The next few slides show the sericite fibres in sections of silicotic lungs and in the mineral residues obtained from them. The fibres are in the unhealthy tissues, and they are absent in the normal lung tissue. Quartz does not break into fibres, no matter how finely it is pulverised; this is illustrated in the next slide.

Sericite is a secondary mica formed by the alteration of other minerals, frequently by the alteration of felspar. What happens is that a crystal of felspar becomes converted, under certain circumstances, into hundreds of minute fibres of sericite. These are only loosely held together in the matrix of the rock. By breaking with a blow of the hammer a piece of sericitic rock, such as that worked in the

"Inspiration is more likely to come to a busy man than an idle one."

"The good that men do lives after them."

Fig. 9.

Fig. 10.

Fig. 9. Microscope section, taken under polarised light, of sandstone from a Scottish coalmine. The clear and dark areas are quartz; the crystal marked "F" (white, below centre) is felspar which is has not been sericitised. × 50.

Fig. 10. Microscope section, taken under polarised light, of sandstone from a Scottish coalmine distant from that referred to in Fig. 9. The clear and dark areas are quartz; the crystal marked "F" (near centre) is the felspar, microcline, showing complete absence of sericitisation. × 50.

South African goldmines, or in some of the South Wales coalmines, these fibres are freed easily and can be collected on a film of Canada balsam, or of gelatine, at some distance from the rock. This conversion of felspar to sericite is illustrated in the next slide.

In certain mines, where quartz has been worked for a long period of years without causing silicosis, fibres of sericite are absent or rare. Sandstone, for example, is very common in the working-places in the Scottish coalfields; quartz veins are worked in the Kolar Goldfields and the Lonely Mine of Southern Rhodesia, to name only two gold-mining areas out

Fig. 11

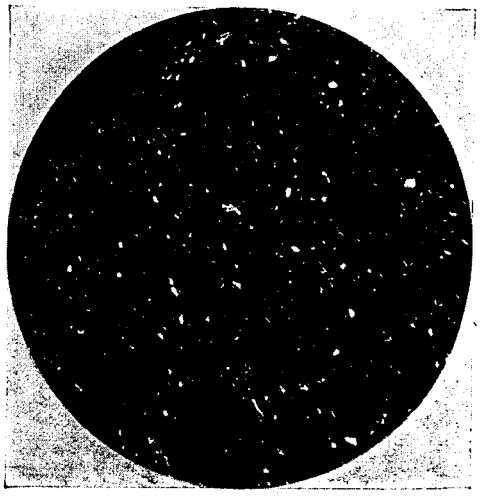

Fig. 12.

Fig. 11. Microscope section, taken under Ordinary light, of sillimanite gneiss from Broken Hill, New South Wales. Minute fibres of sillimanite are numerous. × 30.

Fig. 12. Microscope section, taken under polarised light, of a clay-body used in English potteries. Fibres of sericite are numerous. × 70.

of many. Why is it, therefore, if free silica is the only cause of silicosis, cases of this disease have been certified by the thousands in South Africa, and by the hundreds in the South Wales coalfield, whereas in the Scottish coalfields, in Kolar, and in Southern Rhodesian mines, silicosis is very rare or absent? Slides of the rocks of the Kolar goldfield and of the Scottish coalfields are shown to illustrate the absence or rarity of sericite fibres in these rocks.

I should like to make it very clear that I have never suggested that quartz, flint or chert, could not give rise to dangerous dust. What I do contend

Continued on Page 183.

"There is in every human countenance either a history or a prophecy."

Silicosis and Asbestosis—continued from Page 180.

is that silicate minerals such as sericite and other fibrous minerals (e.g., asbestos fibres in asbestosis) do give rise to a fatal pulmonary disease and have

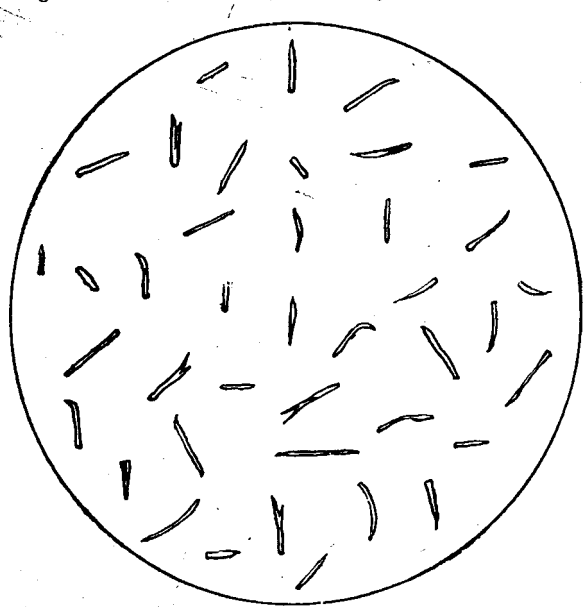

Fig. I.—Sketch to illustrate the form, as seen under 1/12in. oil immersion objective, of fibres of sericite in the various residues obtained from silicotic lungs. The longest fibres, few in number, are 5 microns in length; the great majority are under 2 microns in length.

been the chief culprits in the cases I have personally investigated.

I would ask those who still believed that dust from quartz, flint or chert was the only possible cause of silicosis, if they were content to leave unexplained the impressive fact out of a total of 432 cases of silicosis contracted in British coalmines since 1st June, 1931, no fewer than 385, that is over 89% of the total, were in the South Wales coalfield which gives employment to only 18% of the total employees in British coalmining. Were they content, in other words, to assume that it was merely a most extraordinary coincidence that 9 out of every 10 cases of silicosis in the British coalmines were in the very mine where there was the greatest development of sericite fibres; and as a still more remarkable coincidence that these fibres which were in the rock were also in the silicotic lungs of those who had worked the rock.

Important as it is to know the precise nature of the dust that causes silicosis, it is still more important to find means of preventing its incidence. I have dealt in a recent paper with the preventive measures now in operation in the South African goldmines and other mines, and have given the reasons why they have not proved successful. The full extent of the problem can be briefly put as follows:—the most dangerous dust is that in which the particles are below one twelve-thousandth of an inch, or thereabouts, in diameter; and if a man accumulates in his lungs every working-day for a period of 14 years, a little daily dose weighing less than half an inch of human hair, he will, in all probability have contracted silicosis at the end of that period. And this is not the whole story; in some fatal cases of silicosis, I found in the lung less than half the amount taken for this calculation. Can anyone suggest a method of clearing the atmosphere of these fine particles, so small that the unaided eye cannot see them (the dust which can be seen is not the most dangerous dust), so minute that the microscope reveals their presence only under fairly high magnification.

