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SILICOSIS

RECORDS OF THE INTERNATIONAL CONFERENCE
HELD AT JOHANNESBURG
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GENEVA
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INTRODUCTION

The International Labour Office has never under-estimated the importance of the silicosis problem, in which it has manifested a lively interest since its inception. It has in fact carefully followed scientific research effected, as well as the reports of practical men in the different countries, and it has made every effort to assemble and collect the extremely scattered scientific data relative to the subject.

Gradually, thanks to national and international meetings of doctors or experts in industrial pathology, and thanks especially to results obtained in those countries in which silicosis is legally compensated as an occupational disease, it has been rendered possible for the Office to tabulate completely the data obtained, and to draw up a programme of activity.

Simultaneously the workers' organisations affected have inscribed the silicosis problem on the agenda of their meetings. Notably is this true of the national stoneworkers' associations and in particular their International Federation which, since 1921, has at each of its meetings voted resolutions demanding that silicosis should be considered as an occupational disease and compensated as such. Other workers' organisations such as those of the miners, pottery workers, etc., have also adopted motions in support of this demand. In 1925 the international secretariat of the stoneworkers' organisation placed before the International Labour Office a request for inclusion of respiratory diseases amongst those entitling the worker to compensation.

On reception of this request the Hygiene Service of the Office in January 1926 addressed to a certain number of experts a questionnaire accompanied by a note explaining the object of the enquiry. The replies obtained were embodied in a report submitted to the Correspondence Committee on Industrial Hygiene during its meeting at Düsseldorf in 1928. The experts assembled by the Office engaged in a lengthy discussion on the problem of silicosis and adopted a resolution in which they requested the Office to delay inscription of silicosis in the list of diseases to be compensated

since, in their opinion, the problem in question was one which presented special complication and difficulty. Two points especially were said to call for further research, that is to say, precise diagnosis of the disease, and determination of the indispensable elements for establishing the degree of capacity of the worker to continue his work. The Committee asked the Office to continue the enquiry undertaken and to submit to it at a future session the data assembled.

Meanwhile Mr. Butler, Deputy-Director of the Office, during his journey in South Africa, discussed with those interested in the question the suggestion made by Dr. Orenstein, of calling an International Conference on Silicosis in Johannesburg.

Thanks to the generous aid of the Chamber of Mines it was possible to carry out this scheme, and the first proposal as to the holding of such a Conference was made to the Governing Body in June 1928. The Governing Body then agreed that a Conference of experts should be convened to study the medical aspects of silicosis and suggested that a Conference held at Johannesburg would afford an opportunity for experts from other countries to examine the remarkable work done by the Miners' Phthisis Bureau on the Rand. The Governing Body also expressed the hope that such a Conference might bring about closer international co-operation in the study of the disease and that it would endeavour to organise a programme of research on the subject.

In March 1929 the Governing Body approved a provisional agenda for the Conference as follows:

- (a) Medical aspects of silicosis (pathological and clinical);
- (b) Preventive measures;
- (c) Compensation.

Scientific investigations on the subject engaged in chiefly in German and in English speaking countries, the work effected by Committees appointed by the British Government in recent years, the results of the discussion of the report submitted to the International Congress of Occupational Diseases at Lyons, 1929—a discussion which dealt chiefly with the research effected by the Medical Research Bureau and the Miners' Phthisis Bureau in South Africa—as well as research engaged in in Germany, all these contributed naturally to paving the way for the calling of a Conference exclusively concerned with the problem of silicosis.

This Conference, in accordance with the decision of the Governing Body, met in Johannesburg from 13 to 27 August 1930.

In view of the length and cost of the journey to South Africa the funds available only allowed of the appointment of a limited number of experts. In the course of consultation with the Governments it, however, became apparent that certain Governments were willing to pay the expenses of delegates chosen by the Office in agreement with them, and it was therefore possible to issue an additional number of invitations and to increase both the size and the authority of the Conference. The Office is extremely grateful to the delegates who accepted its invitation, to the Governments which bore the expenses of certain delegates, and to the British Medical Research Council which undertook to defray the expenses of Professor Kettle.

The Office records with interest and appreciation that the United States, in accordance with its policy of participation in the activities of the International Labour Organisation, was represented by two delegates, the expenses of one of them being borne by the United States Government.

In order that the Conference might secure the fullest possible information on the work done in South Africa, there were also appointed a number of South African observers, who were able to be present at the meetings and to assist in the elucidation of questions in regard to which they were specially competent.

As soon as the delegates had been appointed, each was asked to write a paper either on the problem of silicosis or pneumonococcosis in his particular country or on that particular scientific aspect of the question with which he was specially competent to deal.

Certain firms at Johannesburg also kindly assisted the Office by the loan of microscopes, an epidiascope, and screens for the showing of radiographs, required for the exhibition of the slides and other specimens brought by the different experts to illustrate certain phases of their work.

The firm of Kodak, South Africa, presented the members of the Conference with an album containing photographs of anatomical-histological preparations and radiographs prepared by the services of the Miners' Phthisis Bureau and of the Medical Research Bureau. The delegates were also given opportunities of visiting these two institutions, as well as a gold mine and the sanatorium for fibrotic patients, and of studying the system of medical examination in force for native workers before they are passed for service in the mines.

Mr. Phelan, Chief of the Diplomatic Division, was appointed by the Office to be in general charge of the organisation of the Conference; *Dr. Carozzi*, Chief of the Industrial Hygiene Section, as technical expert of the Office; and *Messrs. Weaver* and *Little* and *Miss Macrae* to assist in the secretarial work. The material work of the secretariat was performed by members of the staff of the Chamber of Mines who were generously placed at the disposal of the Conference by the Chamber and who worked under the direction of the secretarial staff of the Office.

LIST OF MEMBERS

Australia

Dr. CHARLES BADHAM, Medical Officer of Industrial Hygiene, New South Wales Department of Public Health.

Dr. W. E. GEORGE ¹, Medical Officer-in-Charge, Bureau of Medical Inspection, Broken Hill.

Dr. KEITH R. MOORE ², Director of the Division of Industrial Hygiene, Commonwealth Department of Health, Member of the Committee on Industrial Hygiene of the International Labour Office.

Canada

Dr. GRANT CUNNINGHAM, Director of the Division of Industrial Hygiene, Department of Health, Ontario.

Germany

Professor Dr. BÖHME, Director, Augusta Hospital, Bochum.

Professor Dr. KOELSCH, State Industrial Medical Officer, Munich.

Great Britain

Dr. S. W. FISHER ³, Medical Inspector of Mines.

Professor ARTHUR J. HALL, Professor of Medicine at Sheffield University, Chairman of the Medical Research Council Committee to investigate pulmonary diseases from silica and other dusts.

Professor E. H. KETTLE, Professor of Pathology, St. Bartholomew's Hospital Medical School, University of London.

Dr. E. L. MIDDLETON ³, Medical Inspector of Factories.

Italy

Professor Dr. GIOVANNI LORIGA, Chief Medical Inspector of Factories, Member of the Committee on Industrial Hygiene of the International Labour Office.

Netherlands

Dr. W. R. H. KRANENBURG, Medical Adviser to the Labour Inspectorate, Member of the Committee on Industrial Hygiene of the International Labour Office.

¹ Representing New South Wales Government.

² Representing Federal Government.

³ Representing British Government.

Union of South Africa

- Mr. A. B. DU TOIT, Chairman, Miners' Phthisis Board.
 Dr. A. I. GIRDWOOD, Chief Medical Officer, Witwatersrand Native Labour Association, Ltd.
 Dr. L. G. IRVINE, Chairman, Miners' Phthisis Medical Bureau.
 Sir SPENCER LISTER, Director, South African Institute for Medical Research.
 Dr. A. MAVROGORDATO, Fellow in Industrial Hygiene, South African Institute for Medical Research.
 Dr. A. M. MOLL, Chairman of the Miners' Phthisis Medical Appeal Board.
 Dr. A. J. ORENSTEIN, Superintendent of Sanitation, Rand Mines, Ltd.
 Dr. Hans PIROW, Government Mining Engineer.
 Mr. F. G. A. ROBERTS, Technical Adviser, Transvaal Chamber of Mines.
 Dr. W. STEWART.

United States of America

- Dr. L. V. GARDNER, Trudeau Sanatorium.
 Dr. ALBERT E. RUSSELL¹, United States Public Health Service.
-

LIST OF OBSERVERS

- Dr. PETER ALLAN, Medical Superintendent, Nelspoort Sanatorium, Cape Province.
 Mr. G. E. BARRY, Legal Adviser, Transvaal Chamber of Mines.
 Mr. JAMES BOYD, Secretary, Anglo-American Corporation of South Africa.
 Mr. J. BUIST, Senior Dust Inspector, Transvaal Chamber of Mines.
 Dr. E. H. CLUVER, Department of Public Health, Pretoria.
 Mr. NORMAN P. DALE, Secretary, Miners' Phthisis Board.
 Mr. MALCOLM FERGUSON, Chief Inspector of Mines.
 Mr. D. SPENCE FRASER, Actuary to the Miners' Phthisis Board.
 Mr. W. GEMMILL, General Manager, Transvaal Chamber of Mines.
 Mr. G. R. HEYWOOD, Manager, Rose Deep, Ltd.
 Mr. H. R. HILL, Consulting Engineer, Union Corporation, Ltd.
 Mr. A. F. McEWEN, Chief Chemist, Transvaal Chamber of Mines.
 Dr. A. J. MILNE, Medical Officer of Health, Johannesburg.
 Dr. ALEXANDER MITCHELL, Secretary of Public Health, Department of Public Health, Pretoria.
 Mr. B. G. ORPEN, New Consolidated Gold Fields of South Africa, Ltd.

¹ Representing United States Government.

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- Dr. A. D. PRINGLE, Medical Superintendent, Transvaal Miners' Phthisis Sanatorium.
- Mr. C. S. RAATH, Member, Miners' Phthisis Board.
- Mr. J. P. REES, Dust and Ventilation Officer, Transvaal Chamber of Mines.
- Mr. F. G. A. ROBERTS, Technical Adviser, Transvaal Chamber of Mines.
- Mr. WALTER SCOTT, Assistant Consulting Engineer, Rand Mines, Ltd.
- Dr. F. W. SIMSON, Pathologist, South African Institute for Medical Research.
- Dr. J. M. SMITH, Miners' Phthisis Medical Appeal Board.
- Dr. A. SUTHERLAND STRACHAN, Pathologist, South African Institute for Medical Research.
- Dr. R. M. TRUTER, Miners' Phthisis Medical Appeal Board.
- Professor G. A. WATERMEYER, Professor of Mining, University of Witwatersrand.
- Dr. ANDREW H. WATT, Medical Officer, Rand Mutual Assurance Company, Ltd.
- Mr. C. J. WILLIAMS, Deputy Chairman, Miners' Phthisis Board.
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REPORT OF THE PROCEEDINGS

OPENING SITTING

Wednesday, 13 August 1930, 10 a.m.

Chairmen: Sir WILLIAM DALRYMPLE and Mr. E. J. PHELAN

The opening sitting of the International Silicosis Conference was held in the Selborne Hall, *Mr. E. J. Phelan*, Chief of the Diplomatic Division of the International Labour Office, was introduced by *Sir William Dalrymple*.

Mr. E. J. Phelan: My principal function this morning is to ask the Minister, Mr. Sampson, to open formally this Conference; but it might be interesting to you (and it would certainly be fair to him) if I first indicated briefly why the International Labour Office of the League of Nations in Geneva convened this Conference and what we expect it to do.

The International Labour Office is the executive secretariat of the International Labour Organisation. The International Labour Organisation is a society of fifty-five States. It is a purely official body which is the result of fifty-five States in the world having entered into a contract to collaborate for the improvement of conditions of labour. It began as you probably know at the same time as the League of Nations, and its Constitution is to be found in the Treaties of Peace. One of its original Members is South Africa. The Organisation has to have a secretariat—an executive to carry out its decisions; that executive is the International Labour Office, which I have the honour to represent here to-day.

Now, the International Labour Office is, as I have said, an executive body. It does not take decisions; decisions are taken by the International Labour Conference, which meets once a year and to which every member of the Organisation—that is to say, every one of the fifty-five Member States—sends a delegation composed of Government representatives, representatives of the workers and representatives of the employers. They compose the annual Conference in which is vested what I may call the sovereign power of the Organisation and they alone can formulate decisions, which later become international treaties. I want to make that clear, because the conference which Mr. Sampson is going to open in a few minutes is not that Conference; it is not the General Conference of the Organisation which can adopt Draft Conventions and Recommendations.

Before that General Conference actually meets to formulate decisions which Governments must take into consideration and which Governments, if they ratify, must observe, the function of the International Labour Office is to see that it shall have the fullest possible information at its disposal; therefore, long before the International Labour Conference

considers the taking of any decisions, the Office in Geneva is busy collecting, summarising and analysing information, translating laws and regulations, and attempting to make as complete a survey as possible of all the law and practice bearing on the question which, later, the International Labour Conference will discuss. This preparatory work of the Office covers the whole area of labour problems. The programme of the Organisation which is laid down in the Preamble to Part XIII of the Treaty of Peace is of the most comprehensive character.

I am not going to discuss that programme this morning; but it does contain, as you would naturally expect it to contain, a very specific reference to industrial disease; and therefore, among the studies which the Office is constantly carrying on with the ultimate object of some day or other going to the International Labour Conference for discussion and a decision, is the question of industrial disease. One section of the organisation of the Office is specially devoted to that work, and as its head we were fortunate enough to secure ten years ago a very distinguished European authority, Dr. Carozzi, who is with me here in Johannesburg to-day as the technical expert of the International Labour Office. The very important work which the Office has already done in the field of the study of industrial disease is due above all to Dr. Carozzi's vision, energy and intelligence. He has, in the course of ten years, built up a unique organisation; he has created a network of experts and technicians scattered all over the world, with whom he is in constant contact by correspondence; and, since science need not regard the political differences which sometimes separate political units, that network of medical correspondence covers, not only the members of the International Labour Organisation, but certain very important units which lie outside it, like the United States of America and Russia. Dr. Carozzi's work takes a material form in the publication every three or four months of a bibliography of everything he is able to discover which has been published on the subject of industrial hygiene. He is able to supply the Government services of the different countries and experts in industrial hygiene with some two or three thousand references a year, so that each of them, in dealing with his own work, can keep abreast of the work which is being done elsewhere. He has also been able to undertake the immense task of compiling an *Encyclopaedia of Occupation and Health*, the first volume of which will appear in a few months. To give you some idea of the field covered and the amount of work he has performed, it will be a volume of something like 2,200 pages.

You have now, I hope, some idea of the organisation which lies at the origin of this Conference—the International Labour Office, with its general responsibility for securing better conditions of labour, and the Industrial Hygiene Section with its particular interest in industrial disease.

But the Office does not by any means work only on paper. We have had the great advantage of having had at the head of the Office a great international figure, Mr. Albert Thomas, a man with outstanding qualities of leadership and a unique capacity for achievement. He realised from the beginning the danger of the Office becoming a mere Academy and the necessity that the officials of the Office, whenever the occasion might arise, should get away from purely paper work and gain as much direct experience of the problems with which we are dealing as possible. It was in pursuance of this policy that three years ago the Deputy-Director of the International Labour Office, Mr. Butler, undertook a visit to South Africa in order to become familiar with the labour problems

of South Africa, and with the object of intensifying relations between South Africa and the International Labour Organisation. During his visit here in Johannesburg, in conversation with Mr. Gemmill and other representatives of the Chamber of Mines, and Dr. Orenstein, his attention was drawn to the unique collection of material which had been made by the Chamber and by the officials of the Government in connection with silicosis. He saw at once the importance of making this knowledge internationally available and he accordingly discussed with Mr. Gemmill and Dr. Orenstein the possibility of discovering some method whereby the scientists and the officials of the other countries might be able to profit by the immense experience of that disease which is centred in this city. Out of those conversations grew the present Conference: out of those conversations grew the suggestion that it might be possible to convene an International Conference of experts to study the medical aspects of silicosis and the measures for its prevention.

The proposal was laid before our Governing Body, which is the executive council of the International Labour Office, and was approved by them; but South Africa being a very long way away from Geneva, and the International Labour Office being a far from rich body, it would have been difficult perhaps to have held this Conference if it had not been for the generosity of the Chamber of Mines; and I should like to-day, on behalf of the International Labour Office, formally to thank the Chamber of Mines for the generous assistance which has made the holding of this Conference possible. I should like also to ask Mr. Sampson to convey to the Government of the Union of South Africa the appreciation of the International Labour Office of the facilities which the South African Government has given to us, and of the generous way in which, as soon as it was consulted, it offered to give every assistance to the Conference and to lay before it all the material which it has in its possession.

But before I ask Mr. Sampson formally to open the Conference, the origin of which I have described, I would like to draw attention to an aspect of it which is not purely medical. This Conference, distinguished as it is by the scientists who have come from different countries in Europe, Africa, Canada, Australia and the United States of America, represents perhaps a more remarkable body of expert opinion than has ever come together to deal with the question of an industrial disease; but, important and remarkable as it may be in that respect, it is still more important and more remarkable in another respect. We in Geneva often feel that we are a long way from the distant countries, and I have no doubt that South Africans often feel they are a long way from Geneva and that Geneva is dealing with problems which are remote from South African preoccupations and that, to put it quite frankly, the League of Nations spends the greater part of its time discussing purely European problems. That is only a half truth. It is true that the League of Nations and the International Labour Organisation do spend a great deal of time discussing problems which are of major European interest; but it is not true that these problems, or the solution of them, has not a direct interest to the more distant countries. It is unfair to make these observations as a criticism of the League of Nations or of the International Labour Organisation. The League of Nations is not an international dictator; the League of Nations is not a super-State; the League of Nations is not some Martian body which meets in Geneva and decides what is good for France, or Italy, or South Africa or Australia. The League of Nations is exactly like the South African Parliament or the British Parliament or the German Parliament. It is in no wise different from the members which compose it; and when it meets in Geneva, its

activities are dictated by the influence of its most active members. The League of Nations does precisely what a Parliament would do when a majority in the Parliament decides on a certain course of action, and therefore it is not a just criticism of the League of Nations to say that its activities are too European. But it is perhaps a regrettable fact—a fact, however, for which the less active members of the League are themselves responsible and not some super-body.

Now, what is the corrective to that? What is the way in which the League of Nations can be led to pay more attention to non-European questions and to become really in its activities a more world-wide organisation? The only way in which that can be done is for the non-European States to bring their problems to Geneva and insist, as the European States do, that their problems shall be dealt with. South Africa has now taken the lead by furnishing an example of how this can be done.

The fact that this Conference is meeting in South Africa is therefore a historic occasion. It is the first Conference convened by the League of Nations which has met outside of Europe. It is true that the first International Labour Conference met in Washington, in the United States of America; but the first International Labour Conference was not convened by the International Labour Organisation nor by the League of Nations. It was settled in the Treaties of Peace, and it has not met in Washington since, for the simple reason that the United States did not join the League of Nations or the International Labour Organisation; and with the disappearance of the United States of America, the balance between the European and the non-European sides of the League was perhaps weighted in favour of Europe, so that European problems have played a predominant part in the discussions of Geneva.

To-day, for the first time, Mr. Sampson is going to open an International Conference officially convened by the International Labour Office of the League of Nations, meeting outside Europe and having as its object the study of a problem that is not by any means wholly European in its interest. For that reason, I think that I may say that this Conference marks a historic point in the development of the League's machinery. It shows, I think, too, that the South African Government has understood the real meaning of Membership of the League. The Covenant is not a final nor an absolute guarantee of the world's peace or of industrial progress or of anything else. The League is, and can be, nothing more than its Members make it. But the difference between the League world and the pre-League world is this: that in the pre-League world every State guided its policy in its own purely selfish interest. In the League world we hope that States will more and more consider, not what they can get out of the League, but what they can put into the League for the common good of the whole; and this Conference is a gesture of that kind. South Africa has asked these experts, through the intermediary of the International Labour Office, to come to Johannesburg in order to see what contribution South Africa can make towards the solution of a grave medical problem.

I cannot speak for the experts, but my impression is that South Africa has more to give than she can hope to receive. She has had a longer experience and she has accumulated a greater collection of material going back for a longer period than perhaps any other country. But the essence of South Africa's gesture is her desire to put at the disposal of humanity as a whole the peculiar knowledge which she has to offer for the solution of a very pressing problem in the industrial world. I can assure Mr. Sampson that that gesture is appreciated in Geneva to its

fullest; and though possibly South Africa may gain little direct benefit from this Conference, her reward is that she will have contributed to the good of humanity as a whole. I have now great pleasure in asking Mr. Sampson to open formally this International Silicosis Conference. (*Applause.*)

The Hon. H. W. Sampson, M.P. (Minister of Posts and Telegraphs): The problem of silicosis has been one of considerable gravity in South Africa, and the Government of the Union has therefore special reason to welcome the assembly in Johannesburg of this International Silicosis Conference.

Our Chairman, Mr. Phelan, has dealt very minutely with the particulars of the preliminaries that led up to this Conference, and has saved me the necessity of repeating those matters. I want to thank him very much for his exposition of the reasons for which the League of Nations exists. After all, as we get further and further from the war time of the past, commercialism will sometimes ask: what do we get out of the League of Nations? It is well we have one in our midst this morning who has explained to this assembly the spirit which led up to the formation of that body and the spirit in which this work is to be carried on in the future. I am quite sure that this audience is particularly interested in that matter, and I am sure they realise the necessity of getting the world closer together in dealing with world problems apart from the problems in regard to their own special countries. He has explained very fully how you, Gentlemen, come to be here this morning, and it is my duty to extend to you all a very cordial welcome in the name of the Government of the Union of South Africa. I welcome especially those who have come long distances to attend this Conference. Their presence here is a signal proof of the great international fellowship in scientific investigation and research which overrides all national boundaries, and affords so hopeful a manifestation of that growing international spirit which the International Labour Organisation embodies.

The presence of the representatives of so many countries and the high standing of the members of the Conference affords also an indication of the widespread distribution of the menace of silicosis and of the serious light in which it is regarded by the health authorities of many countries.

Silicosis is indeed, I suppose, individually perhaps the most important of all occupational diseases and one which, as we know, is a danger to health and life in many industries besides that of metal mining. The problem of "silica risk" and how it is to be countered forms to-day one of the most important preoccupations of industrial hygiene.

To the metal miner who works in certain kinds of hard siliceous rock the disease has everywhere been a special danger, and we in South Africa fully realise that our local problem is only part of a world-wide problem. We realise that the prominence which the disease has attained in this country has been due primarily to the great extent and unique geographical concentration of the great gold-bearing reef of the Witwatersrand, and to the extraordinarily rapid development of the mining industry which is based upon it, and the magnitude which that industry has attained.

The problem has thus been with us at once larger and more sharply concentrated and the deaths and suffering caused by that disease have been more clearly apparent than might have under other circumstances been the case.

It is impossible to state with any degree of definiteness what the entire ramification of the disease here has been. Statistics (and many of them

not comparative) are only available since 1911. But the enormous burden which the industry has to bear of nearly £1,000,000 per annum, totalling, I believe, some £15,000,000 for compensation claims since 1911 and arising chiefly from an average of 21,000 to 30,000 whites employed underground during that period, will convey to your minds the suffering to the victims and loss to the industry.

It took a good many years before the gravity of the situation in respect of silicosis was fully recognised in this country; but one may, I think, fairly state that from the time that it was fully realised neither the Government of the Union nor the mining industry have relaxed their efforts to control the situation.

The Union Government definitely recognised "miners' phthisis" as an occupational disease in 1911, and since that date no fewer than nine Acts of Parliament have been passed dealing with the matter of compensation to miners affected by silicosis, or to the dependants of deceased miners. From time to time also there have been incorporated in the Mining Regulations a large and increasing number of detailed provisions aiming at the prevention of the disease.

The mining industry has closely co-operated with the Department of Mines in the investigation and trial of preventive measures, and this fact has greatly facilitated the development of a systematic preventive policy.

For many years one seemed to see no very marked amelioration of the situation, although one realises that with such a disease as silicosis it takes a considerable time before changes in occupational conditions show their full effect.

I am glad now to learn from the Chairman of the Medical Bureau that the annual number of the cases of silicosis which are arising to-day is only about one-third of the number that were arising fourteen or sixteen years ago, and that a substantial improvement in the situation has occurred within the last three years. This is very welcome news, but we should wish for something even better, and if you, Gentlemen, can help us to do better we shall be very grateful.

It is perhaps too much to hope for that your deliberations will lead to any very great reduction in the incidence of silicosis; only a larger expenditure in preventive measures than many of our older mines are able to afford and continue to work can do that; but it will save many lives and much suffering, besides being of great financial assistance to the industry, if means can be found of arresting the disease or of preventing silicotic sufferers from infection with the tubercular germ. This we have hitherto tried to do by the arbitrary method of forcing a man to leave underground work; but the problem of finding him alternative employment is a difficult one, ranking, as he does, among the unfit, and leads to constant demands for more compensation.

I observe from the provisional programme of your discussions that every aspect of the silicosis problem will be dealt with. I am sure that this conjunction of the trained minds of many countries, each of you with a diverse and varied experience of the question, may be expected to have fruitful results. I have every confidence that this Conference will advance the knowledge of the causation and prevention of silicosis to a great degree. After all, it is prevention that counts most of all, and it is to the medical experts that we look for wise counsel in that direction. I wish you, Gentlemen, every success in your important task, the results of which, I am sure, will mark an important stage in the ultimate solution of this grave question. I have pleasure in declaring this Conference open. (*Applause.*)

The Conference was also addressed by *Dr. Keith Moore* (Australia), *Dr. Grant Cunningham* (Canada), *Professor Böhme* (Germany), *Dr. Middleton* (Great Britain), *Professor Loriga* (Italy), *Dr. Kranenburg* (Netherlands) and *Dr. Gardner* (U.S.A.) who expressed their thanks to the South African Government for the welcome which it had offered.

(The Conference adjourned at 12 noon.)

FIRST SITTING

Wednesday, 13 August 1930, 3.30 p.m.

Chairmen: MR. E. J. PHELAN and DR. L. G. IRVINE

Mr. E. J. Phelan: The first point is the election of the Chairman of the Conference.

ELECTION OF CHAIRMAN

Dr. Middleton proposed the election of *Dr. Irvine* as Chairman. *Dr. Gardner* seconded this proposal.

Dr. Irvine was unanimously elected Chairman.

The Chairman read the following telegram received from *Mr. Albert Thomas*, Director of the International Labour Office:

Please convey hearty greetings to Delegates and warmest wishes for a successful and fruitful conference. ALBERT THOMAS.

He also read the following letter received from *Mr. Thomas*:

LEAGUE OF NATIONS.

INTERNATIONAL LABOUR OFFICE,
GENEVA.

SIR,

On this, the first occasion upon which the International Labour Office has convened a Conference outside Europe, I venture to ask you to convey to its members my warmest wishes for its success, and to express to the Chamber of Mines my gratitude for the assistance which is making the holding of the Conference possible. I feel confident that the labours of experts drawn from four continents cannot but result in a substantial and valuable contribution towards the solution of the problem of industrial silicosis. The International Labour Office is happy to be associated with the Transvaal Chamber of Mines in this attempt to increase the protection of mine workers and workers in other silicotic industries against the dangers to which they are exposed from this disease.

I shall be grateful to you, Sir, if you will be good enough to convey my greetings and good wishes to the members of the Conference and to assure them that I shall watch the progress of their work with the deepest sympathy and interest.

I have the honour to be,

Sir,

Your obedient Servant,

(Sgd.) ALBERT THOMAS.

The President of the
International Silicosis Conference,
Johannesburg.

The Chairman proposed that this telegram and letter should be inserted in the Minutes of the Conference. *Dr. Orenstein* moved that receipt of this telegram and letter should be acknowledged and the thanks of the Conference expressed by cable.

The Conference unanimously adopted this proposal.

ELECTION OF VICE-CHAIRMEN

Dr. Loriga proposed the election of *Dr. Russell* as Vice-Chairman. *Dr. Pirow* seconded this proposal.

Dr. Russell proposed *Sir Spencer Lister* as second Vice-Chairman. *Professor Kettle* and *Dr. Loriga* seconded this proposal.

Dr. Russell and Sir Spencer Lister were unanimously elected Vice-Chairmen.

PUBLICITY OF PROCEEDINGS

Mr. Phelan: The Conference should decide at once whether its proceedings were to be public or private. He believed that the medical experts might prefer to conduct their discussions without the presence of laymen, but they might consider whether the closing session at which the suggestions and recommendations might be adopted should be public.

Dr. Middleton proposed that all proceedings should be private.

Dr. Loriga agreed.

The Chairman asked whether medical men not members of the Conference and representatives of the medical profession should be excluded.

Dr. Middleton: He was of the opinion that all sittings should be private unless the Conference otherwise resolved. It might at moments be useful to hold their discussions before a wider public, but an *ad hoc* procedure would allow this to be done.

Mr. Phelan asked whether *Dr. Middleton* had any objection to the issue of Press communiqués to keep the public informed of the progress of the Conference.

Dr. Middleton: Any communiqués should be approved by the Conference before they were issued.

Mr. Phelan: It would be difficult to consult the Conference daily for approval of communiqués, and he suggested that the staff might be allowed to issue communiqués to inform the Press of the progress made, but that where any references were made to differences of opinion, the Conference should first be consulted.

Dr. Mavrogordato suggested that a publicity committee be appointed.

Dr. Orenstein: The Resolutions Committee could fulfil this function.

Dr. Cluver: What would be the position of the medical Press? He was himself both an observer and the representative of the *British Medical Journal*, and the *Journal of Industrial Hygiene*.

Dr. Orenstein proposed that this question be referred to the Resolutions Committee.

The Chairman submitted the following text to the Conference:

1. The Conference as such should be regarded as private and the meetings confined to members and observers, and any publicity

given to its proceedings should be agreed to *ad hoc* by the Conference with the exception that a short resumé of the daily proceedings may be issued to the Press by the Secretariat.