The use of water for laying the dust has failed in South Africa; and the dust-traps can only be used

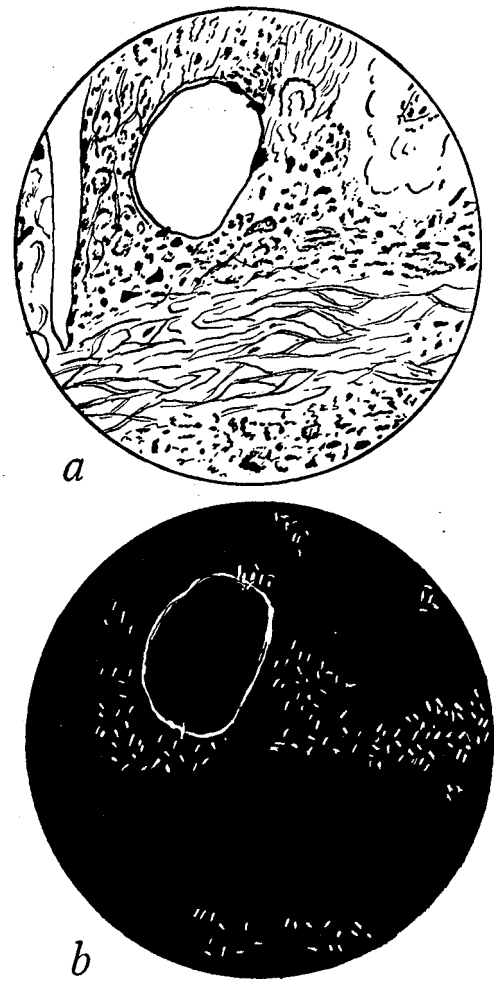

Fig. II.-(a)—Sketch of a microscopic section from near the root of a silicotic lung of a collier. To the extreme left a part of the bronchial cartilage is just in the field. (b) Sketch of section (a) as seen under polarised light, to show the distribution of sericite fibres. The large oval outline, not seen under polarised light, has been inserted as a location mark. The size of the fibres (but not their number) has been exaggerated for purposes of reproduction. The majority of the fibres are from 0.5 to 1.5 microns in length.

"He is a wise man who can interpret a woman's silence."

when drilling rock and not at the chief dust-producing sources, namely, in blasting, handling, and shovelling broken rock.

I should like to refer to the preventive method suggested by Professor J. B. Haldane, namely, the spreading of shale dust, and I do so because I consider that it is a positively dangerous method. His suggestion was based on the presumed absence of silicosis in British collieries, a presumption that has since been disproved completely by the fact that since 1st June, 1931, no fewer than 432 cases of silicosis in British collieries have been diagnosed by the Medical Board, the figures given being those quoted in the House of Commons on the 6th Feb., 1934. Even on the basis of the free-silica content of shale-dust, as compared to the dust from granite,

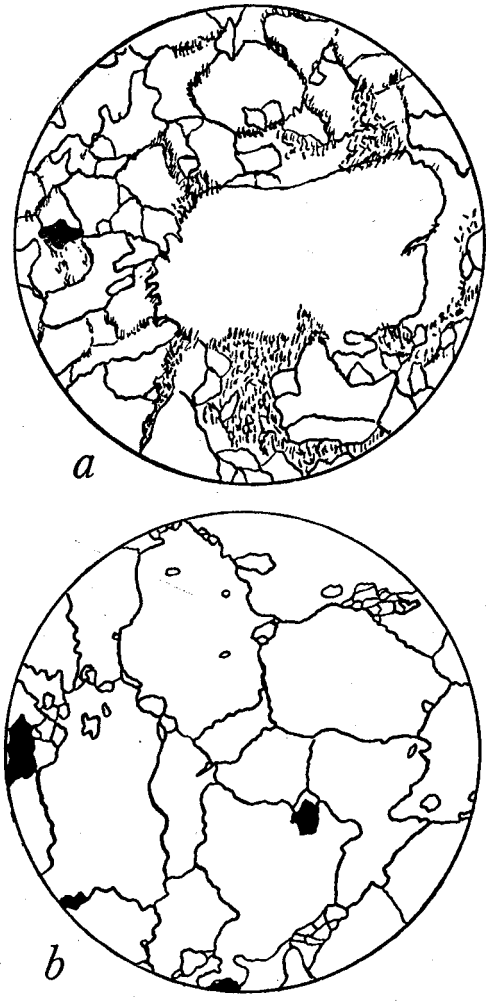

Fig. III.—(a) Sketch of the microscopic section of the Transvaal "Banket" shown in Fig. 5. The clear areas are quartz; the micular aggregates are mostly sericite; the black patch is pyrite. (b) Sketch of the microscope section of the Kolar gold bearing quartz shown in Fig. 7. The clear areas are quartz; the black patches are pyrite. No fibrous minerals occur between the inter-locking quartz grades.

which is known to give rise to silicosis-producing dust in the Cornish tin-mines, and as compared to the dangerous dust in the British potteries, Professor

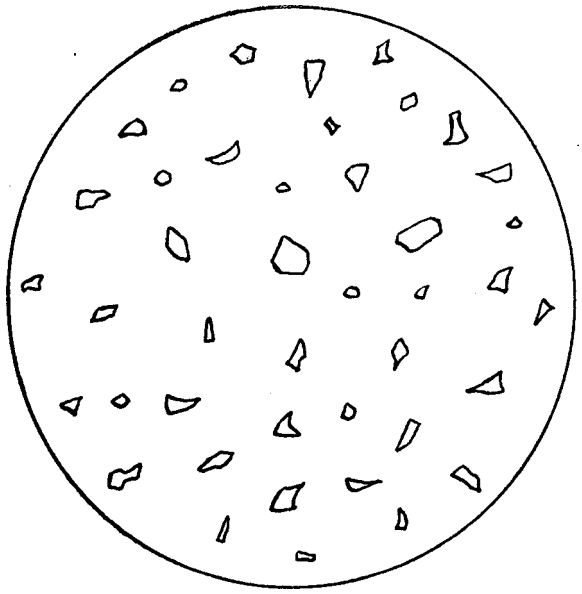

Fig. IV.—Sketch to show the form of the coarser particles of quartz seen in the various residues from Silicotic Lungs. The largest particles are from 5 to 10 microns in diameter.