2. Dr. Orenstein's proposal that Dr. Cluver's proposal, "a resumé of the proceedings might be drawn up on behalf of and for publication in the medical Press subject to the approval of the Resolutions Committee", be referred to the Resolutions Committee for a recommendation, is accepted.

The Conference unanimously adopted these proposals.

NOMINATION OF REPORTERS AND OF RESOLUTIONS COMMITTEE

Mr. Phelan: He proposed that the Resolutions Committee should consist of Dr. Irvine (Chairman), Dr. Russell and Sir Spencer Lister (Vice-Chairmen), Dr. Moore, Dr. Kranenburg, Dr. Böhme, and Dr. Orenstein.

He also proposed that reporters should be appointed for the three groups into which the subject was most easily divided as follows:

- (1) *Prognosis, After Care and Compensation:* Dr. Cunningham, Dr. Koelsch, Professor Hall.
- (2) *Preventive Measures:* Dr. Loriga, Dr. Badham, Mr. Roberts.
- (3) *Medical Aspects* (to cover Pathology and other subjects separately if necessary, but to submit a single report): Dr. Orenstein, Dr. Gardner, Dr. Middleton, Dr. Steuart.

The Chairman put to the vote the appointment of a Resolutions Committee constituted as proposed by Mr. Phelan.

The Conference unanimously appointed the Resolutions Committee as proposed.

Dr. Cunningham proposed that South Africa should be represented by a reporter upon "Prognosis, After-Care and Compensation".

Dr. Fisher seconded this proposal.

Dr. Orenstein: The South African point of view was that it would be better for the report to be drawn up uninfluenced by South African opinion.

Dr. Cunningham: It would be serious to omit all South African experience.

Mr. Phelan: The discussion would be open to all the members, and all that was proposed by the South African members was that it should be summarised impartially by overseas members.

Dr. Cunningham and *Dr. Fisher* withdrew their proposal.

The Conference unanimously appointed the reporters as proposed by Mr. Phelan.

Dr. Orenstein: Dr. Steuart, who had been appointed one of the reporters on "Medical Aspects", was an observer and not a member of the Conference.

Mr. Phelan: The International Labour Office was prepared to invite Dr. Steuart to be a member of the Conference.

Professor Kettle moved that Dr. Steuart should be invited to become a member.

The Conference unanimously adopted this proposal.

REPORTS OF THE PROCEEDINGS OF THE CONFERENCE

Mr. Phelan: A summary of the discussions of the Conference would be prepared by the Secretariat and roneoed, and members could hand in any corrections which they desired to make. The reports submitted to the Conference by the reporters for the three groups would eventually be published in a volume, which would include the various reports submitted to the Conference beforehand, and an account of the proceedings. This volume would also be published in French at a later date, and possibly in other languages. The non-technical discussions would be reported by the Secretariat. For technical discussions he suggested that speakers should prepare a summary of their own observations and hand it in to the Secretariat. In such cases delegates could hand in a summary in their own languages.

The Chairman asked whether the Secretariat would prepare a summary when the technical discussion was more in the nature of a debate than of a series of speeches.

Mr. Phelan: In such cases a stenographic note taker would be provided, and the Secretariat would also take notes. In this way a sufficiently accurate report would be obtained, which delegates would also have the opportunity of correcting.

Dr. Middleton asked whether the reporters would be required to take notes of the whole discussion.

Mr. Phelan: A distinction should be drawn between reports of the discussion and the reports eventually drafted to summarise the discussion. No disagreement was likely to arise over the summary of the discussion, but the reports themselves might be controversial. It was therefore desirable that the reporters should take some notes.

The Chairman suggested that speakers' summaries should be handed to the Secretariat within twenty-four hours.

PROGRAMME OF THE CONFERENCE

The Chairman: The programme proposed followed a logical sequence, viz. (1) Preventive Measures, (2) Medical Aspects, (3) Prognosis, After Care and Compensation. The South African reports were intended to serve as starting points for discussion, and the relevant parts of other reports would be taken at the same time. The subject matter rather than the papers themselves would be discussed. Papers 1 to 6 were more occupational in character than medical; anything bearing on the medical aspect could be discussed under the second heading, and anything bearing upon prognosis and compensation under the third.

THE CHAIRMAN'S SPEECH

The Chairman then addressed the Conference as follows:

Gentlemen, I have to thank you most deeply for the signal honour you have been good enough to confer upon me in nominating me as Chairman of this International Silicosis Conference. It is an honour which I am very proud to accept because I view it as a recognition of the place which the Miners' Phthisis Medical Bureau, whose work I have at the moment the privilege to direct, has gained for itself as a pioneer institution in

the field of industrial hygiene. I am only sorry that my predecessor Dr. Watkins Pitchford, who founded and organised the Bureau fourteen years ago during a time of stress and difficulty, and who directed its activities for a period of ten years thereafter, is not here to occupy in my place a position which he might so adequately have filled.

For myself I accept this honour with much diffidence. Although I have been in contact with the silicosis problem for eight and twenty years I am but a humble musket-bearer in the army of the Lord, and, when I face as I do now this friendly but critical gathering of the chosen experts in industrial medicine of many countries, I feel much like a company commander in a battalion of the line who is suddenly called upon to lead a composite force of all arms in a highly mechanised post-war army. Happily, little or no leading will be called for. But since you have been good enough to place me here I shall do my best to discharge the great privilege which you have conferred upon me, and which I esteem most highly.

My first duty is to welcome our visitors in the name of the mining and medical professions of South Africa as colleagues to meet whom is to us a very great pleasure and a unique opportunity. The other day when a solitary male hippopotamus which had wandered from Zululand along the coastal margin of Natal into the native territories beyond, a lonely tour of more than two hundred miles, reached a native kraal and rested with the cattle, the headman of the village gave orders that an ox should be slaughtered. This is the customary way in native circles to honour a distinguished visitor. Gentlemen, we hereby figuratively slaughter our ox in your honour. I hope that we shall establish at our meetings a close and friendly contact with each other which will make this Conference a landmark for each of us of good fellowship and good work done, and will make it also a notable landmark in the history of the silicosis problem.

That the International Labour Office has called this Conference to meet at Johannesburg is to us a great satisfaction. But it is a chastened satisfaction. For while we welcome the opportunity of having the benefit in the consideration of our local problems of the advice of so many trained minds, whose experience must in many respects have been different from our own, yet that satisfaction is tempered by the knowledge that probably the main reason why you have come to us is, that, owing to the magnitude and the unique concentration of the mining industry of the Witwatersrand, and to its rapid and intensive development within the space of a generation, the problem of silicosis has been with us at once of greater gravity, and has had results which have been more clearly apparent than might have been the case in other countries, in which the mining communities may be of older standing and individually perhaps relatively smaller and more scattered. Our satisfaction is tempered also by the fact that although after many years of intensive effort we can claim a very considerable measure of success, we have as yet reached no final practical solution of the difficult problem of the prevention of silicosis.

It is significant of the local attitude toward "miners' phthisis" that the initial suggestion to hold this Conference at Johannesburg should have come from Dr. Orenstein on behalf of the mining industry of the Witwatersrand, and that the Transvaal Chamber of Mines has actively co-operated with the International Labour Office and the Government of the Union of South Africa in making this meeting possible. Since the time that the gravity of the menace of silicosis was fully recognised there has been manifest a close co-operation between the Department of Mines

of the Union Government and the gold mining industry in the investigation and application of preventive measures. This wholesome spirit animated the work of the first Miners' Phthisis Prevention Committee, to which we in this country look back as having set on foot a really energetic and systematic policy of prevention. Our present meeting is the outcome of the same spirit, which alone can render possible a satisfactory solution of this grave question.

I do not wish to detain you overlong with preliminaries. You have come here to see things for yourselves and not to listen. But perhaps you will bear with me if I offer at the outset a few very general remarks upon the subject-matter of our programme, as viewed particularly in the light of our South African experience, inasmuch as this is the only aspect of the general problem of silicosis of which I have any direct knowledge. The papers which have already been contributed to the proceedings of the Conference are in your hands. Those prepared by local mining and medical men may appear to bulk largely in the programme, but they are designed to serve simply as an introduction to the general discussions on the several aspects of our subject. They will be taken as read, and will thus provide a short and happy method of disposing of the local contributors, and of opening the way to discussions, which, as is plain from the contributions from other countries which have already reached us, will be enriched by the results of wide and intensive studies of the general problem elsewhere.

The contributions already received from other countries, together with others which may be forthcoming, will fit naturally and most profitably into the course of the relevant discussions of the different aspects of our subject, and will be of especial value in widening their scope. In South Africa, so far, the silicosis problem has been practically a mining problem, and the local contributions are accordingly practically confined to the subject of the silicosis of the gold miner. The record of experience derived from other industries and other countries will serve to correct this apparent limitation. I trust, however, that you will forgive me if the few remarks I have personally to offer may also appear to have an unduly local colour.

§ 1. — The topics set down for our discussion follow a logical sequence. The first main subdivision of our programme deals with the causation and prevention of silicosis. The first six papers deal accordingly with the physical and chemical characters of the gold-bearing conglomerate and the contiguous country rock of the Witwatersrand reef, with the history of local occupational conditions, the nature of the preventive measures which have been adopted, and with the general history of silicosis on the Witwatersrand.

The problem of what constitutes "silica risk" and of how it is to be countered is obviously fundamental since if that can be solved the pathology of silicosis becomes of no more and no less special significance than that of any other disease, and the matter of compensation may be left to look after itself. The question of the aetiology and prevention of silicosis is plainly primarily a medical one, and I trust that this Conference will be able to contribute to the formulation of some definite lead in this respect, since without a well-informed and convincing lead from the medical side, the mining engineer or the industrialist must remain at a loss. As a world-famous engineer said at a recent Empire Mining Congress: "Let the physiologists tell us plainly what they want done, and we shall find ways of doing it for them." Hitherto the physiologists and other medical people have themselves been pursuing a painful process of self-education. Are we now in a position to do better?

We know that certain dusts which contain free silica are phthisis-producing dusts, and that other dusts which also contain free silica are not, and the accepted view at present is that the difference lies in the presence in the latter kinds of dust of other constituents, which nullify the harmful potentialities of silica. The facts are there, but their explanation is still somewhat obscure, although we can picture several possibilities. And can the facts be applied in a practical way to the prevention of silicosis, either in mining, or, if not in mining, in other industries in which "silica risk" exists, and in which dilution with other "antidote" dusts might be possible? The line hitherto taken in South Africa has led far away from any attempt in this direction, although Dr. J. S. Haldane has more than once called our attention to its possibilities.

The history of silicosis in South Africa, and indeed in all other countries, makes sad reading, and to those who have worked in close contact with the disease throughout, the personal experience has been more sad. To the actual sufferers it has meant cases of disablement or death which have run into thousands. I think that those of you who have read the several local contributions to the history of the subject, to which I have referred, must have been impressed by the manifest candour of the writers. Our ten years of ignorance, our further ten years of partial realisation, hesitation and tentative improvements, our eighteen years of increasingly energetic effort to deal with the problem have been set down without extenuation, and make up, I suppose, the common story of such things the world over, except that with us that story has been condensed into the period of one generation instead of being spread out over several or many.

The slow awakening to the gravity of the situation and to the fact that we have in silicosis a condition which is individually the most important of all occupational diseases, and one most difficult to deal with, particularly under mining conditions, is faithfully reflected in these papers. At the outset this circumstance was due in part to simple ignorance of the character and extent of the danger, in part to the dominance of the metal mining tradition of those days. The coal mining tradition stood then, as now, for ample ventilation to combat the danger of mine gases. The metal mining tradition, exempt as most metal mines are from any such obvious and immediate risks, was still content to rely upon natural ventilation. And there is an immense inertia in tradition the more so when it has become extensively embodied in actual material structure whether above or below ground.

The main lesson of the history of silicosis appears to be that in metal mines which create a phthisis-producing dust there are just as cogent reasons for ample ventilation as exist in coal mines, since in the last resort we are dealing, in an atmosphere laden with impalpable silica dust, with what is for practical purposes a dangerous gas, perhaps more extensively dangerous to life in its remote effects than are fire damp or black damp with their immediate and obvious risks. Had this been generally realised thirty or forty years ago we should have been spared at least a large part of our troubles. And although mine ventilation has for many years received great and increasing attention on these fields we are still to-day in this respect hampered and handicapped by the heritage of the old tradition, as embodied in the original layout and mode of working of the older mines.

It was not until the report of the Miners' Phthisis Medical Commission was published in 1912 that the widespread character of the menace of silicosis and the real gravity of the situation were recognised in this

country, and that year marks the beginning of the really energetic measures for the prevention of silicosis which have since been taken. Even so, medical men and engineers were still agreed that water was the main remedy.

Since that date preventive policy has been increasingly systematised in the measures summarised in several of the papers before you; measures which have incorporated and extended the many important preventive methods which had already been introduced.

They form to-day a large and complicated system. There are the "medical methods" of strict examination of recruits, and of the detection and removal of the subjects of active tuberculosis; and there are the "engineering methods", such as provision for adequate ventilation, and the regulation of shifts and blasting and also those methods which depend upon the use of water to prevent the escape of dust into mine air, or to lay it when actually in suspension. The success of this combination of measures has undoubtedly been great. One may claim, I think with truth, that the number of cases of silicosis which are arising annually to-day is only about one-third of the number which were arising fourteen or sixteen years ago. During the past three years there has been a continuous drop in the production rates of the disease. Nor do I think that the potentialities of our "present day" methods are by any means exhausted. Within the last ten years, to take one feature only, the whole practice of rockdrill work has been completely and favourably revolutionised, and such things take time to show their full effect. The improvement attained therefore has been substantial, and there is reasoned hope for further improvement in the future. But, although we are satisfied so far, we are also disappointed. We have scotched the snake, but we have not killed it. We realise in particular that although water will take one a large part of the way, it will not take one all the way, and that its use has certain positive disadvantages both hygienic and economic. Hence the minds of medical men and engineers are turning to-day to the question: have we not been overdoing water? Could we not do better with less water, and a greater extension of alternative methods? On this point the experience of other countries will be welcome.

§ 2. — The second main subdivision of our programme is concerned with the strictly medical aspect of silicosis.

In this section discussion will be opened by the papers on the "Aetiology of Silicosis" and on "Experimental Silicosis" by Dr. Mavrogordato. The pathology, radiology and symptomatology of silicosis as met with in South Africa are dealt with in three further papers. The first of these, contributed by Drs. Strachan and Simson, is written from the strictly pathological standpoint. The same writers have co-operated with members of the Medical Bureau in the preparation of the two succeeding papers on "The Clinical Pathology, Radiology and Symptomatology of Silicosis", in which that condition is considered particularly from the standpoint of its practical diagnosis as an occupational disease. These two papers present the conclusions drawn from a careful correlation of the results of pathological, radiographic and clinical examination in a consecutive series of 400 individual cases of silicosis occurring amongst European miners, in each of which a post mortem examination was performed, and in each of which a radiographic and clinical examination had been carried out within the six months preceding death. It is upon this triple correlation that the standards of diagnosis and classification adopted by the Medical Bureau are based. It forms at once the front line and the last ditch in our trench system of diagnosis.

It is common ground that it is the high potential predisposition to the

ultimate development of an active tuberculosis that is the chief factor in making silicosis the serious condition which it in most cases is. The relation between silicosis and tuberculosis has been repeatedly discussed, and will, one presumes, be once more thoroughly discussed at this Conference.

The view taken in these papers is that a non-tuberculous nodular fibrosis of the lung due to the arrest of silica dust within that organ is a definite and distinct pathological process, and is the predominant feature of the great majority of cases of what is clinically termed "simple silicosis" particularly in its earlier stages, and further that that process may advance *as such* up to a point, but to a very considerable degree without the intervention of tuberculous or other infection, even although the affected man is removed from underground work.

On the other hand, not only are a number of cases of silicosis obviously complicated from the outset by active tuberculous infection, but pathological observation leads one to the view that there exists in many and probably in most cases of clinically "simple" silicosis, from the time that it becomes definitely detectable, some latent circumscribed focus or foci of low-grade tuberculous infection in association with certain of the silicotic lesions.

Some such infective lesions may originate from persistent active foci dating back originally to a primary infection during early life: some may be due to a limited re-infection occurring during and associated with the early development of the silicotic process.

This feature forms one factor in the explanation of the tendency to the ultimate development of active tuberculosis which is manifest in so many cases of silicosis. The future history of the case will depend on whether such foci remain inactive, or become active and progressive or whether again a further infection occurs from a source outside the lungs. Other features in the situation however heighten that predisposition. The sites of the silicotic lesions offer possible points of arrest of bacteria in the lymphatic system of the lung. And probably the most important factor of all is the circumstance, as has been shown in particular by Gye and Kettle, that finely divided silica acts as a soluble cell poison and has in consequence a specific effect in determining the selection by a tuberculous infection of sites where silica is aggregated.

The local toxic action of silica may also go far to explain the characteristic modification of the silicotic process by chronic infections, and particularly by a chronic tuberculous infection, with the production of slowly progressive infective lesions of comparatively low virulence, accompanied by an excessive fibroid reaction, which form so striking a feature of many cases of the disease. To this characteristic modification of silicosis the general term "infective silicosis" or in the more restricted sense "tuberculo-silicosis" has been applied.

It would appear that the typical effect of silica dust when inhaled in excessive quantities over long periods is, generally speaking, first to cause a "dust bronchitis", then to produce the simple nodular "dust fibrosis" which is "silicosis", and finally to lead on to a condition of "infective silicosis" and a true "dust phthisis". But there are in individual cases many variations in the course of this development, determined mainly by the relative preponderance of the dust factor or of the infective factor, and by the earlier or later manifestation of the latter.

At this point I would call your attention to the photographic *Atlas of the Pathology and Radiography of Silicosis* which lies beside each of you. It contains a reproduction of the original illustrations to the three

papers I have just mentioned and has been prepared for and is presented to the medical members of this Conference through the courtesy and generosity of Kodak, South Africa. It will form, we hope, a useful addition to the literature of the Conference, and I am sure you will desire that the cordial thanks of the members of the Conference should be conveyed to the donors, Kodak, South Africa, for their handsome gift-book. To Dr. Steuart and myself it is especially pleasing, because the endeavour to secure satisfactory reproductions of radiographic negatives of chest conditions has hitherto been a standing heart-break; the reproductions achieved by the printer conveying for the most part nothing of any value, either to their authors or to other people. This is particularly unfortunate in the case of such a condition as silicosis, in the diagnosis of which radiography forms so essential an element. But the *Atlas* in your hands has, I think, made good this deficiency in a very satisfactory manner, so far at least as the material which it contains is concerned.

If I may be so bold as to offer the suggestion, I should wish to see this Conference consider particularly three points in connection with the pathology and diagnosis of silicosis.

First: Can we agree from the fundamental pathological standpoint upon a definition of what constitutes silicosis *regarded as a definite condition of disease*, the presence of which renders the affected man capable of being certified as suffering from a specific and identifiable occupational malady, which may constitute a valid basis for a possible claim for compensation in that respect?

Second: Can we agree upon a terminology which will render a description of the characteristic lesions and varieties and possibly the "stages" of silicosis mutually intelligible to observers in different countries?

Third: Can we agree upon a terminology descriptive of the various types of radiograph found in cases of silicosis, or other conditions of pulmonary fibrosis, which may similarly be mutually intelligible to different observers? The Medical Bureau has in this regard tentatively put forward its own private conventional terminology for the consideration of the Conference. Suggestions for improvement will be welcome.

I think it would be a matter of real value if some agreement could be reached on these three points which would facilitate mutual understanding in the future.

§ 3. — The third subdivision of our programme deals with the incidence and progression of silicosis, with the legal aspect of the disease, and with the question of compensation.

There are included here several papers, mainly from the administrative side, on the work of the Medical Bureau and the Medical Board of Appeal, on the sanatorium treatment of silicosis and on the examination of native mine labourers.

Finally the programme concludes with three papers which deal with the development of miners' phthisis legislation in South Africa, with the question of compensation and of other ameliorative measures undertaken by the Miners' Phthisis Board, and with the great monetary burden, past, present and prospective which the disease has placed upon the mining industry. Yet great as this burden has been it represents only a portion of the very serious economic loss which has been occasioned in South Africa by this "scourge of the metal miner"

Miners' phthisis, we are told in these papers, has been in this country the subject of five Government Commissions, the earliest having been appointed in 1902, of ten Parliamentary Select Committees, and of nine Acts of Parliament, the first of which was enacted in 1911 and the last, or what is for the present the last, in 1925. The subject has thus occupied a great deal of the attention and time of the legislature during the past nineteen years. In general each successive Act has been marked by an increase in the amount of the awards payable to fresh cases, and by additional provisions for surviving beneficiaries under previous Acts or for the dependants of deceased miners, and one cannot say whether finality in these respects has yet been reached. Legislation on the subject has in this way grown to be extremely complicated. I fear that those who approach the study of the present Act will find in it an extraordinary maze of "sage provisos, sub-intents, and saving clauses" which only those who know its history can hope to understand without a guide.

Such a legislative history in itself proclaims that the problem of compensation has proved to be a difficult one. And the standing difficulty as you will well understand is, that, viewing silicosis as an industrial "injury", the "injury" inflicted is in the majority of cases not a stationary one, but one which tends to get worse, yet which does so very erratically and in general over a period of a good many years. As I have said elsewhere the important point about an early case of silicosis is not what he is at the moment but what he may become. Mr. Spence Fraser has computed the average expectation of life of an early case of the disease when first notified, to be about fourteen years, with a wide variation above and below that average. In the earlier stages of silicosis in which disability may be absent and is at all events not serious, the real crux of the question is not so much medical as economic—it is almost wholly that of securing alternative employment by the men affected. In the later stage it is one of definite and permanent invalidity. The question of serious invalidity has been met since 1919 by the payment of a life pension to those who suffer grave incapacitation from the disease; the earlier stages are dealt with by single lump sum awards. The practical problem is complicated by the fact that in this country, in which most of the unskilled labour is done by the native, alternative avenues of employment especially for partially disabled men are probably less easy to find than in other more fully industrialised communities with a homogenous population. It may be suggested also that the term "miners' phthisis" itself has unfortunately acted as a deterrent in the obtaining of employment by beneficiaries and has also had, perhaps, in a good many instances, a subtle psychological effect in this and other directions upon the beneficiary himself. It seems unfortunate therefore that this term appears to have become entrenched in the titles of the local Acts although it is nowhere mentioned in their substance. This is not, one would suggest, an example to be followed.

One word in conclusion. The Medical Bureau by the sanction of the Minister of Mines has recently installed a new three-phase X-ray generator from which we hope for much and of which we invite your inspection.

May I also venture to call your attention to the plans of the proposed new Medical Bureau which adorn these walls. The Bureau has long outgrown the swaddling clothes generously presented at its birth by the South African Institute for Medical Research and has indeed long been hampered and even endangered by insufficient accommodation. These plans are a vision of a larger future. They are an earnest of how seriously the present Government of the Union regards the matter of silicosis in

South Africa and of the high standard of efficiency for which it is prepared to provide the means and of which it expects to obtain the realisation in the detection and investigation of the disease.

May I be permitted to offer a final suggestion, namely, that this Conference should not terminate without making arrangements for future international intercommunication upon this very important matter of silicosis and allied dust diseases by the establishment and maintenance for example of central libraries of the literature of the subject under the relevant Government department of each country, and possibly by formulating a provisional programme of future desirable lines of research, and by endeavouring to secure a greater international co-operation in carrying out such a programme than at present exists. The experience of different countries and different industries differs in important respects, and some systematic means of collating and comparing that experience might be found.

And now, Gentlemen, I have done. I must thank you for hearing me so patiently. I have attempted merely to offer you a very general perspective of the silicosis question in South Africa. I am well aware that this is but one of many aspects of the general problem which will occupy our discussions, and that it has many other aspects. But it is best to speak of what one knows.

VISITING MEMBERS' EXHIBITS

After some discussion it was agreed that the exhibits brought by visiting members should be shown at 8.15 p.m. on Friday, 15 August.

(The Conference adjourned at 5.55 p.m.)

SECOND SITTING

Friday, 15 August 1930, 2.30 p.m.

Chairman: DR. L. G. IRVINE

The Chairman proposed that the members of the Miners' Phthisis Medical Bureau and the medical staff of the South African Institute for Medical Research should be permitted to attend the sittings of the Conference.

The Conference unanimously adopted this proposal.

OCCUPATIONAL CONDITIONS AND METHODS OF DUST PREVENTION

Dr. Middleton: The scheme prepared by him and circulated to members of the Conference did not attempt to cover silicosis throughout the world, but only as it occurred in Great Britain. By "ætiology" in the title of his first heading he intended to cover all the factors in the causation of the disease silicosis.

The first factor was exposure to silica dust, by which he meant dioxide of silicon in a free state, and not chemically combined in the form of silicate. British legislation was based on the view that disease was

caused by silica dust, and a precise definition of what was silica dust was therefore necessary. During his own investigations into silica processes he had never been able to determine the standard of air dustiness which produced silicosis. He was aware that in South Africa and in Sydney, Australia, a figure was laid down in connection with preventive measures. It was however impossible to discuss a figure until all the facts were known. He suggested that the Conference might usefully lay down a basis for the comparison of results.

In England the apparent exposure to silica dust necessary to produce silicosis had been short; there was a case of fatal result from tuberculosis with evidence that it had been produced by silicosis after two and a half years' exposure.

A third factor which must be taken into account was the extent of exposure period plus latent period, and the relation of these to each other.

The influence of intermittency of exposure by alternation of occupations had preventive bearing. When dust was reduced by certain hygienic conditions silica was still present, but intermittent employment might save the worker from pulmonary disease or at least from disablement. This had already been attempted in some factories in Great Britain.

Another important aspect was the influence of the presence of other dusts on the occurrence and course of silicosis. Some dusts were referred to as restraining silicosis; this was said to be due to colloidal or to chemical action. Other dusts had a physical action in preventing inhalation by aggregation. Some rocks containing clay, for instance, produced large aggregations of particles too large to be carried to the alveoli.

As regards dusts other than silica which might produce evidence of lung change similar to silicosis, he pointed out that a worker might worsen his condition by passing from one industry to another. Asbestos damaged more rapidly than silica. Sandstone grinding was very largely replaced by the new forms of grinding, such as by emery; there was not yet sufficient evidence to indicate the effects on the health of the workers of these processes.

Different industries produced different types of pulmonary disease. Workers in the refractories industries and potters died alike of silicosis, but the type of silicosis was different. Living conditions might have some influence, but in highly skilled trades, such as that of stone masons, where the standard of living was good, the mortality was high.

Finally, as regards the occurrence of infective processes, he asked the Conference to express an opinion on the statement that silicosis was not developed in a healthy lung, but must be preceded by inflammation.

Dr. Russell: In the United States the proportion of silica dust in various industries varied from 1 up to 90 per cent. The medium between the two was probably the average type of dust. Granite dust in the United States contained less silica dust and a higher percentage of other chemicals, to which its inhibitory action had been attributed. The development of silicosis in the granite industry varied in direct proportion to the extent of exposure (30-35 per cent.).

Dr. Moore: The period of exposure necessary to contract silicosis depended primarily on the individual. In the Bendigo mines the silica content was over 90 per cent. On the West Coast of Tasmania it was about 60 per cent. and in Kalgoorlie a little more. The Bendigo statistics were not very trustworthy, since the miners were loth to be examined, but about ten years seemed to be the average period to produce silicosis.

In Kalgoorlie it was nearer sixteen years. Silicosis seemed to depend pretty accurately on the silica average in the country rock.

Dr. Böhme: German experience showed that sand blasters and grinders had well-developed silicosis sometimes after three years. Generally silicosis developed after nearly ten years. Comparable statistics were required in order to distinguish between various dusts. He did not believe that coal dust had any inhibitory effect. There were cases of workers employed on hard rock for three years and then working ten years as colliers who were still developing silicosis and were no more protected than those who had never worked as colliers.

Dr. Koelsch: In his short report just distributed, the question under discussion had been already emphasised, as well as the decisive influence of free silicic acid, and attenuation of the effect of silicic acid by combination with other substances, etc. As regarded the period of time required to develop the disease, he had seen two cases of severe silicosis after two-and-a-half years of employment on sand-blasting; the workers in question were aged twenty-five and thirty years respectively. Timely suspension of work might in the earlier states bring about retrogression of the silicotic changes. Yet in this instance, as likewise in the development of silicosis, the individual constitution and the resistance capacity of the tissues is of capital importance.

Dr. Fisher: Three factors were to be considered: (1) concentration of dust; (2) time; (3) percentage of free silica. It was desirable to use the same konimeter throughout, preferably that which gave the most delicate results. In Great Britain there was up to 60 per cent. free silica in mines. He would like to know whether this was a safe figure. He also asked the Conference to express an opinion as to the best konimeter. Two preventive measures were water and dust traps. He would like to see an efficient mask invented; a mask which did not obstruct respiration would keep back half the dust, and this might be sufficient if 300 particles per cubic centimetre was a safe count.

Dr. Badham: The air of parts of the Rand mines which he had seen did not contain 4 milligrams of dust per cubic metre since dust was not visible under that figure. At about 400 particles per cubic centimetre (using Owen's konimeter) dust particles below 10 microns with a size frequency ratio of 3 did not form a visible cloud.

The size frequency ratio of particles was important, for by its study it could be seen that in a dust such as that referred to, though the 1 micron particles were very numerous, the 2 micron particles contributed the greatest percentage of surface; for this reason he did not subscribe to the belief that ultra-microscopic particles were culpable in producing silicosis. He thought that further work should be done in dust counting to ascertain definitely the amount of silica which could produce silicosis. It appeared that the average exposure on the Rand was about 1 milligram per cubic metre, and if this was so then the prospects of their attack on the disease by reducing the amount of dust inhaled seemed rather hopeless.

He hoped that further attempts would be made in South Africa to correlate the konimeter counts with the sugar-tube results. He thought that Owen's konimeter was capable of doing all the work now being done on the Rand with another instrument and stated that it produced a record from which carbon particles and water-soluble salts could be eliminated by selective counting.

He thought that more attention should be given to the particulate

count and urged South Africans to try more recent methods of dust estimation.

Dr. Kranenburg: It was necessary to know how much silica was contained in different kinds of stone. In marble, for instance, silica was always present. Italian marble had 6.7 per cent. free silica and Belgian marble 1.2 per cent. The figures for samples of limestone were: Belgian, 1.1 per cent., 1.5 per cent., 0.1 per cent., 0.5 per cent.; French, 0.6 per cent., 0.4 per cent., 0.6 per cent.; German, 0.2 per cent.

Dr. Pirow (in reply to Dr. Fisher): The konimeter standard must depend on the percentage of free silica present. He had found with the Rand percentages that 300 particles per cubic centimetre could be taken as a standard, but he was not convinced that it was safe. Water and ventilation were the only preventives in South Africa. Dust traps had been tried, but the general opinion was that they were much too dependent on the personal element and could be applied only in isolated cases. Up to the present no fool-proof and practical masks had been produced. He did not agree with Dr. Badham on the relation between microns and the count of particles per cubic centimetre; 400 or 500 particles per cubic centimetre could be found; it depended on the fineness of the dust. Hand-drilling produced much coarser dust than machine drilling. It was usual to state the percentage of coarse particles (those above 5 and 10 microns). Owen's konimeter was used by the Mines Department as a check, but required too much care to be practical for routine work. He regarded the South African results as only comparative. Efforts to correlate konimeter and sugar-tube results had not been very successful. The concentration entirely depended on the percentage of free silica in the dust. The present concentration was known to be dangerous and attention was being given to the highest concentrations. Attempts were being made to standardise konimeter practice.

Dr. Cunningham: In Ontario gold mines the exposure necessary to produce ante-primary silicosis was nine to ten years. The percentage of free silica was about thirty to thirty-five. This period seemed short compared with South African experience. It was perhaps impossible to correlate the various dust-counting instruments used in different countries.

Sir Spencer Lister: There was some confusion in the term "silica content", which might refer to rock or to dust. Only the silica content and amount of the atmospheric dust mattered and the size of the particles. If very small particles were produced the weight standard was of small significance since the large particles were innocuous.

Dr. Mavrogordato: He hoped the Conference would initiate an epidemiology of silicosis. They needed to know where it occurred, what was its comparative incidence, and what were the comparative conditions. Free silica in country rock was a very important factor, but they must know the percentage of free silica in the air, and also what other dusts were present. Of these dusts, which increased liability to silicosis? In Great Britain there were two kinds of firebrick with the same silica content, but the incidence of the disease was very different.

It was easy to remove all visible dust, leaving all the dangerous dust, so that size frequency was of great importance. A method which was effective for coarse particles might be useless for fine dust. Water would keep down dust, but how was dust to be removed when it was

once in the air? In rapid cases of the disease what part was played by the size of the particles?

Masks were most likely to be successful with a system of positive pressure, but he did not believe that other types of masks at present available could stop the dust without stopping the air when the wearer was at work.

South Africans used a dark ground konimeter count and believed that the results were as good as with oil immersion and a light ground. He agreed that 500 particles per cubic centimetre with a dark ground amounted to 1 milligram. When the count was down to 300 it meant a dark ground count in South Africa.

Dr. Kranenburg asked whether it was possible to free air of invisible dust by precipitation (flocculation).

Mr. Boyd: South African dust counts were regarded as empirical. The sugar-tube method had been retained for purposes of historical comparison. Finality had not been reached with the konimeter. It had needed much experiment to reach the present method of counting, which had now been in use for two years. A Committee was at present sitting to investigate methods and to devise a standard. Owen's konimeter was too delicate for routine work. In size classification the largest particles were found in the ore-bins, next in hand-drilling, and then in machine-drilling, which produced the finest dust except blasting.

Dr. Middleton: Working conditions in factories sometimes made the use of water or exhaust draught impossible and masks were therefore required. No standard mask existed, and those on the market were not of known value. The Home Office was investigating the possibility of a mask to protect against fine silica dust, while at the same time providing for an air pressure which would allow a man to wear it some time without fatigue.

He thought that the Conference should adopt a resolution of a progressive kind on the subject of correlating the results of dust counting. The percentage of free silica seemed to be in relation to the silicosis induced in the lungs, but this was an unproved assumption. He hoped that the Conference would draw up a scheme for the exchange of views and knowledge. The dust counts which he had reproduced in his paper led nowhere. He had always used Owen's konimeter and found it useful, but it was impossible to reach a standard for a safe dust figure. In South Africa conditions from mine to mine were fairly comparable, and it was therefore easier to arrive at a figure, though he was aware of the difficulties, partly due to the change in instruments and underground conditions. He did not personally favour dark ground illumination, because it did not eliminate carbon particles, which were very numerous in most dust samples in Great Britain. Silica dust tended to remain discrete, but it was impossible to say how far the same mixture of dust occurred in the atmosphere as in the smear. Where moisture was used to lay dust the number of particles below two microns in a droplet might be over 800. He urged that a uniform method should be adopted so as to render results comparable.

Professor Hall proposed that a sub-committee should be appointed to consider the possibility of standardising dust counts.

Mr. Roberts: A sub-committee would not have sufficient time to reach a satisfactory conclusion. A Joint Committee of the Mines Department and the Chamber of Mines had been working at the question for more than a year.

The sugar tube had given good results, but it showed weight and not numbers. An attempt was now being made to reduce numbers with the help of Owen's and other konimeters. Mining engineers would endeavour to carry out whatever the silicosis experts recommended. It was possible that ventilation ought to be increased and humidity decreased, even at the risk of more dust.

Dr. Loriga: He had nothing to add as cases of silicosis are rare in Italy, and such as have occurred have not been recorded in detail. He had therefore no communication to make in regard to the disease, but would like to ask the members of the Conference, in view of the fact that up till now exclusive mention has been made of silicosis: were there not other dusts which produced forms of pneumoconiosis other than silicosis?

Reference has been made to the outbreak of the disease after a lapse of ten, fifteen, and twenty years. How was it possible to measure such periods with any accuracy? Silicosis did not resemble an acute disease occurring at a given moment, but it was a disease with slow progressive evolution. What stage in the disease was considered to constitute its commencement? He believed it was when the lungs could no longer eliminate the dust so that the disease either increased, was arrested, or led on to death, but he would like to know what criterion was used in stating that a worker was now no longer healthy but must be regarded as silicotic.

Firstly, what dusts favoured or retarded the action of silica? And what dusts could reach the lungs and get fixed there? Neither the Sub-Committee nor the Conference could solve these problems. It would never be possible to say precisely what quantity of dust would provoke silicosis since it was not absolute quantity or quality that mattered but the relative quantity. One individual might resist a proportion of 100 particles per cubic centimetre, while another would react unfavourably to 50 particles per cubic centimetre; there was further the whole question of the rate of inhalation. A medical inspector who was inactive would inhale much less than a worker engaged in heavy physical labour. Accelerated respiration would in the latter case cause inhalation of particles which the former could not inhale. The special pathological aspect seemed, therefore, incapable of solution, but the general pathological side of the problem was already solved. In each case it was necessary to direct attention to the size of the particles, the rate of inhalation and possible penetration and fixation—that was in each given case, but a general estimation of these to meet all cases was impossible.

The Sub-Committee was therefore obliged to confine its attention to the hygienic and practical issue, that was: (1) to determine the standard amount of dust tolerable in the air of a mine for an individual of average health; (2) to determine means of assuring reduction of dust to this amount by analysing the air and to determine the best methods of estimating the dust present in the atmosphere.

In regard to (1) the standard would depend on such factors as quality and admixture with other dusts.

In dealing with the hygienic aspect secondary questions must be left out of account. The problem was that of reaching agreement as to a standard limit for all countries to be determined by future research.

Dr. Orenstein: Two different questions were answered by the dust count: (1) in a given industry it determined how conditions varied (where conditions, as on the Rand, were always comparable); and (2)

for critical scientific study an international standard could be laid down.

About 100,000 samples were taken annually on the Rand, so that a laborious procedure was impossible.

He added that "dark ground illumination" was not the true dark ground of the microscopist.

He suggested that the reporters might co-opt one or two experts to consider a recommendation for an international standard in dust counts for scientific research, and the Resolutions Committee should recommend the experts in question to the reporters.

The Conference unanimously adopted this proposal.

(The Conference adjourned at 5.10 p.m.)

THIRD SITTING

Saturday, 16 August 1930, 9.45 a.m.

Chairman: DR. L. G. IRVINE

PREVENTIVE MEASURES AND UNDERGROUND CONDITIONS

The Chairman suggested that the discussion should be broken into specific subjects.

1. The Relative Value of the Use of Water and of Ventilation in the Prevention of Silicosis.

Dr Mavrogordato: Water had three uses: (1) To keep dust out of the air at the site where the stone was broken; water was very efficient, but the finest dust would pass any form of water. (2) To give a "fly-paper" effect; this had been found less effective; tests made on the Rand in 1912 and 1913 proved that other preparations—e.g. Mr. Ussher's treacle—were no better than water; it was doubtful how much had been gained by keeping the place continually wet, since the air was thus very greatly humidified; when a development end had dried for two days it maintained 80 per cent. relative humidity, but when washed it went up to 100 per cent. almost immediately; it would therefore be desirable to revert to the "fly-paper" effect with some other preparation than water. (3) To remove dust from the air. Lord Lister had said at Berlin in the early eighties that he was ashamed of ever having suggested that spray could affect objects of the size of micro-organisms in the air. The Commission on the ventilation of the House of Commons came to the conclusion that fine particles could be blown through any water-screen. With a high temperature high relative humidity was a great inconvenience; organisms kept alive more easily in a wet atmosphere and this was very disadvantageous as regards infective silicosis. Even though the dust was not highly concentrated in the air, silicosis was still produced in a virulent form. The infective element, therefore, played a considerable part.

The effect of dust did not end with inhalation. He had produced a microscopic fibrosis in seven months with fine silica dust (flint). Progress continued over nine years, however, must be due to some element superimposed on the dust. The droplet of 800 particles to which Dr. Middleton had referred was bombarding the lung with shrapnel instead of rifle fire. It was difficult to infect an animal by blowing dry organisms, but 90 per cent. positive results were obtained from spraying. Water facilitated the entry into the lungs of particles in the air and it was therefore preferable to deal with dust by ventilation.

He doubted whether an effective ventilation system was possible. Silicosis was made in twelve years on the Rand, and in about ten years in Canada and Australia. But in English potteries and grinding a twenty-five year exposure was found. Dry methods of control therefore made silicosis much more slowly. In English collieries there were probably many abnormal lungs, but they had been at work twenty-five years or more. Wet surfaces should be kept down as much as possible and there should be all possible ventilation. Mines with a concentration of dust like the Rand would be much more healthy if this concentration were secured by dry methods. It was however impossible to avoid the use of water on the Rand and therefore the surface must be kept down by avoiding leaks in drills and diminishing all spraying; water would keep dust out of the air without great surface.

Dr. Middleton: In factories water and localised exhaust draught were the two main methods of dust prevention. Water should be used for the suppression of dust at its point of origin, and in some processes could be effectively used upon this principle. If sufficient water was used dust never arose, but ample water supply was essential. If no water could be used at the actual moment of fracture of rock steam had been found efficient, but it must be in a saturated condition. If steam was allowed to fall on a dust cloud at its point of formation, the dust would form aggregates and fall. Argillaceous matter in the dust facilitated the formation of aggregates.

The problem of high temperatures did not arise in factories, so that humidity and dissociation did not occur. Grinding with sandstone wheels of 2 to 7 feet diameter gave working faces of 2 or 3 to 18 inches. The idea at one time prevailed that dust was not produced at wet grinding but that if sparks escaped into the atmosphere dust would also. In Great Britain the revolving stone often dipped into a water trough, but Owen's konimeter had proved that the metal cut through the wet surface and gave rise to fine dust from the stone.

Dry grinding had once been the most dangerous industry; Calvert Holland, of Sheffield, had shown that no dry grinders in the middle of last century reached forty-five years of age. It was still carried on, but with an exhaust draught and the incidence of silicosis was now much less than among wet grinders. There was thus a strong argument for the use of exhaust draught.

British regulations required the suppression of dust as near as possible to its point of origin, either by water or by localised exhaust draught. The dust figure was sometimes very little reduced by exhaust draught—perhaps by 25 per cent. The failure of the localised exhaust draught was due to the idea that it had some selective effect. This was not the case. The system ought to be such as to remove a sufficient volume of air from the point of origin of the dust and to ensure a directional current. The addition of baffles at the sides of the workplace would enormously reduce the amount of air to be removed. It was a special

engineering problem to correlate the size and speed of the fan, the capacity of the duct, and the volume of air to be moved.

Processes were sometimes improperly carried on between the point of origin of the dust and the exhaust system. General ventilation should never be combined with a localised exhaust draught system unless the two could be correlated by some means. The pull of the fans caused directional currents towards them and the two systems together always produced eddies. Dust commonly escaped from the hood and passed directly upwards and was then diffused generally throughout the room, so that high concentrations could be counted.

It was hard to determine the relative value of water and exhaust draught; there was a place for each. Water was efficient where it could be easily and sufficiently used, generally outside the workroom; otherwise exhaust draught was necessary. It was impossible to lay down figures for efficient local exhaust draught; a linear velocity of 200 feet at the throat of the duct was usually enough to remove 80 per cent. of the dust. Where the source of dust was a rapidly revolving object, as in towing earthenware, there was much more difficulty in collecting all the dust. Water could not be used in the vast majority of factory processes.

He had been much impressed by the humidity in the Crown Mines. If even with the use of water minute particles of dust could still be determined in the atmosphere this was a definite argument against the use of water in confined spaces.

Dr. Badham: In textile mills one sees the use of various methods of securing humidity and among the methods used the value of conveying the air along wet tunnels has been recognised and applied, in fact he thought it is even more efficient than water atomizers. One can appreciate therefore the Rand humidity problem.

He agreed with Dr. Mavrogordato that water is of value at the sites of percussion or fracture—drilling, blasting, and breaking ore. He had no data bearing on the important question of whether the use of water increases infectious conditions.

He found some years ago that the use of the water blast did not reduce the number of particles of dust of micronic size in the air and Drinker of the U.S.A. has also shown this.

He was looking forward to getting useful data as to the parts played by infection and by dust in the production of silicosis. He had a group of sandstone masons who work in open air conditions, and are exposed to 4 milligrams per cubic metre or 400 particles per cubic centimetre of sandstone dust. From a recent investigation the incidence of silicosis among these stone masons did not differ much from that found in quarry men and sandstone miners. He was hopeful that future dissection of these figures may show how far the factor of infection comes into play underground in humid conditions. He came into daily touch with the problems described by Dr. Middleton and could appreciate his remarks. At the present time spray painting and the use of silica dust in paints used in spraying was providing them with a new industrial hazard.

Dr. Moore: The lead-smelting works at Port Pirrie received concentrates (slimes) from Broken Hill. They were shovelled from trucks into dumps in the open air. Hoses had been used to lay the dust, but the main result was to raise clouds of dust, and eventually the dumps were plastered with whitewash.

Dr. Böhme: The German Mines Safety Office has recently offered

a prize for protective measures against injury by stone dust, and many suggestions have been received and examined.

The formation of dry dust in drill holes can be diminished by the well-known methods of wet-drilling, but these methods cannot always be used.

Many methods of precipitating the dust which is diffused from the drill-holes in dry drilling have been proposed. Sprinkling with water has no effect in the case of the dangerous fine dust. On the other hand, previous trials have shown the value of a new method; dust from the drill-holes is caught at the mouth of the hole by newly formed soapsuds. Absorption of dust at the mouth of the drill-hole has also proved successful. In using this method it has been found valuable to draw off the particles of stone separately from the dust.

Amongst the masks which were sent in for examination, one model is especially worth mentioning. In this model the filtering surface is much enlarged by using the form of a sack, and at the same time the resistance to inhalation is greatly reduced.

Good ventilation at the place where drilling is going on is also of great importance.

The Mines Safety Office is continuing its investigations.

Dr. Cunningham: Granite cutters, moulders and grinders usually took twenty-five to thirty years to develop silicosis. These were dry operations, while under wet conditions miners develop the disease in between ten and twelve years. In some of these operations the dust exposure is roughly comparable.

Dr. Kranenburg: In 1912 Dutch stone masons wetted the working place where possible. He personally preferred exhaust draught.

The Chairman: No reference had yet been made to the very fine dust caused by blasting, for which water was ineffective, and ventilation was therefore the important alternative. The single shift system and the limitation of blasting to once in the twenty-four hours gave in conjunction with ventilation an important measure of protection against the risk of inhalation of dust from blasting.

The Miners' Phthisis Prevention Committee in 1916 had shown that 97 or 98 per cent. by weight of the dust could be removed after blasting or in drilling, by means of water, but their standard of dangerous dust was 12 microns, which was now known to be too large. A Joint Committee on Ventilation and Dust definitely reported in 1922 that the water blasts in use were ineffective in laying the fine dust produced by blasting. He did not believe, however, that the possibilities of present methods had been exhausted. The exclusive use of drills with axial water feed had not been made obligatory until 1926, but he agreed with Dr. Mavrogordato as regarded the disadvantages of water.

Mr. Roberts: While the water blast did not necessarily catch fine dust at the time of blasting, it did wet the ground while in process of being broken and thus tended to prevent the formation of dust when the broken ground was subsequently handled, e.g. shovelled into trucks; it also dissolved the noxious gases formed by the blast. Its present use was confined to development ends, and ventilation was arranged so as to replace after blasting all the air between the development face and the next through-ventilation connection.

Dr. Kælsch: He desired to draw attention to a question of fundamental importance. Research undertaken by Teleky (Düsseldorf) in Solingen—the German Sheffield—has revealed the fact that cases

of silicosis occur amongst wet grinders there with considerably greater incidence and of a more severe type than those affecting dry grinders. He thought he was in a position to confirm these observations from his own experience. The causes are to be sought, in fact, in the varying biological reaction of the system to wet dust imprisoned in the finest droplets of water, or to dry dust as the case may be; in the varying reaction, in other words, of the tissues in a damp or dry atmosphere.

Dr. Russell: There was a high incidence of silicosis and tuberculosis in grinding in Connecticut. The abandonment of wet grinding and substitution of dry grinding with an exhaust system had reduced the incidence materially, and much reduced the dust count.

Dr. Loriga: In regard to the use of water in mines he believed it was impossible to pronounce either a favourable or unfavourable verdict. It was essential to study carefully the conditions under which water is applied. In the various operations, which they saw yesterday and which were practically similar in all mines, the first in which the dust problem arises was that of drilling, when done by hand or by machinery utilising the dry method, in which case the amount of dust produced was great. With the use of waterfed drills the quantity of dust produced was reduced to a minimum. He believed there was no atomisation of water or projection of water droplets with dust in suspension, because the drill worked within a tube the wall of which prevented the atomisation of the water and the water left the tube in a liquid state; he considered the use of water not only necessary but indispensable.

The second phase of the work in which much dust was produced was blasting. Here the worker had to be protected not against dust alone, but especially against injurious gases. The workers were always removed to a distance, but the air currents from the blasting might arrive in other parts of the mine. What could be done to prevent this air laden with dust and injurious gases from reaching the parts of the mine in which workers are situated? Dry ventilation would not suffice. Ventilation alone was therefore inadequate except when the plan of the mine permitted of the air current being led off in another direction.

A water curtain at the end of the blasting area was useful to condense the gases and allay the dust, as Mr. Roberts had said, and would prevent poisoning as well as diffusion of dust.

The third very dusty operation was the handling and transport of rock debris and dusty material from the blasting, etc. It was necessary to discuss the utility of projecting water on to the material and walls of the mine and into the atmosphere. He was here in agreement with several of the previous speakers as to the danger of undue humidification of the air. It encountered various obstacles. It was likely to render the temperature conditions more harmful. Humid air, as was well known, was less tolerable than dry air at equal temperature, and humid heat was more likely to cause heat stroke than dry heat. Water spraying would prevent the dust from settling, it was true, but constant humidification of the ground favoured the evolution of certain disease germs, such as those of ankylostomiasis. While therefore it was advisable to use water for dust removal, recourse must also be had to ventilation and to those means of handling and transporting material least likely to raise dust.

In conclusion, it was impossible to express a decided opinion for or against the use of water. At certain moments it was extremely necessary—i.e. in drilling and in the elimination of noxious gases after blasting—but in addition it was essential to have air in large quantities

to provide at the same time for air renewal and removal of the dust and gas laden air.

The Chairman: The reporters might summarise the feeling of the Conference on this point and draw up suggestions for further research.

Dr. Orenstein: Data were of more value than opinions. What data were available on the reduction of small particles at the point of contact? Many localised exhaust draughts were applied in a very amateurish way. The speed of the rotating body and its situation in relation to the hood was important, because the rotating body acts in certain ways as a fan. The dust reduction was much smaller than it should be. He asked whether Dr. Russell could put on record the experience of his Department with two neighbouring sets of mines in Missouri. In one set of mines the lay-out made ventilation poor; in the other it was relatively good. Both worked quite dry, but in the better ventilated mines the incidence of silicosis was very low.

He was much impressed by the reduction of visible dust by water, but he doubted whether water did not do more harm than good.

It was noteworthy that silicosis had become much more virulent and that, except on the Rand, it was contracted only after many years. Was water or something else responsible for the situation on the Rand?

He was much interested by Professor Kettle's investigations into the solubility of silica. Only the nebula was dangerous and water could not wash those particles out of the air or make them settle on the walls. A film which had been made of drilling without water recorded an enormous amount of dust which was invisible to the naked eye.

Dr. Middleton: The Medical Research Council had set up a Committee to investigate pulmonary disease due to dust in industry. The speed of the sandstone wheel and the direction of the air current were very important factors. Wet grindstones in England turned in the direction opposite to their direction for dry grinding in Germany, while in Germany more stones were sometimes found in one room than in Great Britain. In dry grinding very rapid revolution of the grinding wheel needed much more closely localised exhaust draught.

Dr. Russell (in reply to Dr. Orenstein): The Missouri mines were worked out. Similar investigation, however, was proceeding in Oklahoma.

Dr. Kranenburg: There was a danger of heat stroke from radiant heat in tunnels in the Netherlands where there were temperatures of 90° and 124° F. If the men protected their heads there was no danger to health from dry heat.

11. *Possible Rôle in the Prevention of Silicosis of an Admixture of Adulterant Innocuous Dusts with Silica Dusts*

Dr. Mavrogordato: Adulterant dust might help the aggregation of fine particles in the air or might facilitate the exit of dust after inhalation. Phthisis dusts were those which overcame the lung's ability to get rid of them. Coal, limestone and other dusts retarded the accumulation of phthisis dust in the lungs, while other dusts, such as abrasive powders, encouraged silicosis. Metallic mineral dust would modify the behaviour of any free silica which was present in the air at the same time. It must therefore be decided whether artificial dust was to be used to cause aggregation or to assist expulsion from the lungs.

Dr. Kranenburg: A dust of voluminous particles, light in weight, should be introduced as high up as possible in the air, over some distance in the development end, subsequent to blasting. This dust must be such that it remains suspended in the atmosphere long enough to enable it to bring down any fine silica dust which may be in the air. What the nature of this dust should be was another question.

Dr. Böhme: Miners who have worked for several years as rock drillers and who later change over to hewing, contract silicosis in the same way as miners who continue to work as rock-drillers. It is not therefore possible to prove that the inhalation of coal dust has a favourable influence on the development of silicosis.

Dr. Mavrogordato: Any dust which assisted expulsion from the lung must be inhaled at the same time as the silica. In his experience the filling of the lungs with other dusts, whether before or after exposure to silica dust, did not favourably influence the accumulation of silica.

Professor Hall: This was a suitable subject for research. It seemed an extraordinary procedure deliberately to put a second foreign body in the lungs. He asked whether the effect of metallic mineral dust was favourable or not.

Dr. Middleton: Aggregation outside the body was very important and deserved investigation.

Dr. Gardner asked whether the accelerating effect of abrasive powders was chemical or not.

Dr. Fisher: Coal was dusted with shale which often contained 60 per cent. free silica. This was inhaled simultaneously with coal dust, but colliers nevertheless got solidified lungs. He always advised coal owners not to use dust containing free silica.

Dr. Badham: Autopsies and pathological study must clear up the causation of fibrosis.

Professor Kettle: It had been suggested that acute silicosis was due to alkali mixed with silica which aided the solubility of the silica. He did not understand how this could happen without acute damage to the lung tissue. The only case of acute silicosis alleged to be without tuberculosis which he had been able to examine proved to be definitely tuberculous. Judgment must be suspended upon the fixing action of alkali allied with silica dust.

Mr. Roberts: No precautions were taken against dust in the Mysore goldfields, but a man who had been employed there was found to be free from silicosis.

Dr. Orenstein: Professor Haldane's guarded proposal concerning the admixture of dust was intended not necessarily to prevent silicosis, but to prolong the period of useful work and render the disease less disabling. It was claimed that there was no disabling silicosis in Mysore or in Cripple Creek, U.S.A., although dust samples showed a considerable content of free silica in the vein and in the country rock. The U.S. Public Health Service reported, however, that there was considerable silicosis at Cripple Creek, and he was not convinced that the situation was not similar in Mysore.

Dr. Kelsch: Observations made in regard to workers on shell limestone, who had between times also worked on sandstone, revealed the fact that marked silicosis occurred, that is to say that the effect of the silica was predominant. It was not possible to establish a *clearly*

marked retarding effect due to the limestone dust. In any case the question of the prophylactic application of relatively harmless dust during work in an atmosphere laden with silica dust is one which must be approached with the utmost caution.

Dr. Gardner: Guinea-pigs dusted with marble dust showed no damage; quartz dust given later gave the same results as when no marble dust had been given,

Dr. Kranenburg: In the Netherlands the simultaneous inhalation of sand tone and limestone gave apparent protection.

Dr. Middleton: There was no evidence of silicosis from limestone alone.

Dr. George: At Broken Hill the ore is of extremely complex composition. It contains only 14 per cent. of free silica on an average, the other constituents being chiefly manganese silicate, lead sulphide and zinc sulphide. The country rock contains no free silica. In spite of this we find silicosis, and this, as you have seen, shows exactly the same radiographic picture as your silicosis on the Rand.

TABLE OF MINERALOGICAL COMPOSITION OF MINE ORES
(PERCENTAGES)

	North	Junct- ion North	Brit- ish	Block 14	B. H. P.	Block 10	Central	South	Zinc
Silica free	15.16	1.62	15.20	11.74	12.00	7.01	12.24	17.73	17.40
Silicates	39.57	66.41	48.77	47.43	46.28	57.25	37.81	24.37	33.28
Galena	17.69	14.34	13.69	16.88	14.64	12.76	16.87	18.38	18.21
Blende	22.03	14.05	19.64	21.48	23.74	20.07	29.20	26.95	19.92
Sulphides of copper	0.22	0.25	0.22	0.22	0.19	0.22	0.22	0.21	0.27
Sulphides of arsenic and anti- mony	0.09	0.07	0.10	—	0.07	0.09	—	{ 0.06 0.04	0.12
Calcium carbonate	5.24	3.02	1.70	2.57	2.73	2.60	3.79	11.15	10.80
Calcium fluoride	—	0.20	0.19	—	—	—	0.22	0.39	—
Pyrite or marcasite	—	0.04	0.21	—	0.15	—	0.30	0.32	—
Pyrrhotite	—	—	0.28	—	0.20	—	—	0.40	—
	100.00	100.00	100.00	100.32	100.00	100.00	100.65	100.00	100.00

An examination of the figures in the above table gives the following information. The figures in the table are arranged in the order of the mines from north to south.

The percentage of free silica in the average ore samples varies from 1.62 to 17.73. The ore is thus nowhere rich in silica, and in one mine the percentage is very low indeed.

The samples of country rock were obtained partly from the walls of stopes and partly from development drives. A number of samples were taken in each mine, the places sampled being chosen to give, in

the opinion of the mine managers, fair specimens of the general type of country rock encountered in the different mines.

Complete analyses were made of these samples. The results are given in the following table:

COMPOSITION OF COUNTRY ROCK FROM DEVELOPMENT DRIVES AND
HANGING AND FOOT-WALL OF BROKEN HILL LODE

	Junction North	British	Broken Hill Pty	Block 10	South	Zinc Corporation
Silica, SiO_2	63.90	64.34	59.64	59.10	64.66	60.26
Alumina, Al_2O_3	15.22	15.58	16.82	18.20	14.88	17.97
Ferrous oxide, FeO	7.43	5.74	7.25	7.25	5.98	7.25
Ferric oxide, Fe_2O_3	0.15	0.20	0.15	0.40	0.10	0.08
Titanium dioxide TiO_2	2.34	0.98	1.96	2.50	2.46	2.10
Manganous oxide, MnO	2.33	2.46	3.25	2.40	2.56	1.74
Lime, CaO	1.30	2.60	0.85	0.66	1.32	1.01
Magnesia, MgO	1.09	0.76	0.73	1.25	0.84	1.44
Potash, K_2O	4.04	2.31	3.56	4.76	3.77	4.78
Soda, Na_2O	1.08	1.14	1.03	1.13	1.60	0.95
Sulphur, S	—	0.54	—	—	—	—
Water hygroscopic	0.20	—	0.35	0.15	0.06	0.20
Water combined	1.47	2.14	3.65	2.27	1.42	2.29
	100.55	99.66	99.24	100.07	100.00	100.07

These figures show a striking contrast to those obtained for the various ore samples. The composition of the ore samples from different parts of the lode showed very considerable variations. The composition of the country rock from different parts of the lode, on the other hand, shows a surprising uniformity. About 80 per cent. of this rock, in all the samples, is made up of aluminium and iron silicate. The bulk of the remainder is made up of silicates of potash and manganese. The only acid radical present in any quantity is silica.