Haldane's suggestion is based on incorrect mineralogical information, as I have shown in a recent publication.

In July, 1933, Professor Haldane wrote that coalminers could not contract silicosis unless they had been exposed to "dust containing 50 to 60 per cent. and more of free silica." The Cornish granites contain less than half of this amount of free silica, and their finest dust contains considerably under 20 per cent. free silica. This is true also of the dust from the clay-bodies used in the manufacture of pottery. Why, therefore, should Professor Haldane assert on circumstantial evidence based on incorrect data, that before they can contract silicosis, coal miners must inhale dust containing twice as much or more free silica than tin miners, and pottery workers? Some shales in certain coalmines, although low in their content of free silica, contain numerous fibres of sericite of the size found so abundantly in the silicotic lungs of diseased coalminers who had worked these rocks. The next slide shows the presence of these fibres in a shale, the dust of which Professor Haldane, on account of its low content of free silica, would regard as suitable for spreading to prevent silicosis.

The preventive method I suggest is to supply the employees direct with clean surface air in the case of mines; and for factories, with clean air from outside the factories. I have dealt with this method,

"The first hour of the morning is the rudder of the day."

which is far simpler and less expensive than appears at first sight, in a recent paper. The clean air is brought direct *under* a light cover over the nose and mouth only. There is no laboured inhalation, as with a respirator, and you will be interested to hear that this method is about to be tried in an asbestos factory in London. When it has been in successful operation, full details of it will be published.

Blocks to illustrate the paper on "Silicosis: the Minerals which cause it" kindly loaned by "The Quarry Manager's Journal."

SOME CHEMICAL OBSERVATIONS ON SILICOTIC AND ANTHRACOTIC LUNGS.

By Dr. F. V. Tideswell.

During the last few years I have had the opportunity, in conjunction with Mr. F. Bradshaw, of making a chemical examination of a large number of silicotic and anthracotic lungs. The main determinations made were of total silica, insoluble organic matter (coal) and insoluble mineral matter. Further, an attempt was made to estimate the proportion of "readily-soluble" silica in the lung.

Methods of analysis. The lungs, from a number of sources, were sampled, and a portion roughly dried and powdered for analysis.

Ash. Some care is needed during ashing, since the lung material tends to froth.

Silica. The determination is made in the usual way after fusing the ash with sodium carbonate.

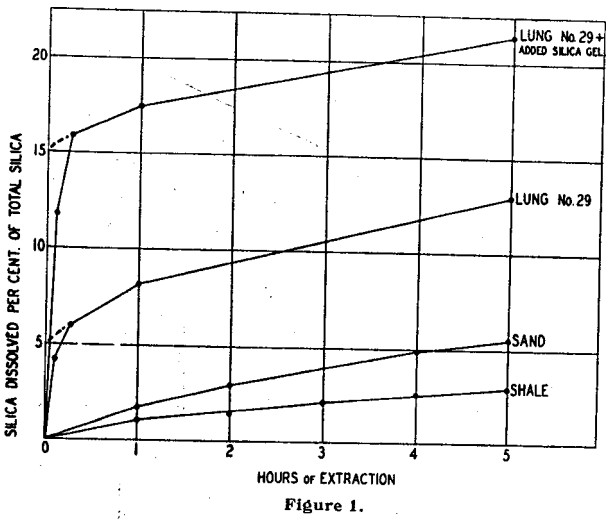

Figure 1.

Estimation of "Coal" in Lung Tissue. A variety of methods of estimating coal in lung tissue were tried, based on the greater resistance of the coal than the tissue to the attack of mild oxidants and of alkalies. The method finally standardised enabled a reasonably accurate estimation to be made of even the most readily oxidisable coal.

The "coaly residue" so determined, necessarily includes any foreign combustible matter present which is resistant to the treatment given.

Method: One gram of the powdered lung is heated with 20 ccs. of nitric acid (10 per cent. of conc. acid in water) during 20 minutes, using a glass boiling tube, fitted with a loose stopper, immersed in a bath of boiling water. Frequent shaking is needed. The mixture is filtered hot through a fine filter paper and washed with 20. ccs. of cold distilled water. The residue is washed off the paper using 40 ccs. of dilute ammonia (10 per cent. of conc. ammonia in water) and the suspension again heated in the bath during 10 minutes. (Care is necessary to avoid frothing). The suspension is cooled quickly and transferred to a 100-cc. centrifuging flask, washing out the tube with 10 cc. of water, and centrifuged at 3000 revs./min. (at least) during 30 minutes. After decanting off the liquor, the residue in the flask is twice shaken up with 20 cc. of water, centrifuged again during 30 minutes and the liquor decanted.

The residue is washed into a weighed dish, the water evaporated and the residue dried either in a vacuum oven or in an inert atmosphere during one hour at 105°C. The dish and its contents are weighed to give the amount of *"coaly residue plus associated ash."*

The dish is then transferred to a muffle and the material ashed during 30 minutes at 700 to 800° C., whence the *ash associated with coaly residue is estimated.*

The *"coaly residue"* is then estimated by difference.

(An alternative and less convenient method of estimating coal dispenses with the preliminary oxidising treatment and uses only the breakdown of the lung tissue by hot 2 per cent. potassium hydroxide solution).

I do not propose to deal in detail with the relationship of the chemical estimations to the physiological condition of the lungs. Cummins and Sladden, on behalf of whom most of the earlier analyses were made, have discussed such relationships fairly fully (Journ. Path. Bact. 1930, **33**, 1095).

The results have disclosed, broadly, that with increasing degree of contamination, the ash-contents of the lungs, expressed on the weight of dried tissue, increase from an original figure of between 2 and 5 per cent. to as much as 20 per cent., with a corresponding increase in the insoluble ash. The contents of total silica rise from < 0.1 per cent., for a normal lung, to as high as 10 per cent. The highest "coal" content observed was 27 per cent.

The ash associated with the coaly residue is derived essentially from mineral matter foreign to the lung nd resistant, as most of it is, to the treatment given. An uncontaminated lung yields a negligible proportion of insoluble ash.

"The greatest holiday haunt is the cost."