Dr. Russell: Workers in the U.S.A. had lost cases in the Compensation Courts because the question of other dusts had been introduced. In U.S.A. silicosis seemed to develop in direct proportion to the silica content and the dust concentration. At Cripple Creek miners who developed silicosis left as soon as the altitude began to trouble them. Silicosis undoubtedly developed there.

Dr. Cunningham described two cases: the first a stone-cutter, who was employed for fifteen years on sandstone and then fifteen years on marble. The post-mortem showed the ash of the lung to contain 5 per cent. silica. The second, a miner who developed silicosis, left the industry and eight years later developed tuberculosis. The ash of the lung contained 45 per cent. silica.

While there is some evidence that with cessation of exposure to silica, in the absence of tuberculosis, much silica finally leaves the lung, marble dust may expedite this.

There have come to one's attention some cases in which silicosis developed in grinders who had worked for a short time on sandstone

and later for many years on artificial grindstone. Some miners with ten to twenty years' exposure to dust very low in free silica developed silicosis rapidly when they moved to an area with much higher silica exposure.

Some interest attaches to one case of pneumoconiosis in a worker with dust exposure limited to nine years' milling talc (magnesium silicate). The industry as a whole has not yet been investigated.

Dr. Russell: Italian workers who had been exposed to Carrara marble dust developed silicosis when working on granite in the U.S.A. as fast as the Scottish workers employed there.

Dr. Badham: Other dusts than silica could produce fibrosis. Cement workers in the United States had been known to suffer from diffused generalised fibrosis. Fibroses resulting from dusts containing little or no free silica should be put on record. Asbestos, talc, ashes, and certain silicates produced fibrosis.

Dr. Mavrogordato: Professor Haldane had added metallic dust to silica. Silica alone was not intense but acted by accumulation; metallic dusts gave inflammation; cobalt and manganese gave the more intense immediate reaction.

III. Dust Trapping and Dust Masks

Dr. Fisher: In dealing with this subject he would, of course, only speak of his own experience. The boring in and blasting of rock in a coal mine is an occasional and not a continuous process. The danger to the workers' health due to dust inhalation having been established, attention was focussed on ways and means of preventing this inhalation from taking place. His only remark about water was that a water-spray, as distinct from water passing down the drill is ineffective and it must be remembered that some dusts have a greater affinity for water than others. Water could not always be made use of, so dust traps were tried—there are several of them either in use or being tried out; two have definitely been approved of as efficient for their purpose.

They all work on the same principle, namely a jet of compressed air coming from the air supply to the drill draws off the dust and deposits it in a bag-container, made of a specially selected flannel, which allows the air to pass but retains the dust.

The weight of the trap shown is 26 lb. only, including the upright support which is telescopic, and which grips the roof and floor in virtue of a spring. It all fits into a long box; bags may be protected by being in some kind of cage.

Dust traps undergo a severe test—they are taken underground and to the actual face being worked. They are fixed up by an employee of the mine and numerous dust slides are taken while the drill is in operation, on many occasions Kotze or Zeiss and Owen's konimeters being used simultaneously.

The standard adopted was an indication of safety, being 300 particles per cubic centimetres with a Kotze konimeter, the slide being untreated in any way.

Whether ultra-microscopic particles pass through the back is of course a question he could not answer.

He would like to remind them that the Various Industries (Silicosis) Scheme, 1928—in force 1 February 1929—applies to coal mines and repeat what he mentioned yesterday, that the demand for these compact, and as far as he could test, effective dust traps exceeds the rate of supply.

He should think that with the serious problem of humidity and tuberculosis the use of dust traps could be reconsidered on the Rand.

As far as masks are concerned workmen have for many years tried to improvise a barrier of some kind that would prevent the dust from entering their lungs. These were practically useless—invariably the nose was exposed and most of the breathing done by the nose. He did not agree with the sweeping condemnation of all masks. There are processes in which the exposure risk is slight—men working on stone in the open, for instance—where a mask would so render the amount of dust breathed in negligible, and where it is not possible to draw off dust by exhaust. A mask made by Messrs. Siebe Gorman is being tried at a quarry where a man worked who died of silicosis. The mask is probably known to you all; it consists of a light rubber face piece, inhaled air has to pass through numerous wafer-like cotton filters, the expired air passing out by means of mica valves. He had worn one in an intense concentration of dust made by throwing it on his face and the back layers of the filters have, as far as ordinary observation goes—which he admitted is not far—been perfectly clean.

A mask which will mean the man being connected by a length of tubing to a main air supply will be limited in its application.

Dr. Cunningham described a mask of positive pressure type consisting of cloth covering the face, to which air is delivered, sufficient to keep the mask ballooned. The air escapes from the sides and front due to an air pressure inside, high enough to prevent ingress of air containing dust or fumes.

Dust counts in samples of air from inside were very low.

Granite cutters are wearing the mask without complaint except for the low temperature of air delivered during the first fifteen minutes of operation in winter time.

The mask is comfortable, inexpensive and effective, but limited in use to those who are relatively stationary at their work.

Dr. Böhme: Masks were objectionable because of the resistance to respiratory air. Masks were, therefore, being introduced presenting a large surface of small mesh which gave a higher resistance to dust and a smaller resistance to air.

IV. Adhesion of Dust by Hygroscopic Methods

The Chairman: These methods had shown no advantage over water, and since at that time the disadvantages of water had not been fully realised no further action was taken.

Dr. Orenstein suggested that Professor Hall might consider research into the question of masks on a labyrinth system without a filtering medium.

The Chairman read the following letter received from Mr. Ussher:

USSHER AND HALL INVENTIONS, PTY. LTD.

P.O. Box 3450, Johannesburg, 14 August 1930.

W. Gemmill, Esq.,
Chamber of Mines, Johannesburg.

DEAR MR. GEMMILL,

Re Silicosis.

As you may remember, I spent over a year underground in research *re* dust laying.

When a solution of crude treacle was used during blasting, no dust could be blown off the drive walls, etc., either a few minutes after blasting, or next morning.

When drives were sprayed twice within five or six weeks, the rock faces remained wet and sticky for over a year in places where water dried up in less than an hour.

Dust laid once by treacle was apparently laid for years—if not permanently. The cost of the process was only a fraction of the cost of dust-laying with water.

The then Government Mining Engineer reported to Parliament that “water would do all that treacle did, but that the effect of treacle was apparently permanent”.

In a second report he claimed, as a proof of the efficiency of water, that in a series of thirteen tests on the City Deep, the average dust content of the samples taken was only 1.6 milligrams, but this result was almost entirely due to treacle and not water.

I was informed that the Medical Officer on the City Deep reported that the death rate for the year had gone down from the highest on the Rand to the lowest, and that the number of cases of pneumonia among the natives had decreased by 50 per cent.

After considering all the reports of the experts and their tests, Sir Lionel Phillips decided to introduce my process in all mines controlled by the Central Mining and Rand Mines Corporations.

The Chamber of Mines opposed my patent, but Mr. Justice Wessels decided, after hearing the evidence, that I had proved the efficiency and novelty of my process, and ordered the patent to be issued.

The then Government Mining Engineer, having insisted that the Regulations as to the use of water must be carried out strictly, it was useless to proceed to lay dust with treacle, only to wash it all away next day.

Where treacle is used, no water-sprays are necessary, and no miner need get wet or work in over humid atmosphere from that cause. The relationship of the quantity of water used to eliminate the dust is roughly 500 gallons of water only, as against 1 gallon of treacle solution.

Even Mr. Stuart Martin, who was at first rather hostile to the process, afterwards said that in his opinion the use of my process would become more essential and inevitable as our mines increased in depth.

My patent has expired and I have no financial interest in the use of my process, but I would be glad of an opportunity of giving the results of my former labours to the Conference, and of answering any questions they care to ask me.

Yours faithfully

(Sgd.) *Lancelot Ussher.*

Dr. Orenstein: Treacle and other fluids had already been tried. The fall in the incidence of pneumonia was universal all over the reef, but it had not been sustained. A hygroscopic substance retained the large dust but minute dust did not settle on treacle, and even with the use of water treacle would be washed away.

The Chairman: The advantage of treacle was that it might reduce the use of water and thus reduce the humidity.

Mr. Roberts: The Mining Regulations required the use of water in quantity, and it was, therefore, impossible to experiment with hygroscopic substances.

Dr. Orenstein: The reporters should bear in mind the possibility of experimenting with hygroscopic and other sticky substances.

Mr. Phelan: The discussion had seemed to show that the dangerous dust might consist of particles so small that they were invisible. He had considerable diffidence in venturing anything in the nature of a suggestion to experts of such recognised competence as the members of the Conference, but the methods of counting dust particles to which reference had been made were apparently not applicable to the counting of particles of this minute size. He wondered, therefore, whether it would not be possible to adopt for the purposes of a dust count the experimental method used by J. J. Thomson for the counting of the number of ions in an ionised gas. The method followed was a simple one. A known volume of the gas was enclosed in a bell jar and ionised. The gas in question was given a degree of humidity and was ionised for example by exposure to X-rays. A sudden reduction of pressure caused the formation of a fog, the water condensing on the ionised particles. The fog was allowed to settle in the bell jar and the rate of fall of its upper surface was observed.

A formula discovered by Stokes established a relationship between the size of the drops and the rate of their settling and the diameter of the drops could, therefore, be calculated. If the total amount of condensation were measured, i.e. the volume of water condensed, the number of drops could then be calculated. It seemed to him that this method could be applied to air containing minute particles around which the water would condense instead of round the ions.

Dr. Orenstein: He believed that the British General Electric Company were experimenting with a method of this kind but he had been unable, as yet, to obtain their apparatus. Photographic and photo electric methods of dust determination were being tried.

(The Conference adjourned at 12.50 p.m.)

FOURTH SITTING

Monday, 18 August 1930, 9.30 a.m.

Chairman: DR. L. G. IRVINE

AETIOLOGY AND PATHOLOGY OF SILICOSIS

Dr. Mavrogordato: Some dusts acted favourably and others unfavourably on silica. Very little was known about their particular behaviour. Any inert, relatively insoluble dust which got into the lungs in sufficient quantity favoured fibrotic conditions; silica was only relatively insoluble.

A dusty lung was a wounded lung and its cleanliness or dirtiness, in the surgical sense, was of great importance. The terminal bronchioles and vestibules were the first places attacked and if they were damaged

the dust which had once passed the terminal bronchiole stayed where it was.

Observation had shown that the smaller incidence of silicosis among natives, as compared with Europeans was due to intermittent employment; natives who were employed continuously developed silicosis more rapidly than Europeans. The sputum of broncho-pneumonia cases indicated that a good deal of dust and pigment might be got rid of during broncho-pneumonia. The natives braved exposure to silica dust and subsequent rest enabled the bronchiole epithelium to recover and eliminate much of the dust by the same way in which it entered the lung.

As regarded the infective element, practically every fatal case in the Witwatersrand fields died with tuberculosis. But tuberculosis was not the only infective factor. Experimentally he had had no difficulty in producing chronic unresolving pneumonia by intra-tracheal injection of an animal that had previously been exposed to silica dust. There must be a definite infective element if simple silicosis progresses. One problem was to find out whether silica rock, like certain other inorganic material, set up an allergic state in the tissues and whether tissues which were allergic to silica were also allergic to tubercle. The relation between certain dusts and a subsequent tuberculosis might be explained if the dust which affected the same tissue as the tubercle toxin not only rendered it allergic to itself, but also allergic to tubercle without any secondary immunity to tubercle. He asked what intractable silicates would do in the absence of free silica and what certain other dusts would do which had been retained in the lungs of the silicotic, in the absence of silica?

Professor Kettle: Nowhere was there such an abundance of pathological material as in Johannesburg and he hoped to learn a great deal during the Conference. He was particularly anxious to hear more about the dry bronchiolitis which was apparently an early manifestation of silicosis. This condition might influence the deposit of silica in the lungs in several ways. Because of the destruction of the bronchial epithelium there might be a deficiency of mucus in the air passage, and dust might therefore pass more easily into the pulmonary tissue; or, because of the breaking down of a cell barrier dust-containing phagocytes might pass more easily into the peribronchial and pulmonary tissue; or the natural elimination of the dust from the lung might be interfered with. He would be glad to know in which direction this lesion was regarded as being particularly important.

He also hoped to have an opportunity of seeing more of the available material, and learning more about the histology of infective, especially, tuberculo-silicosis.

The suggestion had been made that silica might damage the tubercle bacillus as it did the tissues. This was to him a new conception because all his observations lead him to believe that silica aided the growth of the bacillus. This certainly appeared to be so *in vivo*, and he had a certain amount of evidence to show that silica added to culture media aided the growth of the organism *in vitro*.

How the silica acted in influencing the growth of the tubercle bacillus was still unknown, but it appeared likely that it was a metabolic phenomenon. He thought it was generally agreed that the action was a chemical one in that the silica undoubtedly was soluble in the tissues. This could be demonstrated by placing a suspension of silica in sealed collodion capsules embedded in the subcutaneous

tissues of rabbits. In two or three weeks the silica was dissolved and produced its effects on the neighbouring tissues.

He was also anxious to hear the opinion of the Conference on the animal of choice for experimental work on pneumoconiosis. From the anatomical peculiarity of its lungs, the guinea pig, which was so commonly used, appeared to him to be definitely unsuitable.

Dr. Gardner: Commenting on Dr. Mavrogordato's statement that ingested silica particles, in contradistinction to other types of ingested material, exert a preservative effect upon the phagocytes, he would like to call attention to the following observations.

Haythorn produced a limited degree of fibrous reaction in the ear of a rabbit by the injection of large amounts of coal dust. After the lapse of several months the injected particles are found in elongated compressed cells which resemble fibroblasts. If however an oedema is produced by constriction of the base of the ear or by immersion in very hot water the fibroblasts separate and the phagocytes again assume an ovoid or spherical form. This experiment demonstrates that the dust is still retained by monocytes and that it is not transferred to fibroblasts.

In the Saranac laboratory a variety of dusts, silica in various forms, granite, carborundum, aloxite, emery, etc., have been injected into the peritoneal cavity of guinea-pigs. At various intervals up to nine months thereafter samples of the peritoneal fluid when withdrawn still exhibit free phagocytes containing large quantities of ingested dust particles. If any of these cells are stained supravitaly, with neutral red, a delicate indicator of cell injury, there is no evidence that they have been damaged. Furthermore there is no evidence of disintegrated cellular debris in the *exudate*. However there is no way of marking the original host cells so that one cannot be sure that the dust particles may not have been taken up by a successive series of phagocytes. Nevertheless, as far as can be demonstrated, he has produced no evidence to show that ingested silica particles are more protective than any other type of dust. This line of investigation is still being pursued.

Professor Kettle had mentioned effects produced by silica dust upon the tubercle bacillus. He has been conducting a series of experiments to determine whether the development of a silicosis will alter the virulence of the bacilli of a superimposed tuberculous infection. For this purpose he has infected guinea-pigs by the inhalation of tubercle bacilli of an attenuated strain known in the laboratory as R. 1. In normal guinea-pigs such infection regularly produced isolated subpleural tubercles in the lung together with a massive involvement in the tracheobronchial lymph nodes. Macroscopic tubercle in the other viscera practically never occurs. Multiplication of the bacilli in the pulmonary tubercles brings about caseation after some four to six weeks. The bacilli then die off and healing of the focus by resolution within a period of eighteen months to two years is the result. The tubercle in the mediastinal lymph nodes likewise heals although there is often some residual fibrosis. Death from this infection almost never occurs.

If an animal, infected in this manner, be subjected to eight hours' daily inhalation of quartz dust over a period of two or more years it can be shown that after the elapse of three to five months the bacilli in the primary foci of infection do not die but apparently quite suddenly take on a new capacity for growth. The lesion spreads; at first locally and latterly by ulceration into the bronchi the organisms are distributed into all parts of the lung. The result is a generalised pulmonary tuberculosis, chronic in its course but usually fatal in outcome. The

pathological lesions are quite comparable to chronic tuberculosis in man, often with large trabeculated cavities. Macroscopic tuberculosis of the liver, spleen, and abdominal lymph nodes is the rule.

In order to determine if possible the cause of this renewed growth capacity of the attenuated tubercle bacilli in the silicotic lung, animals have been killed at intervals of one month after infection. Under proper aseptic precautions, portions of the lung, spleen and lymph nodes have been removed for culture and subinoculation into other animals. This experiment is not yet completed but in the majority of the sub-inoculated guinea-pigs the infection produced has been comparable to that produced by subcutaneous inoculation of cultures of the R. 1. strain, i.e. a non-progressive infection confined to the lymph nodes adjacent to the site of inoculation. In a certain number of sub-inoculated guinea-pigs the spleens have also been involved but such an effect can be produced by very large doses of this strain of tubercle bacilli. At the present time it would appear that the silicotic process in the lungs of the original series of animals has not increased the virulence of the bacilli used for infection but that a temporary alteration in environment has been produced which favours the continued multiplication of the organisms. Removed from this altered environment the bacilli again revert to their original state.

From the cultures obtained from the silicotic lung a study is in progress to learn whether bacterial dissociation is favoured by silicosis. No definite results are as yet forthcoming.

In vitro, bacteriological studies are in progress to discover whether the addition of silica in various forms to various culture media will favour the growth of the tubercle bacillus. Like Professor Kettle he had observed a definite reduction in the initial lag period after transplants have been made.

Professor Kettle had asked for an expression of opinion as to the most suitable animal for the experimental study of pneumoconiosis. At the Saranac Laboratory he had used guinea-pigs because of their cheapness and because large numbers may be kept in a relatively small space. As he would show in the lantern slides it is possible to produce quite typical silicotic lesions in this animal within a period of two years. However the rabbit has certain advantages. Its lung normally contains large amounts of tonsil-like lymphoid tissue located directly beneath the epithelium of the bronchi and bronchioles. If serial sections be cut through these areas it will be found that the general cuboidal or columnar epithelium lining the bronchi and bronchioles becomes attenuated and flattened over the centres of the lymphoid nodes. Such an arrangement permits a direct and rapid passage of dust-laden phagocytes from the air passage into the lymphoid tissue. As a consequence large quantities of dust soon collect in these regions which ultimately brings about the formation of silicotic nodules much more rapidly than in the guinea-pig.

He had unreported experiments on monkeys, two baboons and six rhesus monkeys exposed to the inhalation of granite dust. While far advanced fibrosis was produced after periods of one to two years, in every case accidental death from spontaneous tuberculous infection introduced a disturbing complication.

Before definitely deciding upon the most favourable animal for use in pneumoconiosis experiments he thought that other non-rodent mammals (cats, dogs, sheep and swine) less susceptible to spontaneous infection with the tubercle bacillus should be tried.

Dr. Strachan: As the result of observations made with Dr. Simson on the sputa of asbestos workers, they had come to the conclusion that the phagocytes had the power of elaborating a substance or substances, usually iron-containing, with which ingested particulate matter was isolated in the cell, and so the cell was protected against the action of this particulate matter.

In time the ingested particulate matter might be dissolved as could be demonstrated in the case of "asbestosis bodies".

Professor Loriga: In his opinion there was no material difference between the reaction of cells to various dusts; it belonged to the category of reaction by irritation. He did not believe in the chemical theory and considered that silicosis was not a different pathological entity to other pneumoconioses.

Sir Spencer Lister suggested the use of B.C.G. culture in experimental work on silicosis developing in dusted animals.

Dr. Gardner: Such experiments were being made in his laboratory.

Dr. Strachan (in introducing the discussion on the Pathology of Silicosis): In Dr. Simson's and Dr. Strachan's Report, they accepted as correct Miller's work on the lymphoid tissue of the lung, but subsequent investigations have led them to modify that acceptance.

They have examined a relatively large series of cases of status lymphaticus and have noted the distribution of the lymphoid tissue in the lung. There were no complicating lung lesions to obscure the picture. A series of photographs demonstrate that the lymphoid tissue in the lung is essentially and functionally related to the air-passages and to their terminations. The photographs show the absence of the tissue in the blood-vessel walls and also that where lymphoid aggregates occur in contact with pleura or trabecular the essential relationship is to the adjacent air-spaces. All the lymphoid tissue in the lung is functionally related to the air-passages and to their terminations. Any other apparent relationship is only accidental.

In cases other than those of status lymphaticus lymphoid tissue is present in the same situations though to a less degree. Any condition, such as silicosis, which may stimulate hyperplasia of lymphoid tissue will make it more manifest and they have frequently seen this in cases of silicosis.

The importance of the lymphoid tissue can be demonstrated from the atlas and from the series of large photographs elaborating the atlas. The dust cells accumulate in and around the sites where normally lymphoid tissue is present, but only round the smaller bronchi and the bronchioles and their terminations. They have never seen a true silicotic lesion in relation to the large cartilaginous bronchi.

There occurs a cellular fibroblastic reaction which terminates in a dense ball of hyaline fibrous tissue.

These lesions show first in the tracheo-bronchial glands, then in the pleural and finally in the lung substance.

There is a definite entity—simple silicosis—i.e., silicosis uncomplicated by infection, particularly tuberculous infection. This condition can progress to a considerable degree—even to massive fibrosis and in these cases biological tests for the presence of tubercle bacilli have proved negative.

In contra-distinction to simple silicosis there is a type in which infection plays a part and to which the term "infective silicosis" can

be applied. The chief type of infection is tuberculosis and then one may use the term tuberculo-silicosis.

The nodules are more exuberant in their fibrous tissue and more rapid in growth and central necrosis occurs early.

Degenerative changes of fatty nature sometimes followed by calcification are seen in simple silicosis but only very rarely is there necrosis. Necrosis may be taken as an indication of infection.

In the progress of the disease, in the simple type structures are displaced whereas in the infective type they are infiltrated and destroyed.

His experience during the last six years suggests that remote effects involving heart, liver and kidneys were not common in simple silicosis, and his impression is that such lesions are not any commoner in miners than in the general population; in infective silicosis where there is massive destruction and fibrosis associated with emphysema there may be however right-sided cardiac failure as a terminal phenomenon.

The picture of the "classical type" of miners' phthisis is in his opinion that of an infective silicosis.

Dr. Simson: Macroscopically, it is often difficult to distinguish between simple silicosis of massive type and tuberculo-silicosis.

Microscopically, some cases of tuberculo-silicosis show evidence of active tuberculosis in the lung tissue intercalated between the adjacent silicotic nodules. In other cases the usual signs of tuberculosis as evidenced by small round mononuclear cells, endotheloid cells, and giant cells are absent. Definite necrosis in the nodules, however, is present. In such cases, although tuberculosis might be suspected from the microscopical evidence, a biological test is the only reliable means of diagnosis.

He would add that in tuberculo-silicosis the tubercle bacillus is living on a medium mainly composed of fibrous tissue, and that this probably accounts for the slowly progressive nature of the lesion. In the lung tissue outside the limits of the silicotic nodules the disease advances much more rapidly as here the bacillus is living on a good medium which includes blood plasma.

The Chairman: It was a well-known fact that in tuberculo-silicosis infection was not readily communicable to members of the patient's family.

Dr. George asked the Chairman how he would decide whether death was due to or accelerated by silicosis and/or tuberculosis in the following cases:

- (1) When the cause of death is lobar pneumonia and simple silicosis is present.
- (2) When the cause of death is broncho-pneumonia and silicosis is present.
- (3) Death due to heart failure, with simple silicosis present.

The Chairman: He agreed with Dr. Strachan that the classical silicosis of former days had an infective element. Many of the miners then employed were Cornishmen who went home when their health began to fail and the majority of these were shown by post mortem to have died of tuberculosis. In miners who broke down suddenly and died, often within three months, there was rapid necrotic silicosis. But on the other hand many at that time died of heart failure with cyanosis and ascites and with no clinical signs of tuberculosis.

At the present day there was earlier manifestation of the tuberculous element, but there was enough normal lung left to place the patient in a better condition to control a tuberculous infection. The cases nowadays lived longer.

In reply to Dr. George he said that a certain rough justice had to be adopted. If a man had a well-marked silicosis and died of a primary cardiac lesion the Bureau would allow that silicosis had been contributory and the same applied to acute respiratory diseases.

Dr. Truter: At the time of the influenza epidemic of 1918 it had been impossible to differentiate between the case mortality of a man with a slight degree of silicosis and of a man with no silicosis at all.

Dr. Pringle: At Springkell Sanatorium and Wedge Farm Sanatorium it had been definitely concluded that fibrotic obstruction was an element in the cause of death; but these cases were not very numerous. The more impressive cases were those of simple silicosis suddenly breaking down. Post mortems had shown that the cause of the rapid breakdown was always the breaking down of a tuberculous infection.

Dr. Watt: In the influenza epidemic some men affected with a slight degree of fibrosis died, while others did better than the general population. He could not say whether the latter had acquired immunity in a previous epidemic.

Dr. Strachan: It might be worth while for the reporters to draw up a definition of silicosis and infective silicosis. A fresh report on the incidence of cancer of the lung should also be asked for.

Professor Kettle asked Dr. Simson for information on his experiments in inoculation, because it was well-known that tuberculosis in the guinea-pig was very variable in its manifestations.

Professor Böhme: There were a considerable number of cases of advanced silicosis in Germany which died of heart failure, with dilatation and failure of the right heart. These symptoms are due to fibrosis and emphysema of the lungs. The combination of silicosis and cancer was found only in the Schneeberger district, and was believed to be due to the inhalation of radioactive dusts.

Dr. Orenstein asked whether there was any experimental or pathological evidence to determine whether dilatation of the heart, and particularly liability to respiratory disease, was associated with pneumoconiosis.

Dr. Middleton referred Dr. Orenstein to the mortality data in the *Decennial Supplement* for 1921-1923 of the Registrar-General in Great Britain.

Dr. Kranenburg asked whether a person with a negative von Pirquet reaction was allowed to be employed in the mines. If so, was he treated with the Calmette vaccine?

The Chairman: It was impossible to make von Pirquet tests on the large number of men examined.

Sir Spencer Lister: Examinations carried out at the Institute for the past three years showed that 60 to 70 per cent. of the natives gave a positive von Pirquet reaction. The tests had been followed up in many cases and post mortems correlated with the results of the von Pirquet tests. It was found that a much larger percentage of deaths occurred amongst those natives who originally showed a positive von Pirquet reaction than amongst those whose reaction was originally negative. The Institute was at present experimenting with B.C.G. vaccine as a prophylactic in adults.

(The Conference adjourned at 12.50 p.m.)

FIFTH SITTING

Monday, 18 August 1930, 2.30 p.m.

Chairman: DR. L. G. IRVINE

Profesor E. H. Kettle showed microscopic preparations illustrating the modification of an artificial miliary tuberculosis by silica.

In the first series of experiments rabbits received four to eight intra-venous doses of amorphous silica or of mine dust over a period of ten to fourteen days. They then received an intra-venous inoculation of living tubercle bacilli as did a control series of animals. In the animals prepared with silica, miliary tubercles were much more numerous than in the control animals, and bacilli were much more numerous in the lesions. The limitations of the experiment were discussed.

In the second series interstitial lesions were produced in mice by silica and other agents, particularly calcium. The mice then received tubercle bacilli intravenously. Tubercle bacilli are found in the silica lesions in much greater numbers than in the lesions produced by calcium.

A further series of sections illustrated preliminary experiments in the production of pulmonary lesions by the intra-tracheal injection of different forms of silica.

GENERAL TECHNIQUE OF DUST EXPOSURES

Dr. Gardner: Guinea pigs and rabbits are kept in cages along the walls of a room 6 by 8 feet in diameter and 8 feet high, provided with two windows 3 by 5 feet in diameter. A cloud of dust is maintained in the atmosphere of the room by apparatus located in an alcove, 6 feet from the nearest animal. This apparatus consists of a horizontal drum $2\frac{1}{2}$ feet long and $1\frac{1}{2}$ feet in diameter, placed 8 inches above the floor. Its top is open and in it rotates a metal paddle whose shaft projects from the end of the drum and then through the partition into an adjacent room in which is located an electric motor. By properly selected pulleys and counter shafting, the speed is so regulated that the desired dust concentration can be maintained. Ventilation is secured by opening one of the windows an inch or more from the top. The animals are kept, generally in pairs in 12 by 12 by 12-inch wire cages set on racks at the back and sides of the room. While it will be shown that the dust concentration progressively decreases from the floor to the ceiling, this factor is compensated by frequently changing the position of the cages on the racks.

As measured by the operation of the motor, the daily period of exposure is eight hours, but since the animals remain in this room during the night, the actual exposure is much longer, a considerable time being required for all the dust in the atmosphere to settle. In warm weather the period is somewhat shortened by opening the windows when the motor is stopped during the evening.

The dust concentrations employed for this work have approximated those of the worst industrial conditions. For asbestos dust we have maintained an average concentration of 46.5 million particles, 10 to 0.5 micra in diameter per cubic foot of air. Recently this concentration has been increased approximately ten times. In granite dust experimentation the concentration was much higher (288 million particles

per cubic foot). The figures for quartz and carborundum dust concentrations he neglected to bring with him but if his memory had not failed they are of the order of those mentioned for granite dust.

Marble Dust

The first experiments were made with marble dust from Proctor, Vermont. This substance contains less than 1 per cent. free silica. Exposure of normal guinea-pigs for periods as long as two years produced practically no changes other than a hyperplasia of the lymphoid tissues in the periphery of the lung, together with some dilatation of the deep lymphatic vessels.

Guinea pigs infected by inhalation of the low irritant strain of tubercle bacillus R. 1 did not develop a generalised pulmonary tuberculosis. But there was, however, a well-marked failure of the normal process of resolution in many of the tubercles associated with the development of calcification of their caseous centres. In the light of subsequent experiences with other dust this calcification is now ascribed to the fact that resolution was retarded and caseous matter was retained in the lung for a sufficient period for calcareous deposit to occur. A similar finding in carborundum dusted animals strongly suggests that the excess of calcium supplied by the inhaled marble was not the determining factor.

Granite Dust

The action of this dust upon the lung tissues is too slow to exert its maximum effects during the exposure periods possible in guinea-pigs. Inhaled particles are ingested by alveolar phagocytes which tend to accumulate in the air spaces along the terminal bronchioles and alveolar ducts. No evidence of fibrosis has been observed in the walls of such passages but only a slight chronic inflammatory infiltration. Considerable amounts of dust are carried by phagocytes into the lymphatic system. A certain number of dust cells can be found in hyperplastic nodules of lymphoid tissue in the periphery of the lung but no evidence of fibrosis is discoverable. Much more dust is accumulated in the tracheo-bronchial lymph nodes and here a true fibrosis of the silicotic type has been observed in animals exposed for two or more years. The lymph vessels in the lung are widely dilated from the hilum to the pleura but evidence of thrombi of dust cells is lacking. The cause of this dilatation was temporarily at least ascribed to the production of an obstructive fibrosis in the lymph nodes of the mediastinum.

Carborundum Dust

Inhalation produces in the normal guinea-pig a reaction similar to that of granite. Great masses of dust laden phagocytes are discovered in the terminal bronchioles and their alveoli. No nodular fibrosis has developed within a period of four years. In the tracheo-bronchial lymph nodes where much greater amounts of dust have collected there is true fibrosis which tends to be somewhat nodular in type. More marked than in any other dust studied are the changes in the connective tissues of the bronchi and large blood vessels. They are definitely thickened by the formation of extremely vascular granulation tissue which is infiltrated with numbers of lymphoid and phagocytic cells. Through this tissue course many greatly dilated lymph vessels.

Quartz Dust

The phagocytes which have ingested quartz particles exhibit a much greater degree of activity than those containing any other type of dust yet investigated. Instead of remaining for long periods in masses along the terminal bronchioles as in the case of granite and carborundum dust the quartz-filled cells which are present in great numbers actively migrate along the walls of the alveoli, atria, etc., until they reach the small terminal collections of lymphoid tissue. They surround these structures and rapidly invade them. The lymphoid elements at first undergo hyperplasia, but very shortly the newly formed lymphoid cells appear to decrease in number and are finally completely overshadowed by the mass of large clear-stained dust containing phagocytes. This collection of cells is then surrounded by a layer of cuboidal epithelium which sharply demarcates it from the surrounding tissues. Thus far no fibrosis is present but only a nodule perhaps similar to that which Dr. Mavrogordato has called a "pseudotubercle". In the centre of this area where innumerable particles of quartz have been concentrated fibroblasts begin to make their appearance. Their source has not been determined; they may have come from the original reticulum of the lymphoid nodule. A cellular fibrosis gradually replaces the entire nodule of phagocytes. Degenerative changes make their appearance at its centre and later the structure assumes the characteristic hyaline appearance of the fully formed silicotic nodule seen in the human being. Presumably the majority of the larger lesions form in the manner described about nodules of peripheral lymphoid nodules. Some may also develop within the alveolar septa or at least these become greatly thickened and often encroach upon and ultimately close the adjacent air spaces.

More rapidly than with other dusts, quartz particles are carried into the tracheo-bronchial lymph nodes and there provoke nodular fibrosis even earlier than occurs in the lungs.

The lung of the rabbit presents peculiar anatomical conditions which favour an early development of well-marked silicotic nodules. The lymphoid tissue being directly under the thinned-out bronchial epithelium can apparently "collect" phagocytes from fair-sized air passages with great rapidity. A case of extensive silicosis in the rabbit is illustrated in which dust exposure was continued for 6 months followed by a period of one year in a normal atmosphere. In rabbits killed at the end of the dust exposure there were great numbers of dust cells in the lymphoid tissues but fibrosis had barely begun to occur. After the elapse of the following year little or no elimination of the dust apparently occurred but on the contrary fibrosis had progressed at a rapid rate. The tracheo-bronchial lymph nodes, 18 months after beginning the exposure, are almost completely replaced by hyaline fibrous tissue.

Asbestos Dust Inhalation

The reaction to this dust has differed markedly from that of any of the above-described particulate dusts. Instead of coming to rest in the terminal alveoli as is the case with these types, asbestos particles and fibres are apparently largely caught in the irregular walls of the respiratory bronchioles. There, phagocytes engulf the dust particles but they do not, in the guinea-pig, migrate with them to the lymphoid tissues. Instead they remain *in situ* and as more particles are inhaled they tend to accumulate in the same location. Some of the material

is carried by the phagocytes directly into the walls of the near-by air passages. Solution of the dust takes place as indicated by the development of "asbestosis bodies". Possibly stimulated chemically by the dissolved silicate of magnesium, the fibroblasts in the walls of the respiratory bronchioles begin to proliferate and ultimately a tube-like formation of delicate fibrosis envelopes these structures. Contraction of this connective tissue constricts the air spaces so that their epithelial lining assumes the cuboidal forms of the atelectatic lung. This gives the involved area a gland-like appearance. The development of asbestosis bodies is thought to be a result of hydrolysis and solution of the magnesium silicate of the asbestos molecule. A theory of their formation is discussed. The reaction to inhaled asbestos dust within a period of two and a third years is strictly localised to the respiratory bronchioles. Intra-pulmonary and mediastinal lymphoid tissues play no obvious part in the defence against this dust.

The reaction of the rabbit's lung differs from that of the guinea-pig in that phagocytes do carry the inhaled asbestos into lymphoid tissues in this animal. Furthermore no typical asbestosis bodies have been discovered in this animal; evidence of fibrosis is likewise lacking. However the observations in the rabbit have only been carried over a period of 330 days.

Albino rats have also been employed and in them it has likewise proved impossible to discover an appreciable number of asbestosis bodies. The high frequency of pulmonary abscess in these animals may have been responsible for preventing the inhalation of an effective quantity of dust.

Radiograms of the silicotic and asbestosis guinea-pigs, taken both during the life of the animals and again after the lungs had been removed from the body have been shown. They demonstrate that much greater evidence of disease can be detected in the somewhat over-distended lung which has been removed from the thorax. The appearances are comparable to those encountered in human disease. In the discussion it would appear that the reaction to inhaled asbestos and to a lesser extent to granite and carborundum is analogous to the condition described as bronchiolitis by the South African observers.

From these studies of the effects of various types of inhaled dust it will be noted that connective tissues react to produce fibrosis in various portions of the lungs or their lymphoid tissues. With granite and carborundum fibrosis has been observed only in the tracheobronchial lymph nodes; with asbestos dust the reaction is confined to the walls of the respiratory bronchioles; with quartz dust reaction occurs both in intra- and extra-pulmonary lymphoid tissues as well as in the fine alveolar septa. It is believed that the difference in localisation of various dusts is a function of the action of these dusts upon the phagocytes which ingest them. Possibly this may be due to the degree to which the cells become filled with dust particles. Overloading would mechanically impede the migration of the phagocyte. In the case of asbestos dust the size of the ingested particles may also hinder transportation. As a working hypothesis it is proposed that any dust possessing the necessary chemical composition will provoke fibrosis when and where the phagocytes concentrate it in sufficient quantities in contact with pre-existing connective tissue cells.

Sir Spencer Lister: It was essential for the Conference to reach a conclusion on the question whether silicosis developed as a primary condition or was always dependent on a prior infective process, usually

tuberculous in nature. The animal experimentation seemed to point to the former conclusion.

Dr. Gardner: Care was taken to eliminate tuberculosis as a spontaneous infection.

Dr. Strachan: Dr. Gardner had shown how investigation into the effect of dust on the lungs should be carried on, and the difference between phthisis and non-phthisis dusts, viz. whether they were brought into contact with the connective tissues or not.

Dr. Moore: The necessity for dependence on radiographic findings in the diagnosis of silicosis and industrial pulmonary fibrosis in general has led to the use of diagnostic standards which must be regarded as very largely arbitrary in nature.

With this fact in view, a series of experiments has been undertaken at the Commonwealth Health Laboratory, Bendigo, with the object of correlating the radiographic with the histological findings in experimental animals subjected to conditions calculated to reproduce those experienced by miners working underground on a quartz reef.

The following report represents an interim statement of work accomplished to date, and the research is still proceeding.

Choice of animals. — The animals chosen for the experiment were Australian rabbits which were in the wild state at the commencement of the experiment. Many of them were not fully grown at the time of capture. Very little difficulty was experienced in keeping them in good condition during experimentation, and only three out of 120 died from illness or injury during the course of over six months. The normal life of these animals is three years.

Scheme of investigation. — In order to reproduce underground conditions of dusty atmosphere as closely as possible, boxes were constructed approximately 10 by 11 inches in area and 9 inches deep, provided with an inlet vent about three inches from the floor at one end, and an outlet vent near the lid at the opposite end. These vents were $\frac{1}{2}$ inch in diameter.

For the purpose of convenience in handling, these boxes were constructed in rows of ten and provided with a hinged lid over the whole length of the row, sawn at intervals to enable one box to be opened at a time. The interior of each box was fitted with a glass jar into which dipped a short length of curved piping connecting the interior of the jar to the inlet vent. In each jar was placed finely-powdered dust to a depth of 1 or 2 inches.

The animals were placed one in each compartment for one hour daily six days in each week, and, by means of a bellows, a dusty atmosphere was maintained in the box throughout that period. This atmosphere was sufficiently dust-laden to cause a readily visible haze in the box, and was maintained by blowing at five-minute intervals. During the rest of each day and at the week end, the animals were allowed to feed and rest in the pens.

The animals were weighed each week and weights recorded.

Variations in treatment. — The rabbits were divided into series for the purpose of representing the possible phases of simple silicosis and silicosis complicated by tuberculosis.

Series 1 comprised animals treated by quartz dust only.

In Series 2 the animals were treated throughout the experiment by quartz dust mixed with killed *B. tuberculosis*.

In Series 3 the animals were treated for three months with quartz dust only and after that with dust mixed with killed *B. tuberculosis*.

Series 4 is represented by animals subjected to the inhalation of killed *B. tuberculosis* for five minutes daily for seven days; then, after a fortnight's interval, subjected to daily dusting with unmixed quartz dust.

A fifth Series was treated by intra-pulmonary injection of 1 milligram of *bacillo-casein*¹, and then by daily dusting with unmixed dust.

These series were controlled as follows:

Series 1, 2, and 3 were controlled by rabbits to which no treatment was administered. Series 4 and 5 received treatment by killed bacilli and bacillo-casein respectively, but no further dusting.

The treatment of the series has been continued to date for a period of over six months in the case of Series 1.

Preparation of dust. — The dust used was obtained from mine dumps in Bendigo and was crushed to pass through a 200-mesh screen. The analysis of the dust is as follows:

	Per cent.
Silica SiO_2	92.46
Ferric oxide Fe_2O_3	6.27
Alumina Al_2O_3	0.53
Loss on ignition	0.36
Undetermined	0.38
	<hr/> 100.00

The dust concentration maintained in the boxes during the experiments has been estimated by the Owen's dust counter and has averaged 5,300 particles per cubic centimetre within half a minute of blowing, 2,200 within five to five and a half minutes of blowing and 2,000 within ten to ten and a half minutes. Thus a high dust concentration has been maintained in the boxes throughout the experiments.

Preparation of bacilli. — A human strain of *B. tuberculosis* obtained from the Commonwealth Serum Laboratories was cultured on glycerin bouillon medium for six weeks, then autoclaved at 120° C. for fifteen minutes. The culture was then filtered through paper, washed with saline, and the residue dried in a hot-air steriliser for three hours at 120° C. The dried killed bacilli were then ground in a mortar and bottled.

As required this bacilli dust was mixed with the quartz dust in a proportion estimated to subject each animal to a daily dose of 0.06 milligrams. The quartz dust was baked to destroy other infective agents prior to mixing.

The process described, according to Calmette², should not affect the toxicity of the bacillary endotoxins. Accordingly, this method was used to provoke a tissue response in the experimental animals with a view to providing a parallel to an exceedingly mild or unobservable tuberculous lesion in human beings.

For animals in Series 4 who were subjected to the inhalation of pure killed bacilli prior to dusting, a special box was made with glued and

¹ CALMETTE: *Tubercle Bacillus Infection in Man and Animals*, p. 53. 1923, English edition.

² *Op. cit.*, p. 70.

morticed joints. This box was divided into two compartments, a small one which contained the bacillary dust communicating with a larger one for the body. Two small observation windows were provided.

General procedure. — The rabbits were identified by numbered leg bands and were weighed weekly and the weights recorded. Those receiving infected treatment were segregated from the others, and boxes used for this treatment were not used for other animals.

From the commencement of dusting, animals from each series and control group were killed at stated intervals, a post mortem examination made with particular regard to the condition of the lungs, the lungs and heart weighed, the lungs radiographed by a special method and macroscopic preparations and microscopic sections prepared.

In Series 1 and 2 one rabbit from each was killed weekly for eight weeks, then one fortnightly for a further eight weeks; since then animals have been killed at four-weekly intervals. In Series 3, five rabbits were killed at intervals during sixty-seven days' dusting with infected dust, after two to two and a half months' dusting with simple quartz dust. In Series 4, the dusting period extended from seven to sixty-eight days in different rabbits, and in Series 5, from seven to thirty-nine days. Normal controls were killed at four-weekly intervals and controls to Series 4 and 5 at intervals of two or three weeks.

Method of killing animals. — At the commencement of the experiment, chloroform was used to kill the rabbits, but this was found to produce intense pulmonary congestion and was abandoned in favour of electrocution. It is not certain that this method is ideal, as in certain of the normal control animals a good deal of pulmonary congestion was observed. The first and second series of dusting were repeated on a fresh set of animals up to the eighth animal, using electrocution.

Examination. — At the post-mortem examination, after inspection of the lungs *in situ* the larynx was severed and the heart and lungs removed together. The heart was then removed and weighed, and the lungs were weighed also. One lung was then tied off at the bronchus for histological purposes and cut off.

Radiography. — The remaining lung with the larynx was then placed in a specially constructed vacuum box and inflated by negative pressure therein. Radiographs were then taken by a special technique for soft tissues.

Preparation of specimens. — The remaining lung was then dissected for the preparation of macroscopic colour specimens and for microscopic sections.

Results of examinations. — At this period, after six months' observation in the case of Series 1, and somewhat shorter periods in the other Series, the microscopic sections and radiographic films have been examined and the weights of the heart and lungs compared with the body weights.

Weights. — Comparison of the weights of the hearts freed from the vessels and the lungs and bronchi with uniform portions of the larynges with the body weights at death has failed to reveal any constant ratio. This is probably due to several factors amongst which varying stages of maturity of the subjects and the varying presence of acute infections and congestion are probably the most important. No constant growth curve has been obtained either, probably largely on account of the second factor mentioned above.

Microscopic sections. — The examination of these sections has hitherto

revealed few evidences of chronic change in the lung tissues although, in the later sections, an increase in the amount of fibrous tissues appears.

The characteristics observed relate to acute changes typified by peribronchial and sub-pleural vascular engorgement with patchy areas of pneumonia. Pleural thickening has been observed in some sections. These acute manifestations appear to come in waves, although there is a wide variation among individual animals in response to the treatment. Reaction appears to be more general in animals receiving tuberculous treatment.

Radiographs. — Certain films of the series show evidence of patchy pleural thickening, others show an increase in the strength of peribronchial markings, while others again show a general clouding of the tissues, in some cases approaching a mottled appearance. The typical picture of radiographic silicosis has not yet been obtained.

Correlation of radiographic with histological results. — Comparing notes from the microscopic sections, it becomes evident that the cloudy appearances on the films are due to acute pneumonic and congestive areas in the lungs. The chronic effects of dust inhalation are not so far evident radiologically.

Discussion. — At this stage in the experiments few conclusions can be drawn, but advantage has been taken of this unique occasion to present a progress report. Underground conditions have been fairly paralleled by the experimental conditions, and accordingly, it is hoped that the near future will show us the development of a chronic fibrotic pulmonary condition.

The observations completed so far, however, are not without interest in view of the greatly increased incidence of pneumonia among miners on new fields where a large proportion of the workers are young men, new to the occupation. This incidence and the mortality rate, are found to decrease as the years advance.

This feature has been discussed at length in the Report of the Royal Commission on Pulmonary Disease amongst Miners in Western Australia, 1911 (J. H. L. Cumpston), and the case is cited in this report of a young man, killed by an explosion while at work, whose lungs revealed at post-mortem examination, a pleuritic patch, and an apical area of pneumonia. This man had been employed underground for one year.

The incidence of pneumonia during the early days at Broken Hill (Australia) was pointed out by Armstrong and the South African experience in this direction is well known.

It is noteworthy that practically none of the animals killed showed emaciation or poor physical condition. The few exceptions were those suffering from hydatid infestation or from injuries inflicted by the other animals.

At the present time a few animals of Series 1 and 2 remain. Dusting has been suspended for the present and these animals will be killed and examined a later date.

(The Conference adjourned at 5 p.m.)

SIXTH SITTING

Tuesday, 19 August 1930, 9.30 a.m.

Chairman: DR. A. E. RUSSELL

A letter from Messrs. Kodak (South Africa), Ltd., was read, in reply to the Chairman's letter of thanks for the albums presented by Messrs. Kodak to the members of the Conference.

The Chairman requested Dr. Russell to take the Chair, as he was himself partly responsible for some of the papers now to be discussed. Dr. Russell took the Chair.

THE DIAGNOSIS OF SILICOSIS AS AN OCCUPATIONAL DISEASE

Dr. Irvine: The practical diagnosis of silicosis as an occupational disease was of great importance from the medico-legal point of view. It appeared to him also to be a subject upon which a considerable measure of agreement might be reached by the Conference. Although there were minor variations in the type of the disease met with in different countries the evidence adduced showed that it was everywhere substantially the same disease. Its general type for example was the same at Broken Hill in Australia as in South Africa despite the difference in the silica percentages in the respective rock dusts.

The standard of diagnosis which was needed was that of a condition of silicosis which either caused slight incapacity at the moment or which was of such a nature as to be a potential cause of incapacity in the future. It had to be a standard which was fair to both parties concerned, the workman and the employer.

It was claimed that such a standard could only be reached by a careful correlation of the results of pathological, radiological and clinical examination in a large series of individual cases. The Medical Bureau working together with the pathologists who carried out the routine pathological work on its behalf had been able to carry out such a correlation in a series of 400 cases.

The practical result was to show that *when interpreted in the light of such a correlation* a technically perfect radiograph taken instantaneously formed the most reliable single criterion in diagnosis. It was necessary, however, to supplement the radiological examination by a thorough clinical examination, including an investigation of the examinee's industrial and medical history before a final decision could be reached in any individual case.

The evidence obtained of the effect of the inhalation of silica dust in excessive quantities over prolonged periods was to show that it had in general the following phases:

1. A "*bronchial phase*" consisting mainly of the production of a dry bronchitis and bronchiolitis. This phase when fully developed appeared to give the radiographic picture of a generalised ramifying "fibrosis" or "commencing generalised fibrosis". There were already in this phase aggregations of pigment-bearing phagocytes.

But it appeared to him that the question of what was happening in the lung and respiratory passages during the long period of

exposure prior to the development of actual silicosis required further investigation, both experimental and by observation in the human subject.

2. A simple "nodular phase" based anatomically mainly upon the small aggregations of lymphoid tissue in the lung. This condition was actual "silicosis". It was a distinct and definite pathological entity characterised by a palpable miliary nodulation under the pleurae and in the lung substance, with fibrosis of the root glands. This condition dominated the picture of a clinically "simple" silicosis, and was progressive up to a point even in the absence of infection. But probably only in a minority of cases did it constitute the whole picture even in a clinically simple silicosis, since in a majority of cases of that condition some small latent focus or foci of tuberculous infection was commonly already present.

3. Ultimately in most cases of silicosis there arose an "infective phase" in which the silicotic process was modified by the presence of chronic infections and particularly by a chronic tuberculous infection with the production of areas of "massive fibrosis". Extensive massive fibrosis in a silicotic lung was usually of infective origin and the infection was most commonly tuberculous. Such lesions were apt to be slowly progressive, but might undergo prolonged arrest and might cause no constitutional symptoms. There was applied to them in a general sense the term "infective silicosis" or in a restricted sense the term "tuberculo-silicosis".

But one did not classify cases as "tuberculosis with silicosis" unless an active clinically detectable and deteriorating tuberculosis was present.

In individual cases of silicosis there were many variations in the course and type of the disease dependent mainly on the relative preponderance of the dust factor or of the infective factor, and on the earlier or later manifestation of the latter.

Tuberculous infection might affect a silicotic lung before silicosis developed, or secondly a limited reinfection of the lung might occur simultaneously with the development of the silicotic process, or thirdly, reinfection might occur after the silicotic process was established. The development of a limited area of tuberculo-silicosis at the site of an old persistent focus of primary infection was shown to the Conference.

There was accepted the importance of the view that silica was a soluble cell poison and that this circumstance is a factor in creating a predisposition to tuberculous infection, but this factor did not appear to enhance the virulence of the infection. One would emphasise the feature that a tuberculo-silicosis, as defined, was generally a process of low virulence, with frequently long periods of arrest, with a slow spread in the lung and to other organs, and with a low transmissibility of infection to other people.

As regards the practical definition of silicosis, a sparse palpable miliary nodulation of the lung was the South African definition of a slight degree of silicosis. In some cases of slight tuberculo-silicosis however there might be no disseminated miliary nodulation but merely a few limited areas of massive fibrosis in the apical or sub-apical regions with a scattering of infective nodules in their neighbourhood. Either condition would be shown in a good radiograph.

(A number of cases illustrating the correlation between the radiographic appearances and the actual pathological condition present in cases of silicosis were then demonstrated.)

In arriving at a standard of diagnosis for purposes of compensation it would be wise to rely as a general standard on the type of radiograph which showed indications of commencing nodulation, since this was definitely representative of a slight degree of silicosis. A certain amount of deviation from this standard might be allowed but only to expert examiners. The Bureau for example, as the result of its experience of the correlation described, recognised cases showing the radiographic appearances of a simple generalised arborisation, especially if accompanied by evidence of an infective element, as indicative of silicosis, provided these appearances had arisen in a miner under observation and were accompanied by definite evidence of respiratory disability.

He had no personal experience of any other type of silicosis than that produced on the Rand.

The South African Act recognised two conditions, "silicosis" and "tuberculosis with silicosis". "Silicosis" however might be predominantly "simple" in type without any indication of infection, or "partly infective" in type, in which group indications of latent infection were present. But one did not speak of "tuberculosis with silicosis" unless an active clinical deteriorating tuberculosis was present.

Some practical definition was needed from the pragmatic standpoint of what constituted silicosis, and he would also like to see the Conference come to some agreed classification of radiographic films.

Remarking on the tabulation in Paper 10 of the causes of death in cases of silicosis, he called attention to the preponderant role played by tuberculosis as the immediate cause of death and the relatively minor role played by acute respiratory diseases. In this connection however it was to be noted that in all cases in which active tuberculosis was demonstrably present that condition was returned as the cause of death although in some cases it was not the immediate cause.

He briefly called attention to the observation that the original physical type of the affected man has an important bearing on the type of the disease which is contracted and upon its clinical course.

Extensive investigations into physical tests had been carried out in the mining districts of Western Australia. A limited investigation of this nature had been made at the Bureau. These were the tests applied in the Air Force and similar organisations. In these institutions the man was bent on doing his best, as his job depended on his physical condition, but in the class of man whose financial outlook would be governed to a large extent by his doing his worst, he did not think that this means was helpful in estimating disability.

Disability was estimated from a clinical examination, the history of the man, and the general impression he made. In ordinary routine work the disability was estimated by common sense clinical methods. In the ante-primary stage the disability might be nil and did not exceed 25 per cent. In the primary stage disability due to the disease ranged from 25 per cent. to 50 or 55 per cent. More than 60 per cent. disability justified inclusion in the secondary stage.

Dr. C. Badham: He wished to draw attention to the fact that, although they found in certain industries, such as the Broken Hill silver mines, radiographic appearances which closely resembled those found in South Africa, nevertheless, remembering that they were dealing with a dust containing certainly not more than 15 per cent. of free silica, the rest of the dust being composed of intractable silicates, he was in no way convinced that the disease which they found in Broken Hill was the pure type of silicosis found in Johannesburg. The difficulty in their case was that they had in their State an Act which excluded from

compensation men who had been affected by silica dust. Many of these men had worked in dusts containing percentages of silica ranging up to 35 per cent., and from observations he was by no means convinced that the silica was the whole story of their disease. He found confirmation of that view in the fact that where they had, as in Broken Hill, 85 per cent. of silicates, most of them intractable, they found in the lung after death intractable and other silicates. It was therefore reasonable to assume that silicates had a considerable effect on the production of fibrous pneumoconiosis.

In regard to the inoculation of guinea-pigs with simple silicotic nodules, if the tubercle bacilli were of only low virulence, as those described by Dr. Gardner, he took it that such slight reaction would not be overlooked by the pathologists in the inoculation in Johannesburg of the simple silicotic nodules.

Dr. Gardner: The reaction was slight as far as generalised tuberculosis was concerned. This low virulent strain of bacilli injected into the guinea-pig often produced an enlarged, firm, palpable nodule, which could not be overlooked. He doubted very much whether the lesions produced by a human silicotic tubercle bacillus would be as slight.

Dr. Middleton: In Great Britain they had a series of conditions ranging from the inhalation of silica dust, containing about 98 per cent. free silica, down to dusts which contain a negligible quantity of free silica, or none at all. Medical examinations with radiological examinations had been made, and showed that the effect on the lung seemed to bear a relationship to a true silicosis in so far as the dust to which the sufferer was exposed contained free silica. By that was meant a type of silicosis produced by free silica dust similar to the dusts produced in South Africa. All the granites in Great Britain were not like the true granite of Vermont. They varied chemically, from about 33 per cent. of free silica to a low free silica percentage. In cotton and other textile workers there was a peri-bronchial type of fibrosis, which approximated to the fibrotic picture obtained in the low silica containing dusts. It was necessary to have names for other conditions which arose, because if compensation was made in respect of them, the awarding of such compensation would depend on the definitions and the indications given for making the diagnosis.

Dr. Russell: Vermont granite had a fairly consistent composition of silica. The average temperature half the year round was 36° F. The workmen worked in closed places, and they had very high percentages and a fairly consistent type of silicosis developing pneumonia. That was directly comparable to other parts of the States, where the men worked in the open, and did not require heating, and were also not in close proximity. In the South they had completed a study in the cotton industry, and found that the X-rays from these cotton workers were quite different.

Dr. Böhme: Edling in Sweden has described pneumoconiosis amongst miners in the Swedish coal mines. The coal worked in these mines is very soft and free from mineral impurities. The Röntgen picture resembles that of silicosis. Anatomical examination has, however, revealed that no fibrosis of the lung occurs, merely filling up of the alveoli and bronchioles with coal dust, together with atelectasis and congestion of the contiguous portions of the lung. Very varying anatomical processes may produce similar Röntgen pictures.

The forms of pneumoconiosis caused by silicates and coal require to be subjected to profound anatomical examination before the Röntgen picture can be accurately interpreted.

Professor Kettle: What was the exact anatomical lesion in the X-ray plates of ramifying fibrosis? In fig. 5 of the paper entitled "The Clinical Pathology, Radiology and Symptomatology of Silicosis", he could not see anything in the microscopic picture to correspond with the cases pictured there.

Dr. Fisher: He had been impressed by the small variety and amount of exposure to silica dust, and the gross changes found in the lungs after death. He had also been struck by the fact that although large cavities appeared in the lungs, the pathologist always insisted that there was absolutely no sign of tuberculosis. He thought that in future cases they should try to get biological tests carried out in some of these lungs.

Dr. Cunningham: The South African classification described by Dr. Irvine was in use in Canada. The cases among miners and granite cutters were divided into these stages for practical purposes. In addition however there was at least one other group of cases which had a mixed fibrosis, which the pathologist described as being due not to silica alone nor to tuberculosis alone, with little or no nodulation.

Professor Hall: Perhaps the most important point discussed was the attempt to come to some general agreement on the definition of silicosis, the definition of how it was to be recognised, and how some lead could be given to countries which had to consider the question of legal arrangements. There were one or two points on which he would like information. Was bronchiolitis one of the early conditions which arose in these miners? A great difficulty was to understand the position as regards tuberculo-silicosis. In considering the nomenclature, they had to consider the difficulty of the layman, who rather objected to the term "ante-primary". He would like to know if there was a simple silicosis of any importance apart from tuberculo-silicosis and silicosis with tuberculosis. Was it to be supposed that persons who became infected with tubercles and died of open tuberculosis later, had gone through a stage of tuberculo-silicosis which eventually had come into a stormy condition, or had they had a simple silicosis throughout until they got a late infection?

Dr. Irvine: To label a case "tuberculo-silicosis" and not a case of "tuberculosis with silicosis" was in a sense illogical, but there was a clinical and pathological distinction, and that was why the term "tuberculo-silicosis" was used. There were men who continued with their work, showing no constitutional disability who had a quite massive consolidation in their lungs. It would probably be wiser in the medico-legal aspect not to apply the term "tuberculo-silicosis" to such cases, but simply to say that they showed a silicosis or fibrosis of infective type.

Dr. Simson: During his stay in Sheffield he had quite an extensive experience with tuberculous guinea-pigs after inoculation, doing about forty or fifty a week. Tubercle bacilli of low virulence or in very minute doses might not give rise to any lesion. In such cases the body reactions were sufficient to destroy the bacilli.

In the experimental work carried out conjointly with Dr. Strachan the site of inoculation was carefully examined. Films were made

from any glands showing the slightest enlargement and examined in the usual way.

If there had been any doubt about enlarged glands they would have been reinoculated.

Dr. Strachan: There was no possibility of their having missed any lesions whatever. Negative results were obtained in every case, which showed that none were missed.