The resistant foreign mineral matter present in the lung is somewhat greater in amount than the insoluble ash recovered. By making certain assumptions, the result can be corrected to give a reasonable estimate of the amount of foreign mineral matter present.

For most of the specimens examined, the ratio of total silica to estimated foreign mineral matter present varies between 40 and 60 per cent. In conformity with this, a number of determinations made of silica in the recovered insoluble ash varied also from 40 to 60 per cent. This proportion probably corresponds to the composition of the contaminating dusts.

Whilst these chemical analyses show that the contaminating material is mainly silicate and afford no evidence of selective contamination by SiO_2, they do not, of course, demonstrate conclusively that the actively dangerous material is silicate and not free silica. Further, they afford no evidence as to whether one or other particular silicate is is especially dangerous.

Soluble forms of Silica in relation to Contaminated Lungs.

To the chemist, the most interesting question relating to a silicotic lung, is the nature of the silica present, whether (1) free, e.g., quartz ; (2) hydrated, as "sol" or "gel" ; (3) combined, as in the silicates and complex silicates ; (4) inorganic combination.

To distinguish between these several forms of silica in a material such as lung tissue, is a considerable task. We have made, with only partial success, an attempt to estimate the hydrated silica which seemed to us to be of particular interest. The colloidal theory of causation of silicosis appeals because it attempts to bridge the gap between the identification of the offending material and the recognition of the pathological condition. In the form in which this theory was expressed to me by Dr. P. Heffernan, it appeared to involve not only the hydration of silica, but also the partial retention of the hydrated silica in the lung, and I was, therefore, encouraged to seek for evidence of this.

Having once passed through the state of hydrosol, silica whether sol or gel should be readily detectable in the lung by its ready solubility in alkaline solutions. Accordingly, a method was developed of extracting silicotic lungs using an alkaline solution just sufficiently active readily and completely to remove gel silica. The extract was separated from suspended particles by repeated prolonged centrifuging and the dissolved silica estimated.

While the early results suggested that a considerable proportion of the lung silica was present as sol or gel, improvement in the technique of separation resulted in a reduced yield of dissolved silica, averaging for the score or so lungs examined rather less than 10 per cent. of the total silica present. This value is still too high, however, for it includes silica which is progressively dissolved by the alkaline solution from the particles of insoluble siliceous matter.

Fig. 1. shows curves relating the amount of silica dissolved, using the standard reagent (2 per cent. KOH) during different periods of extraction, from a silicotic lung, with and without added silica gel, and from ground shale and quartz. The rate of solution of the two latter would presumably be greater were the particles as fine as the dust particles entering the lung, since the rate of solution increases with increasing fineness.

By extrapolation back to zero period of extraction, that portion of the curve relating to the slow continued solution of silica, it can be deduced that there is present in the lung examined, approximately 5 per cent. of the total silica in a readily soluble form, i.e., presumably as once hydrated sol silica. The corresponding ratio for half a dozen other lungs was found to range from 3 to 6 per cent. (in one case, 10 per cent.).

The value for readily soluble silica so determined must be regarded as the upper limit of the true value. Whether it can be used or not to support the hypothesis of colloidal action appears to depend on, amongst other things, the extent to which elimination of dissolved silica can be postulated.

Following the reading of the three papers by Dr. Fowweather, Dr. Jones and Dr. Tideswell, **Mr. Bain** remarked that, no doubt, all had listened with intense interest to all that had been said, particularly the reference by Dr. Jones to the work he had carried out in South Africa.

We are fortunate, continued **Mr. Bain** in having the views of the geologist, chemists and medical experts. Their opinions have been extremely well expressed.

Dr. Jones's conclusions have been particularly clearly drawn and his remarks are of special interest, particularly to asbestos workers. His knowledge, relative to the African and Indian miner, is very valuable.

I should like to ask Dr. Fowweather, who expresses some interesting new ideas, whether he can give any information relative to the prevalence of silicosis or asbestosis in rats (in mines) or in dogs (in asbestos factories) and if man is more susceptible to asbestosis than animals.

During a visit to the Thetford Mines in Canada a few years ago I was surprised to see a very thick coating of dust in the various buildings and even in the offices. On making enquiries regarding the prevalence of asbestosis among the miners, I was informed that asbestosis of the lungs was unknown. I should like to ask Dr. Fowweather whether he thinks it would be possible to contract the disease in such surroundings.

Continued on page 204

Index to Advertisers

OTHER THAN REFRACTORERS

Index to Regular Monthly Features including Details of Various Associations.

Features of This Issue

"The man who says 'It can't be done' is liable to be interrupted by somebody doing it."

Silicosis and Asbestosis.

A Joint Meeting of the Refractories Association of Great Britain, with the Yorkshire Section of the Society of Chemical Industry.

The Discussion which followed the reading of the Papers. (*Concluded from our May Issue*).

DR. P. Heffernan, M.D. (Tuberculosis Officer to the Derbyshire County Council)* said: "I would like to express my thanks to Mr. Rees and Dr. Tideswell for so kindly inviting me to this meeting, to-night, and giving me the privilege of listening to such stimulating papers as those we have just heard.

It is a happy augury to see geologists, petrologists, physicists, chemists, bio-chemists and medical men, meeting to-night to discuss these questions, each contributing his share and co-operating for the advancement of knowledge. Such team-work has been long overdue.

I was particularly delighted with Dr. Fowweather's paper, which conclusively showed that the extent of the fibrosis in a silicotic lung bore no relationship to the amount of the silica actually found in the lung, because, from *a priori* reasoning, I had always believed that this would be found to be the case: that silica entered the lung, did its nefarious work, and then departed. It had been known for many years that the quartz particles which were found in the lymph nodes in early silicosis disappeared from the silicotic nodules and from the silicotic fibrosis with the passage of time. This was foreshadowed by Belt of Toronto many years ago, and was confirmed by other workers, including Professor Stewart, whose absence from the gathering to-night, everbody regrets.

What is not, perhaps, so generally recognised is that silica is eliminated daily in the urine. Normal urine contains, if I remember rightly, up to 80 milligrammes per litre. It is also eliminated by the bowel, and is capable of entering into organic combination in the bodies of men, animals and plants.