Dr. Gardner: In the older literature on silicosis a great point was made of lymphatic obstruction. It had always impressed him that probably this had played a very important part. In dusts such as carborundum they had been able to detect the presence of well-developed silicotic lesions in the bronchial lymph nodes, which were probably responsible for the development of a stasis in the affluent lymph vessels. What physiological activity or lack of activity might result in such a lymph stasis was something to be determined. With such a stasis the channel for the elimination of dust was obliterated. They had not found any dust containing phagocytes, except very rarely, in these dilated lymph vessels. This seemed to indicate that this process of lymphatic obstruction should be given a more prominent part.

There were apparently two types of disease in which silicosis and tuberculosis were co-existent. In one the tuberculous processes were atypical and did not conform to those encountered in uncomplicated cases. In another type lesions comparable to those found in simple tuberculosis could be identified.

The usual lesions of uncomplicated pulmonary tuberculosis are generally divided into three main groups:

- (a) Lesions of infection, the "primary complex";
- (b) Apical tuberculosis, a lesion of reinfection of endogenous or exogenous origin which tends to heal but may be re-activated and may spread at any time;
- (c) Acute and chronic involvement of the lower portions of the lung generally due to broncho-genic extensions from the apical focus.

Blood stream dissemination is more or less accidental in nature and plays little part in the evolution of chronic pulmonary tuberculosis.

In the radiographs exhibited to the members of the Conference there was one beautiful illustration of a "primary complex" in the process of activation by inhaled silica dust. Another series of films illustrated a definite picture of tuberculosis in the apex of a lung which under the influence of inhaled silica dust was actively spreading to involve the lower portions of the lung.

In his own experience the speaker had also seen several cases, chiefly in granite workers, in which an apical tuberculosis was spreading downward through the lung simultaneously involved with silicotic changes. In a series of six lungs from Barre, Vermont, two showed this type of change.

But in many of the radiographs exhibited to the Conference the specific evidence of typical tuberculous lesions were obscured by the silicotic changes.

The question arises whether the combination of tuberculosis with silicosis generally creates a new pathological condition in which the tuberculous elements are no longer typical in their appearance and mode of progression or whether these are only obscured by the concomitant reaction to the inhaled silica.

In attempting to answer this question it would be desirable to obtain exact information as to the incidence of detectable *apical* tuberculosis

both active and inactive in silicotic individuals and as to the frequency of spread of the infectious process into the lower portions of the lungs by aspiration from the bronchial tree.

The speaker also enquired whether it was possible to discover the source of infection in the atypical combinations of tuberculosis and silicosis. If such forms resulted from the combined action of the two irritants, the tubercle bacillus and the silica dust, were each necessarily present within a given tuberculo-silicotic nodule? If so by what pathway did the tubercle bacilli reach the site of reaction?

To mention an entirely new question—is the radiographer correct in ascribing all shadows seen in an X-ray film to the presence of fibrosis? A shadow is cast on the film by any substance which obstructs the passage of the ray through the normally air-containing lung. A pneumonic exudate or even congested and engorged blood vessel may exert such an effect. Why then in dealing with a chronic disease should any area of increased density in the radiogram be called fibrosis?

Dr. Strachan: There was a considerable body of opinion in South Africa agreed in involving lymphstasis as a factor in the development of silicosis. He was of opinion personally that lymphstasis, at least in the simple non-infective silicosis, played no part. Certain workers had shown by experiment that when the lymph glands were blocked with carbon there was no lymphstasis at all. There was, as Dr. Gardner had demonstrated, dilatation of the lymphatics, but there was no apparent interference with the flow of lymph in these dilated lymphatics. The dilatation of the lymphatics might be simply a part in the establishment of a collateral circulation, or opening up the circulation to permit of the further lymph glands being reached. That was their interpretation of the causes in the early simple silicosis. That would not account for the absence of dust cells in Dr. Gardner's cases, but their interpretation of their sections would indicate that there was no lymphstasis. Presumably there would be further findings of cedema, or something of that nature. The point of lymphstasis was important in relationship to the interpretation of the general ramifying fibrosis. It had been raised in that connection because in certain sections of lungs, particularly in the later stages, the lymphatics were filled with pigment and pigment-laden cells, but there were on the other hand cases which showed the typical general ramifying fibrosis in which there was no evidence of dilatation of lymphatics or of pigment-laden lymphatics. To reach a definite conclusion in regard to this condition, much more material would have to be examined.

As regarded statistics on the foci of the typical types of tuberculosis, he could state quite definitely that there were cases of these foci in South African silicotic cases.

Aspiration manifestation of silicosis in the silicotic cases occurred only in the infective cases, and in these only when the infection had gained the upper hand.

Infection elsewhere was a point which had not been emphasised sufficiently. Both in natives and Europeans there were certain cases in which the manifestations in the lung were minimum, if not absent, but in the lymph glands there was evidence of gross tuberculo-silicosis, and a not infrequent sequence of events in these cases was a dissemination of tubercle to other organs.

Dr. Gardner: He had found non-progressive tubercle in the abdominal viscera of about 25 per cent. of cases of simple chronic pulmonary tuberculosis. He did not feel however that the blood stream played

an important part in the dissemination of tubercle bacilli within the lung in the chronic stages of pulmonary tuberculosis. With regard to lymphstasis, a dilatation of the peripheral lymph vessels of the lungs appeared in the absence of an obstructive lesion in the tracheobronchial lymph nodes. In this case, the dilatation could only be related to intra-pulmonary irritation. He was anxious to discover whether this appearance was indicative of any change in the rate or direction of lymph flow.

Professor Kettle: He would still like an answer to his question about the bronchiolitis. He would like to know what was the significance of the pathological process. Was it because of atrophy of the epithelium and a defect of secretion, and therefore a matter of holding up of the dusts, or was it because of the lack of the mucous membrane that the dust got through so rapidly? Was there any evidence of an increased phagocytosis in these circumstances, or was it a question of an interference with the exit of the dust once it had been inhaled?

In experimental work he had been very much impressed by the determination of the dust in the phagocytes to the lymphatic vessels' system. It seemed that the lymphatic must enter into this process rather more than had come out in the various discussions.

Dr. Strachan: He did not deny that the lymphatic apparatus played a very important part in this condition, but it had an active and not a static part.

Professor Kettle: If the lymphatic glands were obstructed and filled with silica, there must surely be obstruction of the lymphatics.

Dr. Strachan: In the specimens at the Institute there was no difference from the normal lung. He thought the glands were enlarged and there was a certain amount of obstruction in the glands. If there had been stasis, and stasis of any degree, the pigment and the dust would remain in the lung.

Dr. Gardner: He considered that the abdominal lymph glands were involved. The individual abdominal viscera simply passed these dust cells through to the lymph nodes which drained them.

Dr. Strachan: If there was a blood stream infection one would naturally expect to find manifestations of the lesions in the spleen. These were extremely rare.

Bronchiolitis was put forward simply as an observed fact. As regarded the question of exit, it seemed that the bronchial epithelium was active in the process of carrying away material. What mattered was not so much the interference with the exit of the particles as the possibility of their increase. The bronchiolitis of asbestosis threw a considerable light on the subject in relation to the X-ray picture. The degree of bronchiolitis in silicosis was less than in the asbestosis. The reaction in asbestosis was much greater, and so great as to prevent the involvement directly of the terminal alveoli.

Dr. Mavrogordato: One of the difficulties the Conference would have to face would be the distinction between a silicosis and a pneumoconiosis. The problem was to get at silicosis as a specific occupational disease and define it. It was much more difficult to spread it to other types of pneumoconiosis. If a man was going to get a silicosis he would precede his silicosis with fibrous ramification. He had seen a number of workers getting on for sixty who would yet show lung fibrosis of that kind. They were apparently physically fit, as men in the neighbourhood of the sixties go.

Personally he did believe that lymph played a part. He looked at the spread from the lungs somewhat differently from Dr. Strachan. He thought that the fact that the pigment cells passed so rapidly up into the neck and down into the abdomen tended to support the supposition that the normal lymph flow was interfered with. An acute infection of the lung in a man who had not been exposed to dust for several months would show the alveoli filled up with pigment-laden cells. That suggested that the pigment-laden cells had been retained somewhere, not necessarily in the lymphatics. In his experience there was in these cases plenty of pigment-laden cells held up in the distended lymphatics. It seemed that at first there was an interference with the normal lymph flow, then a pretty ready passage in an abnormal direction, and then a holding up of the pigment-laden cells somewhere where they could get back into the lung.

Professor Hall: Whilst it was quite true that general fibrosis might go on to silicosis, yet quite a number of cases did not go on to simple silicosis.

Dr. Irvine: The radiographic picture of general ramifying fibrosis was a definite pre-silicotic phase. At post-mortem something like 50 per cent. of such cases showed slight silicosis in the sense defined. The practical working test was that if a man in that condition with a long service had developed the condition underground in the course of his service, and there was disability, generally with some emphysema, they were inclined to compensate at that stage, under these restrictions, but not in general. He would not for a moment say that any worker who showed that type of plate was entitled to compensation. A number of miners could go on for several years in that condition without progressing further, but if a man continued in his occupation, a definite silicosis usually followed in a year or two. A man might however develop that condition and remain in it practically indefinitely if he was removed from underground work. It was necessary to get at the facts of the "actual generalised ramifying fibrosis", which did not necessarily spell fibrosis.

Dr. George: A few figures with regard to Broken Hill miners may be interesting. In eight years, out of 160 mine workers withdrawn as suffering from simple silicosis 76, or 47.5 per cent. developed or died of pulmonary tuberculosis. A total of 66, or 41.2 per cent. died of all causes. The average age at death of those dying of pulmonary tuberculosis was forty-eight years; in those dying of other causes the average age at death was fifty-eight years. In eight years, of 101 men classified originally as suffering from silicosis plus tuberculosis there were living only 15—86 per cent. had died. In all but eight cases the cause of death was pulmonary tuberculosis. The average age at death of those dying of pulmonary tuberculosis was 49.7 years, and in those dying of other causes 51.6 years.

The chief causes of death in these two groups of men who did not die of pulmonary tuberculosis were: lobar or broncho-pneumonia 8, other respiratory disease 7, heart failure 8, chronic nephritis and/or cerebral hæmorrhage 11, malignant disease 4, surgical emergencies 2, suicide 2. With regard to the number of deaths from chronic nephritis and its complications, it must be remembered that many of these men had suffered from lead poisoning in addition.

Dr. Moore: In our experience at Kalgoorlie (Western Australia) and in Tasmania the performance of physical tests and the estimation of vital capacity was of very little aid in the diagnosis of silicosis.

Dr. Kranenburg: The X-ray of the sandstone workers showed, for 16 persons, mottling in 2 cases and snowstorm type in 5 cases. The X-rays of 74 Belgian limestone workers showed mottling in 5 cases.

Dr. Watt: The branching shadows in an X-ray film were caused by the blood vessels, by the lymph vessels, and by the supporting connective tissue. In heart disease, where there might be some increase in the vessels in the lungs, there were ramifying shadows, which might also be caused by the dilatation of the lymph vessels. The air-containing tubes threw a negative shadow. It was conceivable that the dilatation and blood vessels would give those shadows without necessarily a marked fibrosis.

(The Conference adjourned at 12.30 p.m.)

SEVENTH SITTING

Tuesday, 19 August 1930, 2.30 p.m.

Chairman: DR. A. E. RUSSELL

DIAGNOSIS (*continued*)

Dr. Böhme: He had heard that 70 per cent. or more of the natives had a positive von Pirquet reaction; he thought therefore that the conditions were not so very different from those in Europe. Most of the natives had a tuberculous infection in their youth; why were they so liable to contract tuberculosis when they came to the mines, or into contact with European people?

Dr. Mavrogordato: Very weak solutions had been used for testing purposes. Were they to use the same strength as in Europe, the positive reactions would probably be about 90 per cent. But whereas in Europe a positive reaction was regarded as related to a localised tubercle of a more or less chronic type, in South Africa the fact that a native had a positive tuberculin reaction did not make the least difference to his after-coming type of tubercle. It was at death generalised. In fact the natives who got the least tubercle were those who had a negative reaction. This was the exact reverse of the European experience.

Dr. Badham: It has been suggested by speakers that the appearances seen in the radiographs showing diffused generalised fibrosis are due to bronchiolitis or effusion.

He desired to point out that in certain of these cases there is no increase of sputum and the radiograph remains unchanged for three years after leaving dusty work.

The condition appeared to him to resemble closely the radiographic appearance of asbestos and he thought that he would find the pathology of these lungs to resemble that found in asbestosis.

He had not found this condition in individuals not exposed to dust.

Dr. Cunningham: It appeared that cases showing more general ramifying fibrosis did not get tuberculosis to anything like the extent that ante-primary cases did. Having in mind the fact that the amount

of simple tuberculosis was comparatively low, something must happen just short of ante-primary to make tuberculosis much more serious or much more frequent in these miners than earlier.

Dr. Irvine: Tuberculosis in a silicotic lung might arise from a persistence of a primary infection, or it might come along with the dust and develop practically simultaneously. He thought these latent foci either remained inactive, in which case the condition did not advance to an obvious condition, or, on the other hand, these foci became active, either rapidly or over a period of years. They became active and progressive either locally, or spread by inhalation, or through the blood stream and caused a definite tuberculous infection. There was the further possibility of reinfection from outside the lung after the silicotic process had become definitely established.

Professor Loriga: After the Lyons Congress, the Medical Labour Inspectorate in Italy had asked for collaboration from various experts in the study of the pathology of marble workers. Investigations were made by Dr. Turano at Carrara, and Dr. Bianchi at Massa, the chief centres of the marble industry. Further enquiries were carried out by Dr. Lovisetto, of Turin, and by Dr. Mussa. Their conclusions had already been submitted to the Conference. He exhibited various radiographs of the results obtained by these investigators.

Dr. Russell: Having regard to the apparently fairly long period of occupation in the marble industry, it appeared that in Italy it was the practice to teach school children a trade as part of the school curriculum. Children commenced learning in the marble industry from the age of ten years.

Dr. Gardner: He had had the good fortune of seeing a series of asbestosis films, which Dr. Lanza had in his possession. The majority of these cases were much like those Professor Loriga had shown, in that there was an accentuation of the linear markings in the chest film, with relatively little of the fine mottling. Dr. Irvine had shown a film that morning which showed more of the mottled appearance, which was perhaps a little more comparable with the picture obtained by the experimental disease in guinea-pigs.

Dr. Russell: Dr. Lanza was particularly interested in the incidence of tuberculosis in his people. He was of the opinion that they had more of a chronic bronchitis than a tuberculous complication.

Dr. Middleton: He had been recently associated with Dr. Merewether, of the Factory Department, in an enquiry on workers actually employed in asbestos factories at the time of the examination. The general result was to show that asbestos dust gave rise to pneumoconiosis of a fine type, distinct from the silicosis. A number of fatal cases had occurred in Great Britain, and he had been present at one or two of the post-mortem examinations. The findings were that the appearance of the lung corroborated the X-ray appearance, so far as the absence of the large nodules was concerned. There was a general fibrosis throughout the lung. Usually the end of the case was either tuberculosis or a broncho-pneumonia. The remarkable thing about asbestos disease was the apparent small involvement of the lungs when examination, either clinical or radiological, was carried out. The patient lost condition and went down hill fairly rapidly, when the clinical and radiological signs were scarcely to be made out. The incidence of tuberculosis in such cases was interesting.

Intensive study was being made with regard to prevention to ascertain

how far the dust could be eliminated from the industry and with regard to further legislation and compensation. A Bill had been before Parliament in the last few months to provide compensation for persons disabled by the pneumoconiosis due to asbestos dust.

Dr. Badham: No asbestosis disease had so far been found among Australian workers.

Dr. Orenstein: Asbestosis had been studied for some time by one of the medical men in South Africa in the mines of the Eastern Transvaal. Dr. Slade claimed to have found a number of cases of asbestosis with incapacity.

Dr. Irvine: Dr. Slade had sent up one man who had been in an asbestos mill for six and a half years, and showed quite a definite generalised arborisation in the radiogram, not quite of the silicotic type. It resembled it, but was a little more diffuse. Dr. Slade had been sending up to Drs. Simson and Strachan a large number of sputa from natives in which they had recovered asbestos bodies.

Dr. Simson: He had had the opportunity of examining material from six cases of pulmonary asbestosis from a mine in Southern Rhodesia. All showed definite fibrosis and two were complicated by active tuberculosis.

In a case with a history of exposure to dust for two months asbestosis bodies were found in the lung alveoli, but there was no evidence of fibrosis.

He was interested in the examination of sputa for asbestosis bodies with a view to determining whether their presence was an indication of pulmonary fibrosis. Dr. Strachan and he had examined sputa from fifty subjects who had worked in an asbestos mill. Asbestosis bodies were demonstrated in the sputa of forty-eight of these, and in most of them by the direct film method. The shortest exposure was five months. Another case had a history of four months' exposure to the dust of the mill and three months' exposure to underground conditions. Dr. Strachan and he were informed that underground conditions are not likely to cause fibrosis as there was no excess of injurious dust.

Professor Hall: One of the objects of the Conference was to arrive at some international standard which would be applicable to all countries for future investigations. He therefore moved the following resolution:

There is a pathological condition of the lungs due to the inhalation of free silica dust. It can be produced experimentally in animals. It can be detected by clinical, radiological and pathological means with sufficient accuracy to separate it from other pneumoconioses and to afford a fair basis for legislative measures.

Dr. Böhme seconded the resolution.

After some discussion, it was resolved unanimously, on the motion of Dr. Orenstein, that the resolution as amended by Professor Hall should be referred to the Reporters. The resolution as amended was as follows:

There is a pathological condition of the lungs due to the inhalation of free silica dust. It can be produced experimentally in animals. It can be detected by clinical and radiological means, which can be confirmed with the above pathological condition with sufficient accuracy to separate it from other pneumoconioses. It also affords a fair basis for legislative measures.

Professor Hall: The next step was to come to some general agreement, based upon the lines of the South African experience, on a definition of the three stages of silicosis. He moved the following resolution:

The *first stage* shall be defined as characterised by:

- (a) appearance of the earliest detectable physical signs of the disease;
- (b) radiographic appearance not less than the presence of nodular shadows together with an increase of hilum shadows, linear shadows and pulmonary reticulum, and
- (c) with or without impairment of capacity for work.

The *second stage* is characterised by:

- (a) the further development of the physical signs found in the first stage;
- (b) radiologically an increase in the area of the nodular shadows with a tendency to confluence of the individual nodules, and
- (c) the presence of symptoms with some degree of impairment.

The nomenclature classifying fibrosis in various degrees should be replaced by the terms "slight", "moderate" and "well-marked" linear radiation.

Dr. Orenstein seconded the resolution.

It was unanimously agreed to refer this resolution to the Reporters.

Professor Hall: He felt that the so-called infective silicosis, whether tubercular or not, would be much better left out altogether. There should be a separate heading "tuberculosis" with silicosis as a distinct condition.

Dr. Middleton: Tuberculosis should be recognised as an entity, and should be placed parallel with or alongside the three stages of silicosis.

Dr. Orenstein: In connection with silicosis it was necessary for the Conference to express an opinion as to whether tuberculosis was a disease which was accelerated by the presence of silicosis, or whether silicosis accelerated existing tuberculosis. He took it that the general opinion of the Conference was that the two were unconnected. If that was the case, he entirely agreed with Professor Hall's suggestion that tuberculosis should be dealt with as an auxiliary entity to silicosis, but before he would commit himself to that view he would like to know whether the delegates from other countries agreed.

Dr. Cunningham asked whether if silicosis and tuberculosis were defined as distinct conditions the "infective" type of case would be labelled "tuberculous".

Dr. Hall: No.

Dr. Irvine: The South African definition of tuberculosis was as follows:

That a man shall be considered to be suffering from tuberculosis if either he is expectorating the tubercle bacillus or is suffering from closed tuberculosis to such a degree as to seriously impair his working capacity.

They introduced the term "infective silicosis" because that was a pathological entity and because it was necessary to describe it in describing silicosis at all. They did not recognise tuberculo-silicosis as tuberculosis for the purposes of the Act until it was active and progressing and causing constitutional deterioration.

Dr. Orenstein: The Reporters could take it that the Conference agreed to the view that tuberculosis in relation to silicosis should be dealt with as a condition parallel to it and adding to the disability.

Dr. Moore: The question of radiographic technique had not yet been mentioned.

Dr. Russell agreed that it was essential to make some reference to radiography.

Dr. Orenstein asked if he was right in understanding that the Conference had accepted Professor Hall's proposal that in the present state of knowledge of the pathology of the disease the view was not considered justified that silicosis was essentially tuberculous, at least as seen in South Africa.

Dr. Russell replied in the affirmative.

(The Conference adjourned at 5 p.m.)

EIGHTH SITTING

Wednesday, 20 August 1930, 9.30 a.m.

Chairman: DR. L. G. IRVINE

DIAGNOSIS (*continued*)

Dr. Simson: In the lungs of subjects exposed to the influence of injurious dust there might be condensation of the tissues in and about the bronchioles, and these areas of condensed tissue might be responsible in part for the radiographic appearances known as linear radiations. Somewhere in the site of the condensed tissue, in silicosis the nodule of fibrosis developed.

In 1916 Drs. Watt, Irvine, Pratt, Johnson and Steuart demonstrated nodules appearing on the linear radiations, and defined this phenomenon as "segmentation of the linear shadows". Dr. Böhme likened it to beads on a string.

Dr. Gardner, when demonstrating cases of experimental asbestosis, showed radiographs of the chest of a living guinea-pig and of the lungs in a state of over-expansion from the same pig after death. In the living pig the radiograph showed radiating linear shadows, but in the over-distended lungs the radiating linear shadows had become segmented and appeared as a fluffy kind of mottling. This might account for the shadows seen in the peripheral part of the lung. Thickening of the bronchi, dilatation of bronchial lymphatic and blood vessels might account for the increase in the shadows near the root of the lung.

The Chairman: He agreed that in the bronchial phase "fibrosis" was a compromising term. Fibrosis or potential fibrosis did, however, occur in many cases which showed a radiograph of generalised arborisation, because this picture persisted afterwards, as if there were slight but definite fibrosis.

Dr. Gardner: Carborundum dust inhalation gave an example of increased vascularity. X-ray films of human cases exposed to carborundum dust showed a definite accentuation of the linear markings.

Dr. Watt: Stereoscopic films would show the nature of the shadow very much better.

PROGNOSIS AND AFTER-CARE

Professor Hall asked *Dr. Pringle* if he would elaborate the statements made in his paper (page 583) that "the amount of dust a patient may have and the degree of tubercular infection do influence prognosis, it is true; but I hold that the factor of individual resistance to tubercular disease is very great" and "the non-miner with pulmonary tuberculosis stands no better chance than a miner with silicosis complicated with tuberculosis". He would have thought tuberculosis complicated with silicosis had a very much diminished chance of recovery.

Dr. Pringle: The second statement quoted was qualified by the words "but in some of these cases the illness probably was contracted earlier in life". Non-miners seemed to die as early as miners. Where the patient was originally certified as in the ante-primary, primary or secondary stage of silicosis, silicosis did not seem to influence the period of life; it seemed to depend entirely on individual resistance to secondary (tubercular) infection. There were patients at the Springkell Sanatorium between sixty and seventy years old, which showed that individual resistance to tuberculosis was a considerable factor.

The Chairman: The figures of the Medical Bureau showed that silicosis with active tuberculosis at the outset led to earlier death than simple tuberculosis. The average silicotic had a much longer expectation of life. Infective silicosis meant not necessarily active tuberculosis but a chronic condition of tuberculous infection with silicosis, sometimes arrested, sometimes progressing. There were two legal groups: (1) silicosis which was predominantly simple or "partly infective" unaccompanied by obvious active tuberculosis, and (2) silicosis with obvious active tuberculosis. *Dr. Pringle's* remarks evidently referred to the second group.

Sir Spencer Lister: *Dr. Pringle* and the Medical Bureau's views seemed to be in conflict.

Dr. Orenstein: In his opinion the probability of survival of non-miners with tuberculosis was no better than for miners with silicosis and tuberculosis; the factor of age distribution entered largely into the picture; greater age meant greater survival, and miners were generally older than non-miners.

The Chairman: He agreed. Dust phthisis was a disease of later life than was tuberculosis, because of the fact that time was necessary for the predisposing condition of silicosis to develop.

Dr. Pringle: A large number of miners were certified as suffering from tuberculosis, but the tubercular element did not seem very active.

Dr. Badham: If silicosis and overt tuberculosis developed together the prognosis was very unfavourable. He asked whether *Dr. Pringle* referred to that type or to silicosis with a later development of tuberculosis. The expectation of life in these two groups was quite distinct.

Dr. Pringle: Cases were sent to Springkell by the Bureau as suffering from silicosis with tuberculosis, but the certificate did not distinguish

between chronic and active tuberculosis. The case in which the tuberculosis factor was not very active and the sputum negative tended to live a long time while the second type lived only a short time.

The Chairman: The Bureau's report showed that the maximum mortality for tuberculosis with silicosis fell between forty and fifty years of age. The numbers of deaths from tuberculosis in each year subsequent to the year of detection was fairly equally distributed.

Dr. Simson: In tuberculosis with silicosis the organism was living on a medium composed mainly of fibrous tissue which was not very nutritious as compared with the other type which was living outside the silicotic nodule.

Dr. Middleton: The age incidence of the disease was a very important factor. There were two crests in tuberculosis in the adult: (1) between twenty-five and thirty-five, and (2) between forty-five and fifty-five years of age. In the first group there was progressive tuberculosis with acute phenomena while the second group was remarkably free from these phenomena and the disease was much more chronic.

Active tuberculous foci outside the fibrous nodules tended to march alongside silicosis. Silicosis with tuberculosis improved with hospital treatment but there was a tendency to relapse later.

Dr. Böhme: Silicosis with overt tuberculosis in rock drillers generally went faster than tuberculosis alone. Most of the cases which he had seen were between 40 and 50 years of age.

The Chairman: The amount of fibrosis in the lung was a very important factor. Once a tuberculous infection had become manifest, the old type of silicosis progressed very rapidly, while the infective cases to-day ran a more chronic course.

Dr. Orenstein: The attempt to correlate the evidence statistically had shown the age distribution among miners to have shifted enormously during the last few years.

Dr. Russell: In the granite industry two types of tuberculosis occurred: (1) between the ages of twenty-four and thirty-eight which responded fairly well to sanatorium treatment, and (2) after twenty-five years' exposure to dust at an average age of forty-nine years; in this group the tuberculosis had a greater tendency to pulmonary hemorrhages and the course of the disease was unaffected by sanatorium treatment. The type of tuberculosis which did not respond to sanatorium treatment was also found among silicotic potters.

Dr. Gardner: He had carried out experiments on the same kind of animals of the same age and with the same doses. In the first experiment the infection was coincident with the exposure to dust. After three months the infection spread, ran a sub-acute course and ended in death from acute and chronic tuberculosis. In the second experiment the animal was exposed to quartz dust when tuberculosis had already been established. In this case there was a much greater tendency to fibrosis. In the third case silicosis preceded the tuberculosis infection. A very active tuberculosis was produced which in the majority of cases ended in death within 60 days.

Dr. Watt: The racial factor was also very important. The mining population had been drawn first from the densely populated areas of Great Britain and secondly from the country population of the Rand. The Bureau's examinations were so strict that the mining population

was now A 1. He had seen cases improve under sanatorium treatment, but ultimately the end came rapidly.

The Chairman: There was a slightly higher rate of progress among South African miners as compared with miners from overseas. Seventy per cent. of the silicosis cases were among the old Rand miners, and only 8 per cent. among the new Rand miners. The new Rand miners produced as little pure tuberculosis as the Royal Air Force. The present mining population was over 70 per cent. South African.

Dr. George: The Broken Hill statistics showed that in 1920: (1) 102 miners were withdrawn with silicosis with tuberculosis, and (2) 107 miners were withdrawn with uncomplicated tuberculosis. Of the first group only 15 were now alive, while 55 were alive in the second group. The average age of death from tuberculosis in the first group was 49.7 and in the second group 45.5. The average age of those still alive in the first group was 53.5 and 54.5 in the second group. He asked what percentage of Rand miners who obtained certificates actually got work.

The Chairman: All comers on the Rand were eligible for initial examination and some of these came up year after year. The actual number of examinees was at least 16,000. The new Rand miners who entered upon their fifth year of work did not amount to more than 50 per cent. of those of that group who had started work underground.

Dr. Cunningham: We would like to hear the opinion of the Conference on the subject of sending men compulsorily to sanatoria.

The Chairman: There was no compulsion in South Africa and there was no systematic feeding of the sanatorium. A tuberculosis dispensary had been established in Johannesburg, but it was closed down because so little use was made of it.

Mr. Spence Fraser: Dr. Pringle's statement was based on the fact that cases of silicosis with tuberculosis died at a later age than cases of uncomplicated tuberculosis. This, however, was to be expected, since they contracted the disease at a later age. In order to measure the rate of progression it was necessary to calculate the average time from the detection of the disease until death. Table IV in Dr. Pringle's paper appeared to show that the rates of mortality among non-miners were rather lower. He did not think that any case had been made out to show that non-miners had a shorter lifetime.

Dr. Orenstein: Statisticians and medical men were unable to draw any conclusion from vital statistics unless they could use them biometrically. Table IV had no particular meaning because it was not carried to finality. Owing to lack of data it was impossible to reach comparative figures of deaths from tuberculosis in the general population and the mining population. The economic factor had also to be considered, since the economic conditions in which the men found themselves before the disease might not be the same as when they had lost their employment owing to the disease. Statistics were available to show that the higher class of officials did not progress so rapidly as the ordinary worker. It was also necessary to measure the rôle played by physical and racial selection, which was becoming increasingly important; in that should also be included the rôle played by individual resistance to tuberculosis, which differed racially and individually. In 1918 to 1919, 9.6 per cent. of European underground workers had been employed ten years or more; in 1925 to 1926, 36 per cent. had been employed ten years or more. In 1918 to 1919, the silicosis peak occurred

at nine years, while in 1925 to 1926 it occurred at twelve years. It was of the utmost importance to have a biometric examination made of the material in order to discover whether miners' phthisis had been reduced or not.

Dr. Koelsch: The problem of the *connection between tuberculosis and silicosis* and of *prognosis* was very difficult and depended on numerous subsidiary factors—for instance that of early or late tubercular infection, of the spread of localised tuberculosis, of individual susceptibility, etc. By means of very extensive statistical investigation it was possible to gain some ideas on the subject. He had analysed the relative morbidity and mortality returns for well over 100,000 workers in dusty trades and compared them with similar figures for the local population. He was able to establish two critical periods:

The first at the age of about 20-25 years (= 5-10 yrs. of employment).

The second at the age of about 45-55 yrs. (= 30-40 yrs. of employment).

In the first group they had irritation due to dust and tuberculosis which causes death relatively rapidly. Amongst those of the second group conditions depend to a certain extent on the trend of dust—that is, on the silica content of the dust: amongst sandstone workers, for instance, there occurred severe tuberculosis with silicosis which relatively rapidly becomes fatal, while in the case of pottery workers for instance the disease followed a more protracted course.

In regard to sanatorium treatment he had not very much faith in it. As long as the workers remained in the sanatorium they were well, but as soon as they returned to work in a dusty atmosphere they went from bad to worse. The insurance society, therefore, undertakes, in the case of sandstone workers for instance, the application of sanatorium treatment only when the patient gives a written assurance that he will exchange his occupation for work in a dust-free industry.

Dr. George: In 1922 and 1923, thirty-four ex-Broken Hill miners suffering from uncomplicated silicosis were placed on irrigation blocks at Griffith, New South Wales. These blocks were situated on virgin country, they cleared the land, erected their dwelling houses with new timber and lived there with their wives and families. Compensation was paid throughout fortnightly, and advances of money and material were made by the Irrigation Commission. Grapes, apricots and citrus fruits were grown, the former two for sale after drying. The climate was cold, 2,000 feet high and in winter rather damp and the work necessitated fairly frequent work in wet conditions. The men were told that as they had no incapacity they would make a success of their new lives and were eager to take up the work. The men sent were selected after a special medical examination to ensure that they were suitable for the work. Almost from the commencement the scheme was a failure. The men could not stand up to the harder work—ploughing, hoeing, etc.—and soon several men abandoned their farms. Up to the present, of these 34 men 17 had developed or died of pulmonary tuberculosis, 1 had died of sudden cardiac failure and 1 of cerebral haemorrhage. There were about 9 of the men still on their farms, some of whom have definite tuberculosis. They owe sums varying from £2,000 to £3,000 on their farms. Repayments are due, but they are able only to keep going by using their fortnightly compensation payments to pay labourers to do the hard work. He had re-examined these men once yearly and was convinced that the symptoms of which they complain—dyspnoea on exertion, præcordial pain, palpitations, etc.—are genuine and that the miner with fully developed silicosis has a

definite disability for work of this nature. At the age of forty to forty-five a man who has been a miner all his life is unsuited to take up entirely different work which is entirely new to him. He is too old to educate different work muscles and in the attempt to do so develops fairly quickly symptoms and signs of cardiac strain. As the majority of these men have young families, from the point of view of their children no doubt this scheme justified itself to some extent.

Dr. Pringle: The experience at Springkell and Wedge Farm Sanatoria corroborated Dr. George's experience. Men certified as silicotics would not and could not do agricultural work. It was suggested that there was nothing to be gained by warning men to leave the mines because men who did so and received lump sum compensation, although excluded from the scheduled mines, could find employment in Rhodesia and in the asbestos mines in Barberton and elsewhere. He asked whether any special hospital provision was made for silicotics in other countries.

Dr. Middleton: No special provision was made for silicosis in Great Britain, but silicotics with tuberculosis were eligible for sanatorium treatment under the National Health Insurance Act.

Professor Hall: When considering the factor of economic conditions it might be taken into account that managers and other officials were perhaps less exposed to dust.

The Chairman: After a man had received a notification that he was silicotic he forfeited the right to any increase in compensation if he did not leave work within three months; but the number of silicotics who did not leave underground work was small. They progressed no worse than the men who did leave, but they were of a robust type and the majority were mine officials with better economic conditions and less exposed to dust. The Medical Bureau believed that men who continued underground work would be prejudicially affected, but only about 25 per cent.

Mr. Du Toit: The Miners' Phthisis Board had tried to make comparisons between silicotics established in the Cape Province as compared with silicotics at 6,000 feet altitude in the Transvaal. The Cape silicotics had not been able to remain long and had had to return to a higher altitude. The Transvaal silicotics, owing to the difficulties of the work and the extreme cold in winter, were not able to last long on a farm. These men nevertheless had originally come from rural districts. In both groups silicosis had progressed. About £3,000 per man had been expended unsuccessfully.

Dr. Russell: The experience with granite workers in the United States had been similar. The North Italians who had migrated to California to take up grape farming had failed and other granite workers had also failed at farming. They were unable to adjust themselves to new occupations.

Dr. Orenstein asked whether there was any other organisation elsewhere in the world established by private industry comparable to the South African institutions for the care of miners' phthisis cases.

Professor Böhme: Miners suffering from tuberculosis or tuberculosis and silicosis in Germany have for years back been treated in the phthisis hospitals of the Miners' Association (*Knappschaftsverein*). This Association is an insurance society against illness, invalidity and old age. The cost of insurance is borne partly by the employers and partly by the miners. Tuberculosis occurring in other occupations is treated at

the expense of the district insurance societies (*Landesversicherungsanstalten*). The expenses of this society are likewise borne partly by the employers and partly by the employees.

Professor Hall asked what was the effect of silicosis and tuberculosis on natives.

Dr. Orenstein: The incidence of silicosis, simple or not, on natives was relatively low, but it should be remembered that the mines population changed 100 per cent. per annum. The recorded mortality of 8 per cent. was much exaggerated and it could be assumed that intermittent employment gave considerable protection. Natives were more susceptible to tuberculosis, but racial immunity was being rapidly acquired.

The Chairman: Simple silicosis had been shown by radiographic examination to be much less frequent among natives, but the comparative incidence as between Europeans and natives was much the same, given similar conditions.

Dr. Watt: The natives did not breathe through their mouths and, therefore, were protected by a better filter than Europeans.

STATISTICS OF SILICOSIS

The Chairman: The figures showed that silicosis was definitely on the decline, especially among the new Rand miners.

Dr. Orenstein: Without casting any reflection upon the statistical work being done, it must be realised that it was very difficult to visualise all the factors without biological and engineering knowledge. The present statistics were perfectly reliable, but did not reflect the whole picture. It was impossible to say whether the real production of silicosis was more or less than that shown by the figures. Improved radiological technique was now more important than clinical diagnosis, and more diagnoses were now made.

He suggested that the money now set aside for compensating cases of the disease might be better spent upon rehabilitation methods. A pension for a disease which tended to progress might not be doing full justice to a man. South Africa spent about £800,000 a year on compensation and there were liabilities of £9,000,000. Under the present system in the ante-primary stage of silicosis a man was penalised if he did not leave the mine at once, and about £500 was given to a man thus thrown out of employment. He thought it might be better to wait until a marked degree of incapacity was reached and that search should be made for other avenues of employment. The farming experiments had not perhaps been efficiently conducted. There was room here for further investigation and research.

Professor Hall: The statistics of South Africa were so unique and of such importance to all countries which had to initiate legislation and compensation, that every step should be taken to make them as clear as possible. Biometrical statistics involved many other factors and it was desirable that all the material should be handled by the greatest biometrician obtainable.

Mr. Spence Fraser: A biometrician might obtain valuable results from the statistics, but it was very difficult to obtain an all-round man; a mixed Committee might perhaps be necessary. It was doubtful if the data existed to enable a biometrician to produce useful results.

If compensation for ante-primary silicosis had existed from the beginning the figures to-day would appear better to-day because more miners would have been excluded from employment in the earlier years. The duration of work was shown in the statistics as the main factor. The age figures published by the Bureau showed that the factor of age was of little importance; it did not, for example, affect mortality in the secondary stage. The ante-primary stage had not been constant owing to improved technique and other factors, and the production rate had consequently increased for some years, though that had been foreseen. The liability of the miner differed greatly according to the character of his occupation, but for purposes of compensation no distinction was made between the various occupations. The proportion of miners in the different occupations had not, however, changed to any marked extent. He thought that the statements in his and Dr. Irvine's paper of the decrease of production was a fair statement of the conditions.

Professor Kettle: The statistics were beyond criticism, but he hoped that a biometrician could carry out the widest possible survey.

Dr. Mavrogordato: Two factors were at work: (1) the change from a migratory to a fixed population, and (2) improvement in conditions, in which must be included the initial examination. He believed that as much ante-primary silicosis was made as ever, but it now took thirteen years instead of eight to nine years. This was probably due in part to selection of miners and the production rate would fall as the number of picked men increased. If the time taken to produce a clinical silicosis could be pushed up to twenty years silicosis could be considered as eliminated on the Rand from the social point of view.

The Chairman: From 1912 to 1916 compensation was awarded to men at quite as early a stage as at present. A large class of the miners now detected had begun work about 1912 and 1914. Diagnosis was based on the correlation of radiological and clinical evidence with the post mortem, and improved radiological technique alone could not, therefore, upset the balance. 256 cases were detected in 1930 as against 800 to 900 cases in 1912 to 1916. He did not believe as many ante-primary cases were now being made. The old Rand miners still accounted for 70 per cent. of the silicosis, but there was an improvement in the value of their production rates of 30 per cent. He did not agree that as much silicosis was made as ever but taking longer. Nor did he consider that the potentialities of their present methods had been exhausted.

Mr. Spence Fraser: The number of ante-primary cases last year was the same as the average for 1920 to 1923. The rates had decreased, which meant that the duration of working life was much greater, and that a larger number of miners got silicosis in later years. The mining population was much steadier and although there was the same number of cases the production of them was deferred. He thought that an improvement could be deduced from the figures given in his and Dr. Irvine's paper.

(The Conference adjourned at 1 p.m.)

NINTH SITTING

Wednesday, 20 August 1930, 2.30 p.m.

Chairman: DR. L. G. IRVINE

Dr. Mavrogordato showed lantern slides illustrating silicosis and tuberculosis in guinea-pigs and human beings.

STATISTICS OF SILICOSIS (*continued*)

Mr. Spence Fraser: The method of measuring silicosis production could be compared with the method of measuring mortality in a population. In the latter case the rate of mortality at any age was derived by comparing the deaths at that age with the population living at that age. In dealing with silicosis the number of miners working in each respective year of underground service was substituted for the population existing at each age, and certifications in the ante-primary stage were substituted for deaths. The methods were analogous. If the rates of mortality in a population were lower in any period than during a previous period, it was recognised that the mortality had fallen and that there had been an improvement in the position. In the same way, they found in South Africa that the rates of production of silicosis were lower to-day than at any previous period, and they had no hesitation in saying that the position had improved.

It was true that the annual number of new cases of silicosis to-day was about the same as in 1920-1923. A similar phenomenon was found in mortality statistics; the death rates could decrease, while the total number of deaths actually increased, because there was a different age-distribution of the population, many more being alive at old ages where the death rates were highest. The same thing had happened with regard to silicosis, since there had been a large increase in the number of miners working after long periods of underground service where the rates of production were highest. The true measure of the production of silicosis lay in the production rates showing the proportion of miners contracting silicosis in each year of underground service, and these rates showed a very substantial reduction in the incidence of silicosis.

Dr. Orenstein: The discussion had shown that the statistical methods of the Rand were not sufficient to give the facts which practical hygienists wished to know. Professor Hall's resolution aimed at closer and more detailed study in order to find out which factor contributed most to reduction. The present statistics did not give the fundamental data and he was not satisfied that they demonstrated a reduction in silicosis.

Dr. Cunningham requested information as to the number of cases of ante-primary silicosis among miners employed since the beginning of rigid initial examination, in whom tuberculosis had developed. If the numbers were large enough it might give some indication of the value of the initial physical examination.

The Chairman: Professor Hall's motion would be referred to the reporters.

LEGISLATION AND COMPENSATION

The Chairman: South African legislation had the peculiarity that : (1) the duration of time to render a miner eligible for a claim was very elastic; it was two years, with the reservation that the Board might allow a shorter period to count; (2) there was no restriction as to the date when the work had been carried out; (3) there was no restriction as to the amount of work done in other mines in South Africa or elsewhere before or after the work on the Reef.

Cases of ante-primary and primary silicosis received lump sum compensation, while grave incapacity or tuberculosis was pensionable. The ante-primary cases sometimes had disability up to 25 per cent. Primary cases could do moderate physical work and the secondary cases light work or none.

Dr. Middleton: While South African legislation made no provision for suspension in the early stage, under certain schemes in Great Britain any degree of silicosis was liable to immediate and total suspension, whether there was disablement or not. He asked, however, if this was justified or whether suspension would arrest the development of the disease. In some highly skilled industries, provision was made under special schemes to allow men with more than twenty years' work, or who were over forty years of age, to remain at work; this was done for economic reasons. The special schemes for silicosis were not all the same. Under the Refractories Industries and Sandstone Industry Scheme there were periodical examinations with compensation for partial or total disability or for death. Under the other schemes there was no periodical medical examination, but they followed the procedure under the Workmen's Compensation Act for occupational diseases, though they covered only total disablement or death, owing to the difficulty of recognising the disease in the earlier stages.

The Chairman: The ante-primary stage had been introduced in 1919 to secure the suspension of silicotic workers, at the earliest detectable stage of the disease in the hope that such cases would be non-progressive. That hope had not been realised. It was impossible to say that ante-primary cases would not progress; although they lived longer they reached the second stage faster than did the old primary cases.

Dr. Truter: Suspension from work lowered the worker's standard of living, and he therefore deteriorated more rapidly. A miner who continued to work lived as long or even longer.

Dr. Orenstein: Other employment could be found in an industrialised country but was impossible in South Africa. The ante-primary man had to choose between continuing at work with no increased compensation and receiving a lump sum (about £500) and finding other employment, which was difficult to discover at a wage equivalent to that gained by a miner. Their economic status was therefore lowered. He asked whether compensation really discharged the onus which lay upon society and whether the expenditure on compensation was applied to the best advantage.

Dr. Badham: He hoped the Reporters would frame resolutions to give guidance on the framing of compensation legislation.

Professor Hall: No single line of legislation or compensation could apply to all classes. In Great Britain, if there was no trade depression, a suspended man was able to find equivalent employment in some

other trade. Cases in the ante-primary stage which had been followed up had been able to carry on at work for five years. Skilled workers such as potters who had primary silicosis, with some degree of incapacity, were capable of doing their full work. Potters could not be treated in the same way because if they were suspended they could not find other work which was economically as good.

Mr. Du Toit: A number of South Africans had worked underground for two years or less in the scheduled mines and had subsequently been engaged in agricultural work. These men had never been out of South Africa and subsequent to their underground service in the gold mines had never done any other underground work, but nevertheless developed silicosis. For this reason the elastic period of two years' underground service, referred to by the Chairman, had been introduced in South African legislation. These men obviously had to be compensated. A number of men who had previously been miners in South Africa had during the past five years benefited under the South African Miners' Phthisis Act. These men had also done mining work elsewhere. It was an exceedingly difficult matter definitely to say where the disease had actually been developed. Then again, numbers of silicotic beneficiaries had left South Africa and had entered other mines and dusty occupations outside the Union of South Africa.

Silicosis had been found in men who worked in the Cornish tin mines, in the Welsh coal mines, the Cumberland iron ore mines, mines in Canada, in Australia and New Zealand. Miners and beneficiaries who had been employed there should have no claim on the funds of South Africa. Attempts were now being made to restrict South African ex-miners and beneficiaries under the Act who had subsequently engaged in mining work elsewhere from having any claim for compensation or further compensation, as the case might be, on South African Funds.

In regard to the question of rehabilitation, this was a most difficult matter because South Africa was a young country with few industries and it was not easy to find other employment for silicotic beneficiaries. South Africa was not essentially a great agricultural country in spite of what people might say. It was highly mineralised, and base metal mines absorbed a certain number of silicotics.

Then there was another important factor in South Africa with which other older countries had not to contend, namely, the enormous native and coloured population. This also made it very difficult to draft silicotics into other suitable occupations. It was impossible for silicotics to make good on the land as agriculturists since they could not stand exposure to all kinds of weather. He believed this had also been the experience in Australia, Canada, the United States and the United Kingdom. Road construction was also unsuitable. Asbestos mining was now known to be fatal and, in South Africa at all events there were very few other outside mines which could absorb all their silicotics. On the one hand, if silicotics were allowed to remain in their underground occupations without any restrictions, the position might be reached after a period of years when the gold mining industry would be entirely run by employees who were silicotics and this in the end was bound to affect efficiency. On the other hand, no State could be expected to legalise what amounted to slow suicide.

If continued employment underground was not dangerous to silicotics, then the South African legislation should be altered, but in the absence of reliable data there was no conclusive evidence either one way or the other.

A large number of South African silicotics had been placed in other suitable employment, but such employment was not always of a permanent nature. Then there were some beneficiaries who were unemployable. This class one naturally found in all sections of the community; it was a class which might be ruled out as not fit for employment. This class the South African Legislation never intended should be given employment unless of a very light nature and it consisted of men in the worst stage of silicosis or suffering from silicosis with tuberculosis. Beneficiaries belonging to this class were in receipt of monthly life pensions with inclusive allowances for their wives and children. After the death of a life pensioner no post-mortem or certificate as to the cause of death was required. The dependants were entitled to life pensions *ipso facto*.

Dr. Fisher: Alternative employment was impossible for coal miners. Colliers lived in small villages, and if they were unemployed their condition tended to become worse in a general way, as miners' nystagmus had shown, although of course neurasthenia was not part of silicosis as it was in miners' nystagmus. Light employment such as repairs on the main haulage roads at an agreed standard living wage was desirable.

Dr. George: At Broken Hill the medical authority was a Board which consisted of an independent chairman appointed by the Government, one doctor appointed by the employers, and one doctor by the workers. Medical benefits were given for pneumoconiosis and/or tuberculosis up to £125 per man. The Act was administered by a joint committee, two of whom were appointed by the workers and two by the employers, with an independent chairman, who was usually a stipendiary magistrate (a Government official). This committee had no power to interfere with the medical certificates. The condition of the miner had to be certified as reasonably attributable to employment in the Broken Hill mines. They were withdrawn in the ante-primary stage, but the Committee had to find work for them. Up to the present it had never yet been done, and silicotics therefore drew a full pension in many cases with no incapacity. At Broken Hill in case of death from any cause whatsoever the widow received a pension of £2 10s. a week, and children under the age of sixteen of 8s. 6d. per week.

At the start many men had not sustained injury from Broken Hill and the State therefore contributed half the amount, and the employers the other half. When injury was attributable to Broken Hill, the employer found the whole sum. Upon leaving the industry a man had to be examined by the Medical Bureau, and he could come up for examination at any time within the five years following.

Dr. Badham: In New South Wales there were three Acts, namely: (1) the *Broken Hill Act* which dealt with a special mining district; (2) the *Workmen's Compensation (Silicosis) Act*, which dealt with Sydney sandstone workers—a beneficiary might receive a maximum of £3 a week up to a total of £750, but there was no payment to dependants except in case of death; (3) the *Workmen's Compensation Act* (1926), which granted compensation for fibrous pneumoconiosis except at Broken Hill, with a maximum of £5 a week up to a total of £1,000. Diseases caused by silica dust were however excluded and despite the three Acts in New South Wales dealing with compensation for dust diseases of the lungs there was still a number of workers who were not protected.

Dr. Moore: In Queensland the Workmen's Compensation Act covered pneumoconiosis and silicosis. In Victoria and South Australia

silicosis was not included. In Tasmania, a few shillings a week were paid for silicosis. In Western Australia the Miners' Phthisis Act excluded tuberculosis or silicotic men from employment in the mines, but granted no compensation for silicosis. The Commonwealth Government paid a pension of £1 per week for total incapacity.

Dr. Cunningham: Disability and expense might be reduced if it were possible in industries in which silicosis develops rapidly, to remove men from exposure to silica without waiting for specific indications of silicosis and therefore without compensation, at some unknown point before the ante-primary stage is reached.

It would be worth a considerable expenditure to determine when tuberculosis becomes a more serious menace in a lung containing silica. A reduction of two or three years below the average exposure necessary for the development of silicosis would not materially increase labour turnover.

Mr. Barry: Silicosis in South Africa had given rise to nine Acts, four commissions, and many select committees. The average amount of compensation paid to a married man was £3,500. Up to October 1929, 7,633 beneficiary miners were alive; 2,271 of these were in the ante-primary stage, 2,306 in the primary stage, and 2,184 in the secondary stage. There were 2,014 widows and 3,538 children in receipt of pensions.

All this legislation was unfortunately retrospective, and it laid a heavy burden on employers, since many mines had shut down, and paid no retrospective compensation. The legislation contained anomalies such as the semi-compulsion upon a miner to give up underground work, but the lack of any regulation to prevent him taking up mine work elsewhere. Any miner who had been employed two years underground could work in another silicotic occupation and yet receive benefits. Two years' service at any date counted.

It was generally agreed that underground conditions had enormously improved in the last twenty years. The working life which had once been seven or eight years was now twenty years. The new Rand miner had a working life of about twenty-three years, notwithstanding which claims upon the Fund had increased. This was said to be due to the large number of old Rand miners, and to improved methods of diagnosis which kept the cases lower. It was also said, however, that the disease had become more progressive.

Under the Act of 1919 some mines closed down and were compelled to pay only a small portion of their liabilities. The Board collected about £1,000,000, all of which was given to old cases under the 1925 Act. The real cost of miners' phthisis was said to be £1,000,000 per year. The Government Actuary estimated the outstanding liability of the mines at £6,400,000.

When the 1925 Act was passed provision was made for each mine to set aside a sum annually to meet these liabilities. The cost to the mines of miners' phthisis was 7s. 6d. per underground European shift. The South African Act was the most liberal in the world, and the only Act that could be compared with it was in Ontario, where £100 was paid for ante-primary silicosis, £200 for primary silicosis and a pension of about £15 per month. It was impossible to alter the South African legislation without upheaval, because any new benefits granted produced thousands of retrospective claimants.

The Chairman: In South Africa there was no general national insurance Act for invalidity, and there was therefore a tendency to

treat the Miners' Phthisis Act as a kind of substitute for the Poor Laws. The question of compensation for simple tuberculosis might be referred to the Reporters. The cases which the Medical Bureau had certified as "simple tuberculosis" and which came to post-mortem were found in 50 per cent. of the cases to have a silicotic element. It was also desirable to remove simple tuberculosis cases from the mines, because of the risk of communication of infection to others.

Mr. Du Toit: The question whether compensation should be computed on a wage basis or on a flat rate should be referred to the Reporters.

Dr. Orenstein: Legislation had to differ according to the industrial conditions of various countries. The South African situation was quite peculiar, because the gold industry was not subject to the fluctuations of other industries, but was handicapped by the mental attitude of legislators to gold. The legislation on pneumoconiosis was extraordinarily illogical. The Reporters might consider whether any standard conditions could be applied to this disease throughout the world. Great weight was given in legislation to the necessity of fixing compensation for silicosis sufficiently high to stimulate prophylactic measures. The Reporters might consider whether silicosis could be called preventable. It was a misconception that removal from work in the earliest possible stage would prevent further development; if it was true that continued employment was really slow suicide, no man should be allowed to work in any dusty occupation at all. He proposed the following resolutions:

1. That endeavour should be made to deal with silicotics by rehabilitation, diverting for that purpose a portion of the funds made available under present conditions for compensation. Such rehabilitation schemes to be directed by specially constituted governing bodies.

2. That there appears to be reason to consider that there are no good grounds for compulsory removal of silicotics from their customary employment, unless such removal does not tend to lower markedly the economic level of livelihood of the silicotic or until his earning power has reached the level approximating that of the compensation.

3. That removal from employment of men in the fifth and sixth decades of life is generally inadvisable, until a high degree of incapacity is present.

(The Conference adjourned at 4.50 p.m.)

TENTH SITTING

Saturday, 23 August 1930, 9.30 a.m.

Chairman: DR. L. G. IRVINE

DRAFT REPORT ON PREVENTIVE MEASURES

The Conference considered the Draft Report on Preventive Measures and adopted it with the exception of Recommendation 4, after making various amendments.

(The Conference adjourned at 1.35 p.m.)

ELEVENTH SITTING

Monday, 25 August 1930, 9.30 a.m.

Chairmen: SIR SPENCER LISTER (Vice-Chairman) and DR. L. G. IRVINE

ARRANGEMENTS OF BUSINESS

Mr. Phelan proposed that the closing sitting of the Conference on Wednesday, 27 August, should be open to the Press after a certain hour, and that the recommendations adopted by the Conference should be communicated to the Press.

The Conference approved of this procedure.

THE MEDICAL ASPECTS OF SILICOSIS

The Conference proceeded to discuss the *Report upon the Medical Aspects of Silicosis, including Ætiology, Pathology and Diagnostics*, submitted by Drs. Gardner, Middleton and Orenstein. The Report was as follows:

The Reporters, Drs. L. U. Gardner, E. L. Middleton and A. J. Orenstein¹, beg to submit the following:

1. The Conference confined its discussion almost entirely to silicosis, as the other pneumoconioses are, with the possible exception of asbestosis, in the present state of available information, of less importance, and furthermore have not been subjected to sufficiently detailed study.
2. Silicosis is a pathological condition of the lungs due to inhalation of silicon dioxide. It can be produced experimentally in animals.
3. To produce the pathological condition, silica must reach the lungs:
 - (a) in a chemically uncombined condition, although the dust inhaled may be either a natural mixture of silicon dioxide with other dusts, such as occurs in granite, or an artificial mixture, such as scouring powder;
 - (b) in fine particles of the order of less than ten microns. There is no evidence as to the lowest limit of size in which the particles may be capable of producing the disease;
 - (c) in sufficient amount, and over a certain period of time; these two factors are reciprocal variants. The minimum of these two respective factors has not yet been determined.
4. Silica dust plays the dominant role in the production of silicosis, admixture of other dusts tending to modify the picture in the direction of that of other pneumoconioses, in some relation to the proportion of free silica inhaled.
5. There is experimental evidence that the solubility of silica in the tissues is an essential factor in the causation of silicosis.

¹ Dr. W. Steuart was unable to participate in the preparation of this report. He submitted a memorandum which is reproduced later as an Appendix.

6. Infection of the lung with *B. tuberculosis* or other pathogenic organisms, whether it occurs before, simultaneously with, or subsequent to the development of silicosis, alters the disease and influences it unfavourably, tuberculous infection being particularly unfavourable.

7. The establishment of a silicotic process in a lung renders the subsequent inhalation of other dusts, in themselves relatively innocuous, capable of producing serious pneumoconiosis.

8. It was suggested that intermittency of employment retards the onset of silicosis, but the evidence adduced in support of this, though suggestive is not conclusive, when the total period of exposure is not affected.

9. It was agreed that the microscopic pathological changes which may be produced by the prolonged inhalation of silica dust are:

- (a) The development of a condition designated in South Africa as a dry bronchiolitis, characterised by an accumulation of dust filled phagocytes in or in relation to the terminal bronchioles, with possibly some desquamation of their epithelium.
- (b) The accumulation of dust-containing phagocytes about and in the intra-pulmonary lymphoid tissue, and their transportation through the lymphatics into the tracheo-bronchial lymph nodes.

(The conditions described above under (a) and (b) do not constitute the disease silicosis.)

- (c) The gradual development of fibrous tissue within such accumulations of phagocytes and the formation of characteristic nodules of hyaline fibrous tissue.

- (d) Degenerative changes in these foci.

- (e) The hyaline nodules increase in size by extension at their periphery.

Coalescence of adjacent nodules takes place and brings about involvement of further areas of the lung.

(The conditions described under (c), (d) and (e) constitute the disease silicosis.)

10. Macroscopically the changes observed in silicosis are:

- (a) *In the early stage.* A variable number of palpable pearly-white nodules up to 2 or 3 mm. in diameter on the pleural surface of the lung. On section, the cut surface of the lung is studded with pigmented foci, widely scattered, a moderate proportion of which are only just palpable. The tracheo-bronchial lymph nodes are slightly enlarged and deeply pigmented, and may exhibit foci of fibrous induration.

- (b) *Later stages.* The fibrotic nodules are increased in number, size and density, and coalescence of these may be found. The portion of the lung between the fibrotic nodules may be emphysematous. The tracheo-bronchial lymph nodes may be smaller in size than those seen in the early stage and are fibrosed.

11. The presence of tuberculous infection usually modifies the pathological appearance. Special attention was drawn to the three following types:

- (a) In which the picture of silicosis above described may be little, if at all, modified, but in which only a biological test can demonstrate the presence of *B. tuberculosis*.

- (b) In which the coexistence of silicosis and typical tuberculosis lesions is easily recognisable.
- (c) In which the presence of tuberculosis is easily recognisable, but the existence of silicosis is more difficult to determine.

12. There is evidence that with *B. tuberculosis*, *in vitro* the period before growth becomes apparent is shortened in the presence of silica, and that *in vivo* an environment favourable to the continued growth of the bacillus is produced in the presence of silica, but the virulence apparently remains unaltered.

13. In *massive* silicosis cardiac hypertrophy and subsequent dilatation may occur. In silicosis with infective processes, cardiac changes may also occur.

14. No evidence was adduced in regard to involvement of kidney or liver.

15. For the diagnosis of silicosis as a disease it is necessary to take into consideration:

- (a) the employment history;
- (b) the symptoms and physical signs;
- (c) the radiological findings.

16. The disease can conveniently be divided into three stages, designated "first", "second", and "third" stages.

17. In the differential diagnosis of silicosis from other pneumoconioses a history must be established of exposure to inhalation of silica dust in a quantity reasonably commensurate with the clinical and radiological findings.