The feathers of birds, for example, depend upon their silica content for their stiffness and elasticity, as do the stems of cereals, bamboos, etc., and the silica content of some old-fashioned plants such as the equisetums, is very high. In bird's feathers, the silica is supposed to exist, according to Professor J. B. S. Haldane, as cholesterol silicate, and, in plants, as silicates of polysaccharides.

As regards Dr. Jones's paper, it is important to remember that silicosis is only one form of pneumonoconiosis, but is an unique form, and is distinguished from all other forms by its basal unit: the silicotic islet or nodule. It should not be confounded with other forms of pneumonoconiosis or fibrosis.

In Derbyshire, silicosis risk occurred in four industries, viz., the quartzite industry, the mill-stone grit industry, the chert quarries (he showed a skiagram of silicosis in a chert quarryman, and a specimen of the material) and the pocket silica sand industry. Derbyshire coal miners do not suffer from silicosis, although the coal mines are dusted with "shale."

Derbyshire quartzite differs in no material particular from Oughtibridge and Deepcar quartzite. and I should like to ask Dr. Jones if this material contains sericite. There is no doubt as to the deadliness of this quartzite dust in producing silicosis, Millstone grit contains 96.4% of silica, and only 1.3% of alumina and ferric oxide taken together, so that there is very little room for any combined silica in its structure. It contains a good deal of mica, but this appears to be flaky mica of the muscovite type. In fact, a Derbyshire stonemason can always tell the natural bedding of a piece of millstone grit by the disposition of the flakes of mica.

Chert is pure silica of the micro-fibrous or chalcedonic type, in which the individual fibres are so fine that they are said to be measureable only in one dimension. As regards the pocket silica sands or clays employed in firebrick making, I have always maintained that the silicosis risk in this industry is comparatively slight, but I would not be surprised to hear that the material contained more sericite than any of the other three.

I have been taken to task regarding a paper I wrote some years ago on the freedom from silicosis of workers in this industry, because I had not given dust counts, and because I had not stated what changes occurred in the firing of the bricks. Well, Dr. Middleton, who had done the dust counts, is present and could doubtless give us some information about them. He might also say whether continued experience has modified his views as to the risk in these industries.

Firing merely inverted the quartz into tridymite and cristobalite, or left the quartz unchanged. In the potteries, the persons who trimmed the ware after firing were subject to silicosis as well as those who worked with flint before the firing took place.

*On page 204 of our May issue "Medical Officer of Health for Derbyshire" follows the name of Dr. P. Heffernan. Unfortunately, we were misinformed as to Dr. Heffernan's true designation and we hasten to correct the error which occurred. It should have read "Tuberculosis Officer to the Derbyshire County Council."

"Life is not Victory, but Battle."—Roswell D. Hitchcock.

In considering whether silica or sericite caused silicosis, the pathological and experimental work of Leroy Gardner and Cummings at Saranac in the U.S.A. was of great importance. Their pathological conclusions differed in no way from those of Professor Stewart, and no more need be said about them. But their experimental work has been going on for nearly twenty years and is of wide range and great importance. As long ago as 1920, Gardner showed that he could produce typical silicosis in the lungs of guinea pigs by exposing the animals to "pure quartz dust," while similar exposure to granite dust failed to produce silicosis. I would like to ask Dr. Jones which mineral contained the more sericite,† the "pure quartz" or the granite ?

Similarly Gardner and Cummings have compared the action of quartz dust experimentally with that of asbestos dust, dusts of alundum, aloxite, emery and other non-siliceous abrasives, with carborundum dust, and coal dust, and always with the same result ; the quartz dust produced silicosis, and the others did not.

As regards the supply of fresh air to workmen by means of masks. I brought home, from Canada, in 1930, a "Bulmer Air Mask." The Bulmer mask consists of a hemisphere of light jaconet, tied loosely on the face. It was fed with air, under slight positive pressure, by a light rubber tube supported over the shoulder by light webbing harness. It was quite unirritating in use, but the drawback seemed to be in the air compressors, which fed the pipes. These had to be lubricated and the lubricating oil volatilized or became polymerised into poisonous hydrocarbons. I have passed on the mask to Dr. Fischer.

As regards the colloidal theory of the action of silica in the lungs, recent acquaintance with the work of the Braggs has somewhat modified my views. Gardner and Cummings have shown that the rapidity of action of quartz dust depended upon the smallness of the particles, and the work of Sir William Bragg on silica and of his son Professor W. Bragg on the structure of silicates goes to show that, provided the particles are small enough, there is no essential difference in the action of silica and silicates, as aerosols and as hydrosols. Both might very well act chemically, when freshly made, as well as colloidally, and the aerosol might be the more active of the two. One other thing Professor Bragg's work did show, viz : that clays, micas and talcs, were the most inert, electrochemically, of all the mineral silicates, so that if silicosis was the result of chemical action, clays, micas, and talcs would be the least likely to cause it.

† We would refer our readers to "Studies of Refractory Minerals" (No. 20) on page 228 for a description of Sericite.

Note on "Shale."

The term "shale" is, one fears, applied to materials which are often very different in chemical and physical composition. Properly so called "shale," is a compressed carbonaceous clay, occurring near the coal measures, and containing a few quartz particles, just as even the finest clays, *les argiles veritables*, do. Such shale is quite incapable of producing silicosis, and has been used for many years in "dusting" the Derbyshire coal mines.

It is well-known that Derbyshire coal miners do not suffer from silicosis. At our sanatorium at Chesterfield, many thousands of coal miners of all ages have been treated for tuberculosis of the lung, during the past twenty years, (although miners in Derbyshire do *not* suffer disproportionately from pulmonary tuberculosis). Radiographs of the chest have been taken in every case, and these radiographs are available for inspection at Chesterfield Sanatorium. *None show silicosis.*

Unfortunately, the term "shale" is sometimes applied to a formation consisting of thin beds of friable gritstone alternating with thin beds of clayey material containing many quartz particles. Such "shales," incorrectly so called, would, of course, be very dangerous. A typical example is the "Yoredale Shale" (*sic*) formation of North Derbyshire which consists mostly of layers of soft grit.

Note on the Kolar Goldfields.

I am a retired Officer (Major) of the Indian Medical Service, and from 1906 to 1915 (when I went on War Service) I worked in Madras. I am, therefore, acquainted with industrial conditions in the South of India.

I noticed, with some ironic comprehension, the statement that the Indian employees in the Kolar mines "refused to be radiographed"—I think I could suggest an explanation of that refusal.

I would advise those concerned to take any evidence, coming from Indian sources regarding the absence of silicosis amongst Indians employed in the Kolar quartz mines, with some reserve.

Dr. Chas. L. Sutherland, D.P.H. (Chief Medical Officer—Medical Board for Silicosis and Asbestosis—Sheffield) : Dr. Sutherland showed two specimens of lungs of South Wales coal miners who had died of silicosis. The first of these was aged 34 and had been employed in tin mines in Cornwall up to the age of 23, and, after that, for about 4 years as a hard ground worker in the anthracite area. The lung of this man showed a very advanced nodular type of silicosis which contrasted very remarkably with the second lung which was shown. This was from a Welsh coal miner from the anthracite district, who was aged 43, and had been employed in coal pits in that area for 23 years. For a year and a half of this,

"Gird up your loins, the cessation of your intellectual power looms still closer."

"Civility costs nothing, and buys everything."—M. Wortley Montague.

he was definitely employed as a hard heading worker, i.e., working in sandstone, and for over 21½ years, he was employed as a collier, when he might, or might not have been exposed to silica dust. This lung showed the appearance which has been described as that of silico-anthracosis. It is solid, jet black, and appears to be breaking down in the centre. The specimens illustrated the subject matter of the discussion, since the exposure included granite dust, coal dust, and silica dust.

In conclusion, Dr. Sutherland, thanked the members of The Refractories Association of Great Britain (and the Yorkshire Section of the Society of Chemical Industry) for their kindness in inviting him, through Mr. W. J. Rees, to attend the meeting that evening.

Mr. C. C. Davie (Secretary, The Refractories Industries Compensation Fund Ltd.): I understand that ganister does not contain much (if any) sericite and Dr. Jones' contention is that sericite is the harmful material which sets up silicosis. If this is so, how does he account for the fact that out of 465 cases under the Refractories Industries, 167 of them are ganister miners or getters. There are also 85 cases of crusher men and grinders, and probably it would not be an unfair estimate to attribute at least half of these cases to ganister. Of the 314 other cases, again a proportion of them are people working in ganister. It would, therefore, appear that more than half our cases come from working in material where there is very little sericite.

It seems to me that if Dr. Jones' contention is to be accepted (and a very interesting suggestion it is) that the most careful investigation will have to be made as to the presence, or otherwise, of sericite in ganister. Of course, my records do not show what particular ganister the men have been working in. I do not know enough of the subject to know whether some ganister has much sericite and some a little.

Mr. W. J. Rees, M.Sc., F.I.C. (Head of the Refractories Department of Sheffield University) (Secretary of the Refractories Association), remarked that he was particularly interested in the suggestion put forward by Dr. Jones. There seemed to be more than one cause of the fibrosis typical in the silicotic lung.

If Dr. Jones' suggestions with regard to sericite are established, it would appear likely that there are many other raw materials worked on a large scale which are quite as dangerous, from the point of view of silicosis, as silica rock. Kyanite, Sillimanite and similar classes of material are extensively used in the Refractories Industry.

"It is a subject of great interest and importance, and I should like to read you a communication I have received from Professor W. G. Fearnsides, M.A., F.R.S. (Sheffield University)."

Professor Fearnsides' Communication.

Professor W. G. Fearnsides, M.A. F.R.S. (Sheffield University): I should have come along to listen, if not to speak, at your Silicosis discussion... but needs must that I go with my University Field Class to Llangollen instead.

I have been looking at quite a lot of slices of Coal Measure Shales, binds and sandstones collected locally and am clear that one would be unwise to certify any one of them free from the kind of secondary mica which has been called sericite, but how much of that mineral is "platy" or bladed in habit and how much is "fibrous" and, therefore, according to Dr. W. R. Jones, particularly dangerous, I am not prepared to say. There is abundant sericite in all our specimens of Loxley Edge Rock, Middle Rock, Crawshaw Edge Flags and Rough Rock, and in Bastard Ganister, but much less in the good Halifax Hard Bed, Deepcar and Oughtibridge Ganister, and, so far as I can see none survives in the slices I have of burnt silica bricks.

Dr. E. L. Middleton of the Home Office, having been invited by the Chairman to speak, remarked that one or two points had been raised in the papers on which he would make a brief comment.

We are indebted, he said, to Dr. Jones for bringing to bear on this important problem of silicosis, the science of geology; this had been done previously in South Africa to a limited extent, as well as in other countries.

Medical men who have studied the disease realise the diversity of its nature and the difficulty there may be in defining exactly the points at which it merges into other pulmonary diseases. We have realised that the type of silicosis found amongst sandblasters and flint crushers, for example, differs from the type of the disease met with amongst potters and the coal miners of South Wales. It appears to be not inconsistent with the theory put forward by Dr. Jones to maintain that free silica has a fundamental action on the lung tissue and that other substances have a modifying effect on that action. It is conceivable that two kinds of dust may act separately in the same lung at one and the same time.

Dr. Heffernan has referred to the occurrence of silicosis amongst the workers in the pocket clays. Atmospheric dust samples showed that when dried and subjected to mechanical treatment in the processes of manufacture, enough fine dust was given off to account for the occurrence of silicosis, though the period of time the workers had been engaged in the process has to be considered.

Mr. Rees' remarks and the observations of Professor Fearnsides are of particular interest and extremely important, and I hope occasion will be found to publish them.

The subject of silicosis has been one of vital importance to Sheffield, which may be regarded as the cradle of silicosis in this country. In the middle of last century the then Medical Officer of Health referred to the mortality amongst steel fork grinders, who rarely reached the age of 40.

"Great works are performed, not by strength, but by perseverance."

I feel that there is still much information to be gathered together on this subject and I would particularly stress the importance of keeping an open mind until we are in a position to review all the facts.

Dr. C. G. Addingley (British Belting and Asbestos Ltd., Scandinavia Works, Cleckheaton, Yorks.), remarked that the different results obtained by Dr. Fowweather, who found that the ash of the lung was entirely silica, and by Dr. Tideswell, who found that the silica constituted about 50% of the ash, may, perhaps, be explained by the difference in the nature of the dust inhaled. In Dr. Fowweather's case, it was asbestos. Ordinary white asbestos is essentially hydrated magnesium silicate; if this decomposes, the silica will remain largely insoluble, but the magnesium hydroxide, or any other magnesium compound likely to be formed, will be readily soluble and would presumably disappear fairly rapidly from the lung. Hence, on analysis, only silica would be found.

On the other hand, in the cases examined by Dr. Tideswell, the inhaled dust contained principally, compounds of silica with alumina. Any decomposition taking place would leave both the silica and the aluminium hydroxide insoluble. Hence, on analysis, the percentage silica found would be similar to that in the dust inhaled.

In the decomposition of asbestos, a result frequently obtained is the dissolution of all or almost all the metallic oxides, leaving a residue of fairly pure silica. This silica retains the characteristic fibrous structure of asbestos, an appearance which may lead to the erroneous conclusion that the asbestos is unattacked. It is possible, therefore, in the examination of lung residues, that what appears to be unchanged asbestos, may partly, or entirely be silica in fibrous form. This, at any rate, would appear to have occurred in the case described by Dr. Fowweather.

Dr. Addingley asked Dr. Fowweather what type of silica or silicate was used by Prof. Kettle in the experiments on inter-tracheal injection of guinea-pigs, and if Dr. Tideswell could say whether there was any correlation between the degree of fibrosis of the lung and the amount of colloidal (readily soluble) silica in the lungs he examined? Such a correlation, would be valuable evidence on the chemical theory of the cause of silicosis.

In a recent lecture (J. Davidson Pratt and G. S. W. Marlow, "Legal Pitfalls of the Chemical Engineer," Joint Meeting of the S.C.I., and the Institution of Chemical Engineers, London, January 8th, 1934) mention was made of a new, and fairly comfortable, type of gas mask or respirator which was being tested. Was this the same as that mentioned by Dr. Jones?

Mr. E. W. Oakes (Yorkshire Amalgamated Products Ltd.): Relative to the question of water spraying in the shops and, in view of Dr. Jones' remarks regarding respirators, I should like to ask him whether he thinks, that if fans were introduced in the upper part of the workshop and the air changed ten times quicker, the risk of silicosis in such surroundings would be ten times less?

Mr. Frank S. Russell, F.G.S. (Past President of The Refractories Association and The National Association of Clayworks Managers) (Chairman, General Refractories Ltd.), said: May I take this opportunity, on behalf of the industrialists operating in silica for the production of refractories, of saying how deeply grateful we all are to those scientific men who have given such deep study to this horrible disease, and how anxious we all are to find a means of effectively reducing its incidence amongst our workers. I do feel that considerable credit is due to Dr. Jones and his colleagues for the great work they have been, and are, engaged upon.

Dr. Jones seems to be a pioneer in a new direction, and I do sincerely hope that he may be able to continue his valuable research work on the same lines, to ultimate success.

Further, we are still more grateful for the suggestions as to the way in which we can reduce the liability of our workmen to the disease.

Dr. Jones has suggested that we might supply employees direct with clean surface air, in the case of mines; and for factories, with clean air from outside the factories, say, from a supply tank.

I am just wondering how the spent air was exhaled.

No doubt Dr. Jones will be able to enlighten us on that point.

In conclusion, may I just repeat how exceedingly grateful we all are for the privilege of hearing this matter so carefully examined and discussed.

Dr. Fowweather in replying to the various questions raised, said that he did not know whether cases of silicosis or asbestosis had ever been found in rats or in dogs associated with mines or asbestos factories.

With regard to the statement made to Mr. Bain, that asbestosis did not occur amongst the workers in the asbestos mines in Canada, Dr. Fowweather felt that, without definite knowledge as to the precise conditions obtaining in the mining side of the industry, as compared with the manufacturing side, and without knowing anything as to the conditions relating to examination and certification of illness arising amongst workers in the mines, the question could not usefully be discussed.

Referring to Dr. Middleton's comments on the case of asbestosis, in which the whole of the excess ash could be accounted for as silica (SiO_2), Dr. Fowweather agreed that it was usual to find a number of fibres in lungs which were the seat of asbestosis, and thought that his own case was rare, in the completeness with which the asbestos had been decomposed; one would expect that the more recently inhaled fibres would be relatively unchanged, though

earlier ones might be much altered. Nevertheless, the finding of some unchanged asbestos fibres was not proof that decomposition did not occur, and he, personally, could find no other explanation of the figures in this case than the one he had already offered. The so-called "asbestosis bodies" found in cases of asbestosis, were spindle-shaped bodies, in which one often found remnants of a fibre, running through a larger mass of hyaline material. It was not known what the hyaline material consists of, but he thought that it might prove to be silica gel, resulting from decomposition of the asbestos. Moreover, Dr. Addingley had informed us that the asbestos fibre could lose its metallic oxides and still retain its fibrous structure; this also showed that the persistence of fibres did not show that decomposition did not take place.

In Professor Kettle's experiments, Kaolin was the material used for injection.

Appreciation was also expressed by another gentleman present at the meeting who expressed the hope that it would be possible to obtain a verbatim report of the papers (and the discussion) given that evening.

Dr. W. R. Jones, in replying to the discussion, stated that in the type of microscope used by geologists for the examination of minerals, the light was polarised and the optical system arranged specially to bring into prominence the presence of doubly-refracting minerals like quartz and sericite, whilst at the same time rendering the rest of the microscope field quite dark. By this arrangement, with the nicol prisms in crossed position, quartz and sericite, for example, were seen clearly in varying degrees of brightness against the dark background. Biological microscopes, on the other hand, of the type used by pathologists, were not equipped with a polariser and analyser, and were, therefore, quite unsuitable for detecting the presence of such doubly-refracting minerals in lung sections.

It was true, as Dr. Tideswell had stated, that the fibrous form and the higher virefringence of sericite, helped to bring that mineral clearer to the view than the particles of quartz. The fact, however, that they were brought into prominence, and were so numerous, was indisputable proof of their presence in silicotic lungs. Moreover, against the dark background, with a good substage focussing condenser, high magnification, a suitable source of light, using a "sensitive tint" plate, particles of quartz of one micron in diameter (one twenty-five thousandth of an inch) could be brought to view. The findings in this way, under the petrological microscope had received complete confirmation from the chemical analyses of the mineral residues obtained from these silicotic lungs.

Dr. Heffernan had referred to the fact that Dr. Leroy Gardner had found that the experimental exposure of animals to the fine dust of granite, had failed to produce silicotic effects in the lung, whereas exposure to quartz dust had done so. The speaker had the greatest admiration for the extremely valuable experimental work of Dr. Gardner, with whose publications he was familiar, but would point out that the result of this experiment did not alter the fact that granite dust did produce silicosis in the human lung, as evidenced by the notable incidence of the disease among the tin-miners of Cornwall and the granite workers of Aberdeen and certain other places. That very evening, Dr. Sutherland had shown them the lung of a Cornish tin-miner and had described it as being a very advanced nodular type of silicosis. If experimental proof were necessary in support of the speaker's conclusion that minerals other than those composed of free silica could produce dangerous dust, he would draw attention to the recent work of the well-known pathologist, Professor E. H. Kettle of St. Bartholomew's Hospital, who, to quote his own words, "had had greater success in certain types of experiments with kaolin in producing experimental lesions than he had had with pure quartz or other forms of free silica." The important significance of this effect in the lung, of kaolin, lies in the fact that it is composed not of free silica, *but of silicate minerals*; that it contains numerous fibres of sericite, and that the amount of free silica in this kaolin was less than 3 per cent.

He would suggest to pathologists that in their future experiment with mineral dust, it would be helpful to them to obtain precise information from a geologist about the mineral composition of the parent rock, and of its dust. The dust of one granite could be very different from that of other granites; there were many types of granites, and these varied greatly in their mineral composition. For example, the granites worked in the tin-mines of Cornwall were characterised by the sericitization of their felspars, hundreds of minute fibres of sericite had been formed by the alteration of even one crystal of felspar. Granites from certain other districts contained no sericite; the felspar in them was unchanged. There was a further important point which required stressing in view of experimental work on dust inhalation. The dust produced by powdering a sericitic rock in a percussion mortar was of the same composition as the rock itself, *whereas the finest dust released into the atmosphere*, when that rock was drilled, blasted, or otherwise broken, contained a far higher relative percentage of minerals like sericite, which were already present in the rock, before it was broken, as minute fibres loosely held together in the matrix and readily freed into the air when the rock was broken. This had been proved by examination of mine dust under the petrological microscope, and confirmed by chemical analysis. This sorting of the dust by the suspension of the fine particles in the atmosphere, and the settling of the coarser quartz particles, is prevented when a rock

"Clever business men learn as much from their mistakes as from their successes."

is powdered in a percussion mortar; and it was his considered opinion that the anomalous results obtained experimentally, were in many cases due to the fact that the dust from powdered rock, administered to animals, was physically and mineralogically so unlike the fine dust inhaled by those employed in exploiting that rock, that certain far-reaching deductions from such experiments were misleading.

(At this stage of the discussion, the Chairman, **Mr. Bain** and certain members of the Yorkshire Section of the Society of Chemical Industry, had to leave to catch their train for Leeds. **Mr. Alex Lomas** (representing Mr. Wm. Lomas, J.P., President of The Refractories Association), thereupon proposed a vote of thanks to the Chairman for the able way he had conducted the meeting that evening, and to the various speakers, which was seconded by **Mr. Frank S. Russell, F.G.S.**).

Two or three questions had been put to him that evening, continued Dr. Jones, about the incidence of silicosis among the ganister workers in the neighbourhood of Sheffield and elsewhere. He had not so far been supplied with the silicotic lung of a ganister worker, and would avoid theorizing as to the nature of the dust such a lung would contain; he would await the opportunity of investigating this question. He had now, however, been underground in one of the Oughtibridge Silica Firebrick Company's ganister mines, through the courtesy of the management, and had taken samples of the rocks. The ganister bed in that mine averaged approximately 18 inches in thickness. All the rock-drilling, however, was confined to the sandstone underlying the ganister. Mr. Taylor, the Mine Manager, informed him that they never drilled in the ganister bed itself. Microscope sections of this sandstone showed the presence of numerous fibres of sericite between the quartz grains.

Regarding the question of exhalation of air with the breathing apparatus to which he referred, Dr. Jones remarked that there would be no difficulty in this direction, as all the exhaled air would pass through a little valve, which was very easy to operate. No oil is in contact with the air at all.

In reply to Dr. Addingley's questions, Dr. Jones remarked that the respirator described by J. Davidson Pratt and G. S. W. Marlow was not the same as that he referred to.

Time did not permit him to deal with all the points raised that evening, but after he had read the printed discussion he would gladly answer in writing any unanswered questions addressed to him.

He wished, in conclusion, to add that he had been greatly impressed by the valuable papers given that evening by Dr. Fowweather and Dr. Tideswell. He gathered that their important work lent support to some, at least, of the speaker's conclusions. He would await their publication with great interest. He thanked the Council for their invitation to address them, and said the pleasure of the visit to Sheffield was greatly increased by the fact that it was Dr. Sutherland of Sheffield, who supplied him with the first batch of silicotic lungs for his investigations.

Dr. Tideswell, replying, stated that the investigation of soluble silica in the lung specimens was made with the possibility in mind that some relationship might be disclosed between the proportion of readily soluble silica in the lung and the extent of the silicotic condition. The specimens on which the most recent and most reliable estimations had been made were too few in number and too similar, being chosen for their high silica content, to justify an expression of opinion on this point.

With regard to the nature of the contaminating material, he would ask Dr. Jones whether examination of lungs and lung residues under the petrological microscope would not tend to make unduly prominent the sericitic and other fibrous material as compared with other siliceous material which might be present in greater amount.

On the question of prevention of dust diseases, whilst the ideal solution lay in preventing the dust at source, Dr. Tideswell thought that something might be done to minimise the continual raising of dust into the air. Dust in the air of coal mine roadways, apart from its possible danger, formed a very real nuisance. Recent work carried out in the Safety in Mines Research Board laboratories* had suggested that the use of aqueous solutions of special wetting agents now available might solve the difficult problem of wetting fine stone and even coal dusts, and so of laying the dust, for example, that on the floor of a mine roadway.

In conclusion **Mr. W. J. Rees** expressed the gratitude of all who were present to those gentlemen who had given the papers that evening and to those who had contributed to the discussion which had proved to be both interesting and valuable.

"It is important" he added "that the chemists and the geologists should co-operate with the medical man in regard to this dangerous and difficult disease."

*"The Laying of Dust on Mine Roadways." F. V. Tideswell and R. V. Wheeler. Read before Mid.Inst.Min.Eng., March 2nd, 1934.

"Merry goes the time when the heart is young."