18. In the "first stage" symptoms referable to the respiratory system may be either slight or even absent. Capacity for work may be slightly impaired. There may be a departure from the normal in percussion and in auscultatory signs, and the radiograph must show an increased density of linear shadows, and the presence of discrete shadows, indicative of nodulation.

19. In the "second stage", there is an increase of the physical signs observable in the "first stage", and the radiograph shows an increase in the number and size of the discrete shadows indicative of nodulation with a tendency to their confluence. There must be some degree of definite impairment of working capacity.

20. In the "third stage" all the above conditions are grossly accentuated and indications of areas of massive fibrosis are usual. There is serious or total incapacitation.

21. Pulmonary tuberculosis may be present in any of the above described "stages" of silicosis, altering the symptoms, physical signs and radiographic appearances, and the degree of working capacity. Its presence must therefore influence the "stage" classification of the individual, which classification must in these circumstances be based more on the degree of loss of working capacity than on physical signs and radiographic appearances.

22. Radiographs may frequently be met with which show a slight, moderate, or well marked increase beyond the normal in radiating linear shadows. These may or may not be due to fibrosis.

23. The inhalation of asbestos dust produces a definite pneumoconiosis, which may occur also in association with tuberculosis, and deaths have been recorded.

This pneumoconiosis is associated with the presence in the lungs of "asbestosis bodies", but the mere presence of these bodies in the lungs or sputum does not constitute evidence of the disease.

For the diagnosis of this pneumoconiosis the same criteria as described for silicosis should be applied, *mutatis mutandis*.

There is not at present sufficient evidence to show definitely to what extent tuberculosis and this type of pneumoconiosis react upon one another.

24. There are other dusts, such as those from marble, coal, carborundum, etc., which may contain small quantities of silica and which produce demonstrable lung changes, radiographically resembling in some cases the appearances observed in early silicosis.

There is not a sufficient body of evidence available to enable a definite statement to be made of pathological changes in man. In animal experiments, the inhalation of carborundum dust over a period of four years has produced fibrosis only in the tracheo-bronchial lymph nodes; the lungs were entirely free of fibrotic changes. This, and collateral observations on inhaled granite and asbestos dusts suggest the hypothesis that to produce pulmonary fibrosis a sufficient concentration of a relatively insoluble dust must be brought by the activity of phagocytic cells into intimate contact with connective tissues. With the dusts last mentioned the migration of phagocytes, for at least a prolonged period, is ineffective in establishing such contact in the lung. Only in the tracheo-bronchial nodes are these conditions realised during a period of four years.

25. The Reporters beg to recommend that appropriate action be taken:

- (a) To establish an international classification of silicosis on the lines indicated in paragraphs 16 to 21 inclusive.
- (b) To enquire into the possibility of establishing an internationally comparable technique of radiography, and terminology of radiographic findings.
- (c) To institute further studies in the correlation of radiographic appearances, morbid anatomy and symptomatology of silicosis and silicosis with tuberculosis.
- (d) It is desirable that further scientific research into the ætiology, pathology and diagnosis of silicosis and other dust diseases should be undertaken on an international basis, at an early date.

APPENDIX

Memorandum on Radiography of Silicosis

By Dr. W. STEUART

With a view to obtaining uniformity in films of the thorax in cases of silicosis a description of the technique used at the Miners' Phthisis Bureau is given in detail.

The transformer receives three phase current and the secondary current is rectified by means of six valve tubes. Its output is much in excess of the capacity of present X-ray tubes.

The X-ray tube used is a metallic DN type. The distance from focus to film is 48 inches.

The length of exposure and penetration vary with the antero posterior thickness of the examinee according to the following table:

Depth of thorax in inches	Time in seconds	Kilovolts
7	0.1	61
7½	0.11	61
8	0.12	61
8½	0.15	62
9	0.175	62
9½	0.2	63
10	0.22	63
10½	0.23	64
11	0.24	64
11½	0.25	65
12	0.26	65
12½	0.26	66
Filament current 4.3 amperes	Current through tube 200 M.A.	

The man lies in the prone position on the cassette with the tube above him.

Kodak films and Agfa intensifying screens are employed. If other makes of screens and films are preferred allowance should be made in exposure and penetration as films and screens differ considerably as regards speed.

Cassettes are such that uniform contact between films and screens is secured.

The developing solution is made up according to Kodak's formula, but to every 5 gallons 4 lbs of sodium carbonate and 4 lbs of sodium sulphide are added.

The temperature of the developer is 65° F. and the time of development is five minutes.

Terms used in indicating the diagnosis on X-ray report form:

1. *Normal thorax.* — This is taken as that of a healthy youth of about eighteen.

The heart occupies a left medial position and is approximately triangular in shape, its shadow accounts for about one third of the area of the thoracic shadow. Its size and shape vary.

The right hilus is seen as a faint rather small shadow but the left is hidden by the heart.

The diaphragm is dome shaped on each side, the right being higher than the left. Its level is variable.

The pulmonary tissues throw no shadows so that between the ribs the skiagram should be uniformly clear.

Radiating from each hilus faint shadows can be made out due to the roots of the bronchial tree of bronchi vessels and supporting tissues.

The costal cartilages are transradiant.

2. *Rather more fibrosis than usual.* — This is really the normal thorax and is seen in healthy adults, but through the continuous inhalation of dust, smoke, etc., the hilus and bronchial tree shadows become accentuated, otherwise the picture is that of a normal thorax.

3. *More fibrosis than usual.* — This is an extension of the previous condition. The hilus shadows are denser and the bronchial tree shadows are more numerous and found throughout the thorax. It is seen in old people, in cases of chronic bronchitis, asthma, and in old healed infections. Though not an absolute bar to underground work it is a factor that goes far in deciding the rejection of an applicant for mining work.

Then come a series of classifications of conditions that arise from gold mining, viz.:

4. *Commencing generalised fibrosis.*

5. *Moderate generalised fibrosis.*

6. *Well marked fibrosis.*

7. *Very well marked fibrosis.*

8. *Gross fibrosis.* The commencing generalised fibrosis shows itself on the Witwatersrand after five or more years depending on the particular work done and the idiosyncrasy of the miner. It is a further accentuation of "more fibrosis" than usual and here and there small pin head shadows can be made out, the first indication of the typical "mottling" of silicosis.

Thereafter the mottling is common to all the others, the chief indication of the amount of fibrosis being the size of the nodules.

The nodule or unit of mottling varies from about one twentieth of an inch (1.25 mm.) in diameter in moderate generalised fibrosis to about a quarter of an inch (7 mm.) in gross fibrosis. These figures are of course merely approximate and not absolute.

9. *Fibrosis partly or mainly silicotic in type.* — This is deduced when the nodules are clearly cut in outline and uniform in distribution throughout the lung.

10. *Fibrosis partly or mainly infective in type.* — A slow infective process has the effect of increasing the size of the nodules in certain areas so that there is no uniform distribution as in pure silicosis. The outline of the nodules becomes fluffy instead of sharply defined. The usual infection is tuberculous, but syphilis and other chronic infective process may cause the same appearances.

11. *Appearances suggestive of tuberculous, $\frac{\text{right}}{\text{left}}$ lung.* — A sudden increase in the density of the hilus shadows, increased apical density or patchy shadows in other lung areas lead to the initialing of this item in the report.

12. *Apparently definite tuberculosis, $\frac{\text{right}}{\text{left}}$ lung.* — Tuberculosis is often so well established in periodical cases that there is practically no doubt as to the meaning of the shadows shown. The appearance can best be described by imagining some French chalk being thrown on the film and then rubbed round with the fingers. In the shadows produced dark areas of varying size may possibly be seen due to cavities.

The word "apparently" is retained because occasionally syphilis causes much the same kind of shadow.

13. *Peribronchial thickening; hilus thickening.* — From the experience obtained since the commencement of examining individuals

who wished to take up mining, it has been found that the accentuation of the shadows cast by the larger branches of the bronchial tree (called by the Bureau "Peribronchial thickening") and of the hilus shadows is a definite indication of tuberculous susceptibility and examinees are frequently rejected on this account.

14. *Pleural thickening* $\frac{\text{right}}{\text{left}}$ side.

15. *Pleural effusion* $\frac{\text{right}}{\text{left}}$ side.

Both of these conditions are readily noted by a radiographical examination and are considered important on account of their possible association with tuberculosis.

16. *Consolidation* $\frac{\text{right}}{\text{left}}$ side. — This diagnosis is given in the case of any dense shadow that does not alter its size and shape in different positions of the subject. The causes of consolidation are so numerous that unless there is some special indication in size, shape or position no diagnosis is attempted except possibly a query.

17. *Heart, asthenic or vertical in type.* — Although the heart shadows vary to a considerable extent within normal bounds, individuals having a tubular shaped heart or one casting a very small shadow, are regarded as "suspect" by the members of the Bureau for the same reasons noted in 13, 14, and 15.

18. *Heart enlarged.* — In most cases of cardiac disease and pericarditis the heart shadow usurps more than its normal share of the thoracic space, and attention is always drawn to this variation, as cardiac disability is often ascribed by outside medical practitioners as being due to silicosis.

19. *Aorta enlarged and aortic aneurysm.* — These items have frequently to be initialed and are often associated with patchy shadows suggestive of tuberculosis which however disappear after a course of appropriate treatment for specific disease.

20. Other changes, viz.:

(1) *Aerophagy.* — Occasionally at the left base it will be found that the phrenic shadow is pushed up into the thorax and that below it a negative shadow is shown. This is due to the subject swallowing air which distends the stomach and may cause discomfort.

(2) *Aneurysm of heart.* — The condition is very rare, only one case in 80,000 persons having been noted at the Bureau.

(3) *Atelectasis.* — Due to obstruction of a bronchus, gives a fan-shaped shadow radiating from direction of lung root which is as dense as a consolidation shadow.

(4) *Azygos lobe.* — In about one case in twenty thousand there is an abnormal azygos vein which leads to a shadow in the upper lobe of the right lung.

(5) *Bronchiectasis.* — This is indicated in skiagrams by No. 13, "peribronchial thickening". A definite diagnosis however can only be given after a lipiodol injection.

(6) *Bronchitis.* — Chronic bronchitis gives the picture described in No. 3, "more fibrosis than usual".

(7) *Calcification*. — Calcium is deposited in old inflammatory foci and in costal cartilage. It is often deposited in silicotic nodules and in lymphatic glands at the root of the lung. Its presence is indicated by an increased density of the shadow.

(8) *Carcinoma*. — Carcinoma may occur as a primary or secondary condition.

In the former case it leads to a consolidated area which increases in size. In the latter several shadows are seen which are circular and become larger and larger in diameter until the patient succumbs.

(9) *Emphysema*. — The lung tissue becomes more transradiant so that other shadows are accentuated owing to the increased contrast.

(10) *Empyema*. — The shadow is very dense and may occupy any part of the lung field.

(11) *Hodgkins disease*. — The thoracic involvement causes a dense mediastinal shadow.

(12) *Hydatid cyst*. — This is indicated by a dense oval shadow with a uniform contour.

(13) *Liver and subphrenic abscess* normally occur on the right side. The diaphragm is pushed up into the thorax.

(14) *Miliary tuberculosis*. — The appearance is practically the same as a moderate generalised fibrosis mainly silicotic in type.

(15) *Pneumonia*. — The shadow is dense and varies in size according to the severity of the disease.

(16) *Pneumothorax*. — The collapsed lung can be seen in the root area. It is surrounded by a uniform negative shadow stretching to the periphery.

The Conference adopted the Report.

The Conference decided that the Memorandum submitted by Dr. Stewart should be appended to the text of the Report.

Dr. Loriga dissented from the proposal which the Conference adopted to describe paragraph (a) and (b) of Clause 9 as conditions which did not constitute the disease silicosis, and paragraphs (c), (d) and (e) as constituting the disease silicosis, on the ground that these notes should properly be inserted in the Report on Compensation, since arbitrary legal limits might be laid down between the various pathological stages, while pathology must insist on the progressive nature of the evolution of the disease and could not properly indicate hard and fast divisions between its different stages.

The Chairman thanked the Reporters for the work which they had so ably performed.

(The Conference adjourned at 12.45 p.m.)

TWELFTH SITTING

Tuesday, 26 August 1930, 9.30 a.m.

Chairman: DR. L. G. IRVINE

PREVENTIVE MEASURES

The Conference considered the Revised Report of the Sub-Committee on Preventive Measures and adopted it. The Report is given below.

The Sub-Committee, consisting of Dr. Loriga, Dr. Badham and Mr. Roberts, appointed to deal with questions raised under this heading, begs to report as follows:

1. The Conference dealt with the matter of prevention at its sessions held on Friday afternoon and Saturday morning, 15 and 16 August 1930.

The first six papers and the papers presented by the visiting members, were taken as read, and discussed on broad lines, members having remarks to make calling freely upon their experience in regard to one or other of the various aspects of the question.

The feeling of the Conference was that the present opportunity should be used for an interchange of ideas with a view to mutual inspiration which would be of value in future research, rather than that it should be used for the purpose of arriving at conclusions and the making of recommendations.

2. It was generally agreed that so far as the present heading is concerned, the disease which it is sought to prevent is that which arises from the inhalation of free silica (SiO_2) as distinct from silica in chemical combination with other substances.

3. From the information supplied by various members, the disease becomes noticeable after widely differing periods of exposure to siliceous dust, depending apparently, upon:

- (a) The amount of dust inhaled;
- (b) the percentage of free silica contained therein;
- (c) the size-frequency (or fineness) of the particles inhaled;
- (d) the nature and sort of such other substances (including vapours and gases) as may be inhaled simultaneously, or otherwise;
- (e) the powers of resistance of the individual concerned;
- (f) the presence or absence of a complication by an infective process.

In regard to (a), it was agreed that by the use of water and other preventive measures the dust contents of air can fairly readily be reduced to ordinarily invisible amounts. In Australian experience this represents something in the neighbourhood of 4 or 5 milligrams per cubic metre, or say 400 or 500 particles per cubic centimetre when the particles are from 1 to 10 microns with a size-frequency ratio of 3. It was, however, evident from the discussion that it is impossible, under existing conditions, properly to correlate dust determinations made in different countries, in different industries and for different purposes, as well as for different immediate objects.

With regard to (b), it appeared from the information placed before the Conference that silicosis can be contracted through inhaling for

a sufficient period dust containing percentages of silica varying from say 95 per cent. down to from 30 to 35 per cent. and even lower.

In regard to (c), it was pointed out that with existing preventive measures carried out in certain mining areas there are now relatively few large particles in the air; and it appeared from the discussion that the greatest amount of harm is done by particles of less than say 3 microns in size. Some of the evidence seemed to suggest that particles of an ultra-microscopic size are factors in the causation of the disease, but evidence in this direction was not conclusive.

In regard to (d), the experience on the subject of certain members went to show that while exposure to various other dusts *simultaneously* with silica might affect the development of silicosis, the suggestion that other dusts might be used as an antidote against silica should be treated with great caution and reserve. Further research in this direction is urgently called for. It was pointed out that experimental evidence and practical experience under working conditions had shown that prior or subsequent inhalation of other dusts in no way delayed the development of silicosis.

In the course of the discussion under this heading some reference was made to the alleged immunity from silicosis in some districts where quartz in company with non-siliceous rock is mined, but it was pointed out that further investigation had shown in the one case that the allegation was unfounded in that the existence of silicosis had been obscured by the migratory nature of the working population; while in other cases it appeared that by reason, possibly, of the absence of laws relating to compensation, the medical evidence is not so complete as it might otherwise be, and there is sufficient room for doubt as to the exact position of the workers vis-a-vis silicosis. In all cases where there are laws relating to the compensation of silicotics it is but natural that the examination of the workers will be more thorough.

In regard to (e), it was generally agreed that this is an important feature, and there was a certain consensus of opinion that alternative employment and periods free from exposure to siliceous dust tended to increase the resistance and thereby delay the development of silicosis.

4. The discussion on methods for the prevention of dust and the inhalation thereof fell, on broad lines, under the following headings:

- (a) the use of water;
- (b) exhaust draught applied at or near the point of origin of the dust;
- (c) dust traps and masks;
- (d) ventilation;
- (e) other methods.

There was something said in favour of each of the methods referred to. It was agreed that no one method is applicable in all circumstances, but that in most cases, and especially in mining, there should be a combination of methods.

With regard to water, it was pointed out that as far as the Witwatersrand was concerned, it is used in three different ways, namely:

- (1) to prevent the formation of dust during the drilling of holes, in blasting, and the handling of broken rock;
- (2) for the wetting of all surfaces with a view to securing a "fly paper" effect in retaining dust which might settle on those surfaces;
- (3) for spraying into the air in order to allay dust which had been formed.

In regard to (1) it was generally agreed that the application of water at the site of percussion or fracture tends to minimise the formation of dust, but attention was drawn to the fact that in several operations, e.g. rock drilling, stone cutting, grinding, etc., sparks accompanied by dust escape even when the surfaces concerned are actually under a film of water.

In regard to (2), the view was expressed that since there is no particular reason why dust particles of the order of less than 3 microns should settle on the roof and sides of working places, and that they would settle on the floor only after many hours, the value of these wetted surfaces as dust catchers is probably small.

With regard to (3), it was pointed out that the dust particles with which the Conference was concerned are of the same order in size as micro-organisms, and that no one nowadays would expect to catch micro-organisms by means of a spray. In this connection it was mentioned as a matter of interest that Lord Lister, in his famous address delivered at Berlin, had stated that he felt ashamed of ever having suggested such a possibility in surgery.

The consensus of opinion was that as sprays are of little value for removing fine dust from the air and that since, further, a humid atmosphere and the presence of droplets had been shown experimentally to increase the risk of various infections their use should be restricted.

This view, however, does not necessarily apply to water blasts used on the Witwatersrand when firing in development ends, since while such blasts might not catch much of the finer dust (except by the subsequent condensation of water vaporised by the heat generated in blasting) they put into solution some of the noxious gases and wet the broken rock so as to prevent the escape of the dust when that rock comes to be handled.

In regard to (b), it was mentioned that exhaust draught was of great value in those processes of manufacture where there is an objection to the use of water. In some cases water cannot be used for fear of spoiling the material, and in other cases the workmen at times turn it off because it makes them wet. In all such cases, exhaust hoods should, if applicable, be used. It is necessary, however, that these hoods should be placed in close proximity to the work, and that regard should be had to the direction and speed of rotating objects. As an example of what could be done in manufacturing processes by the use of exhaust draught, cases were mentioned of a decrease in the incidence of silicosis which had followed the abandonment of wet grinding in favour of dry grinding with suitably applied exhaust draught. It was also pointed out that before exhaust draught was used for the dry grinding of metals this process was much more dangerous than wet grinding, but that since the introduction of efficient exhaust draught with dry grinding the position had been reversed.

(1) *Dust traps.* — As an example of the application of this method to the drilling of holes in mines, mention was made of an apparatus (such as is referred to below at the bottom of page 114) wherein the drill steel operates through an artificial collar held against the face of the rock; and through which ejector induced suction led the dust produced in drilling into a dust trap or filter. This apparatus was said to be very effective and popular in certain collieries to which laws relating to silicosis had recently been applied.

(2) *Masks.* — It appeared from the experience of members that workmen submit readily to their use only when discomfort from the inhalation of noxious dust could thereby be avoided.

In some circumstances loose fitting masks of the pressure type wherein

a constant supply of fresh air under positive pressure is led in through a flexible tube, have proved very efficacious. Such masks, however, are useful only when the wearers can perform their work without the necessity of moving from place to place. The same applies to tight-fitting masks supplied with air at normal pressure through a tube from a distant source.

Other masks wherein air was inspired through a filtering medium such as cotton wool, sponges, etc., and expired through a light non-return valve, were also described.

Reference was also made to masks in which the air to be inspired is made to pass through a tortuous path and impinge on damp surfaces which will retain the dust.

The feeling of the Conference was that while the masks at present available may be of some value in special circumstances, and particularly in those cases where the formation of dust (and the consequent necessity for precautions) is intermittent; they are so unwieldy or interfere so much with respiration that their constant use is impracticable during hard work and especially in a hot and humid atmosphere.

In regard to (d)—Ventilation—there was but little direct discussion, it being agreed that good fresh air ventilation was desirable, and indeed essential. It was emphasised, however, that to be effective the ventilation currents must be properly split and directed so as to sweep all dust-laden air out of the mine or works, as the case may be, in much the same way as dangerous gases are swept out of collieries.

With regard to (e)—Other Methods—mention was made of the fact that some years ago an endeavour had been made on the Witwatersrand to prevent the roof and sides of main intake airways from drying up (and, incidentally, to secure the "fly paper" effect referred to in 4 (2) and in the remarks thereon) by spraying those surfaces with solutions containing molasses, calcium chloride, and other hygroscopic substances, but it had been found that these preparations absorb moisture so readily that they soon trickle down into the gutters.

In discussion on this matter it was pointed out that if there really are any advantages to be derived from the "fly paper" effect, these solutions, and other sticky substances, could again be tried in the event of it being found possible at a later date materially to reduce the humidity of the ventilating currents. It was also suggested that these solutions might be used instead of plain water, for preventing the formation of dust in drilling, blasting, and the handling of rock.

In dealing with the dust formed by blasting, especially by blasting in development ends, a suggestion was put forward that it might be possible to project into the air at the time of blasting relatively large particles of some innocuous flocculent dust which in its settlement or progress through the mine would catch the harmful dust in much the same manner as micro-organisms are caught in water purification plants.

A further suggestion put forward was that saturated steam might be of some value, it having been found effective in industry in certain special circumstances.

A still further suggestion put forward was that the escape of dust from drill holes might be prevented by the use of a preparation producing a foam.

5. During the course of the discussion, reference was made to the difficulties experienced by investigators in the different countries in properly appreciating each others findings. Some of those difficulties arise from there being no accepted standards for comparisons in regard

to various conditions, dust counts, and so on; and others, through lack of a uniform terminology. The Conference, therefore, decided to put forward the following suggestions in the hope that those investigators who were in a position to do so, and particularly the Research Division of the International Labour Office, would take them up so as to pave the way for some decisions and recommendations at a future Conference.

(1) While the methods of conducting and other details relating to the routine sampling of air are best left to each local authority, it seems highly desirable that for certain special critical and scientific studies of dust particles in air and their effects, there should be established some standard method which for this special purpose would permit of inter-industrial and inter-national comparisons; in this connection it is suggested that the instruments at present approved by the various experts should be taken into consideration. The results of these investigations should be communicated to the Research Division of the International Labour Office for correlation.

(2) That as photographic and photo-electric cell methods of dust determination have been successfully applied in certain special circumstances, research should be undertaken with regard to such methods, with a view to ascertaining their adaptability in other circumstances.

(3) In view of the chemical theory of the causation of silicosis, the importance of estimating the size frequency of particles has increased owing to the fact that the surface exposure (which varies greatly with different sized particles) is the chief factor in the amount of silica which goes into solution. It is suggested, therefore, that investigators should include in their work determinations of the size frequency of particles.

(4) (a) The Conference urges that the investigations suggested in paragraph (1) should be undertaken with the least possible delay in all countries which are interested in the problem.

In view of the thoroughness and outstanding work of research already carried out on the Witwatersrand, and in view of the special facilities which exist in that area, the Conference attaches special importance to the investigations which may be undertaken on the Witwatersrand and ventures to express the hope that they will be initiated at the earliest possible moment.

(b) The Conference recommends that as soon as the standard method referred to in paragraph 1 has been perfected it should be applied for the purpose of making at least one complete survey of the dust concentration in dusty industries throughout the world. The results of this survey should be communicated to the International Labour Office.

(c) The Conference considers that the survey recommended in (b) should include an investigation into the relative size frequency of the dust particles.

Every effort should be made to emphasise the fact that the prevention of silicosis must be achieved by means of a whole series of provisions relating to hygiene in mines—viz. chiefly by reducing production and diffusion of dust, by maintaining the purity of the air, and by means of personal hygiene;

(5) That no opportunity should be lost of stressing the importance of general and localised ventilation as one of the best hygienic measures in dusty industries.

(6) That the personal protection of the workers should not be exclusively confined to such protective measures as, for instance, the wearing of masks, but should be supplemented by secondary measures such as the provision of suitable change houses and shelters, and by the regulation of working hours, etc.

(7) That many of the points raised in the prevention and control of dust call urgently for investigation by the physicists.

Note. — The present Report does not concern itself with medical methods of prevention of silicosis.

PROGNOSIS, AFTER-CARE AND COMPENSATION

The Conference considered the Report on Prognosis After-Care and Compensation and adopted it, after making various amendments. The Report is as follows:

Reporters: Dr. Cunningham, Professors Hall and Koelsch, together with Dr. Badham (co-opted).

In presenting our report it has seemed best to deal with each subject separately.

Prognosis

This subject may be considered under four headings:

Question 1: What is the prospect of a man exposed to free silica dust as regards acquiring silicosis?

This has been considered to depend on various factors:

- (a) The nature, silica-content, size of particles, and concentration of the particular dust to which he is exposed.

Concerning many of these points exact information is not forthcoming and it is desirable that further scientific investigations should be carried out and the results carefully compared with the incidence of silicosis amongst the workers.

- (b) Period of exposure to dust.

- (i) Length of service.

That this is a factor of importance is in accordance with the views expressed by members of the Conference from every country represented.

The length of service necessary to acquire silicosis may be considerably prolonged by improvement in other factors.

- (ii) Intermittence of exposure.

On this point the evidence forthcoming was not conclusive.

- (c) Age of worker.

There is no evidence that *per se* this plays any important part.

- (d) Physique of worker.

This is a factor of primary importance. An initial medical examination to ensure a certain standard of physique should be generally adopted in those industries in which the risk of exposure to silica dust is great.

Periodic medical examination of such workers is also essential.

- (e) Race.

There is no evidence that this is a factor of importance.

Question 2: What is the prognosis in a case of silicosis if the affected man leaves the industry at the first stage of the disease (ante-primary stage of South African legislation) ?

The evidence on this point as regards various industries and from different countries is somewhat conflicting.

In South Africa on the whole the evidence shows that the downward progress of the disease is in most cases not arrested on leaving the industry.

In this connection the questions of so-called infective cases, of reduced economic conditions of life, and of the associated mental worries probably play no inconsiderable part.

The view taken by the Medical Bureau in regard to the so-called "infective types" of silicosis is that they do not come under the classification of tuberculosis with silicosis unless the conditions in respect of tuberculosis as laid down in the Act are complied with. The prognosis in such cases of infective silicosis is much less serious than in cases of tuberculosis with silicosis as the term is used in the Act. Such cases may live for many years in comparative comfort unless active tuberculosis intervenes. The preceding evidence points to the urgent necessity of further experimental study of the exact conditions connected with silicosis of infective type.

Question 3: What is the prognosis in a case of simple silicosis if he remains in the industry after the first stage (ante-primary) declares itself ?

The evidence from South Africa suggests that in so far as mining is concerned the continuation in underground employment of sufferers from silicosis will aggravate the progress of the disease, except in certain selected mining occupations.

As, owing to economic factors connected with compensation, only a very small number of the silicotics in South Africa do remain at work underground after the disease has declared itself, the evidence on this point is somewhat indefinite. It is stated, however, that there are at present about 150 men in this position, and it is said that their progress is no worse than those who have left the mines. Among these 150 men, however, are included a considerable number of higher officials whose duties do not now expose them considerably to dust and whose economic position remains as good as before.

It is desirable to obtain exact information as to how far continuation at work in occupations involving exposure to silica dust will influence the progress of the disease.

Question 4: What is the prognosis in a case of silicosis with tuberculosis ?

(N.B. Before attempting to summarise the views of the Conference on this point it is desirable to make clear exactly what we mean. According to the terminology used in South African legislation "tuberculosis, means tuberculosis of the lungs or of the respiratory organs" and is deemed to be present "wherever it is found by the Bureau either (a) that such person is expectorating the tubercle bacillus, or (b) that such person has closed tuberculosis to such a degree as seriously to impair his working capacity and render prohibition of his working underground advisable in the interests of his health".)

This is always serious.

It is worse:

- (i) when the tubercular infection occurs at the outset of silicosis;
- (ii) in younger than in older subjects;
- (iii) than in cases of tuberculosis alone.

Recommendations

Prognosis of Silicosis

1. Exact information is not forthcoming concerning many of the points relating to the nature, silica-content, size of particles and concentration of dust to which a man may be exposed and it is desirable that further scientific investigations should be carried out and the results carefully compared with the incidence of silicosis amongst the workers.

2. The physique of the worker is a factor of primary importance. An initial medical examination to ensure a certain standard of physique should be generally adopted in those industries in which the risk of exposure to silica dust is great.

Periodic medical examination of such workers is also essential.

3. The evidence points to the urgent necessity of further experimental study of the exact conditions connected with silicosis of infective type.

4. It is desirable to obtain exact information as to how far continuation at work in occupations involving exposure to silica dust will influence the progress of the disease.

Compensation from the Medical Point of View

1. Silicosis complicated or not by tuberculosis constitutes an occupational disease which may involve reduction of working capacity.

2. It should be left to competent authorities to decide, in accordance with their particular conditions, whether other forms of pneumoconiosis should be regarded as occupational diseases.

3. In establishing the amount of disability, account should be taken of the clinical and functional condition as a whole.

4. The determination of disability should be entrusted to an independent medical expert, or body of experts, possessed of the requisite clinical and technical knowledge, and having at his or their disposal suitable apparatus for effecting the examination.

5. It is suggested that removal from all industrial occupations involving exposure to noxious dust should be enforced in all cases of open tuberculosis.

6. Where legislation provides for the compulsory removal from occupations involving exposure to silica dust of workers affected by silicosis, it is suggested that such compulsory removal should not necessarily be applied to workers who have been in the same industry for a period of not less than fifteen years and have reached the age of forty-five years.

After-Care

1. Sanatorium treatment should be provided for suitable cases.

2. Hitherto most of the rehabilitation schemes have been unsuccessful. Further investigation into this problem is urgently called for.

(The Conference adjourned at 12.45 p.m.)

*THIRTEENTH SITTING**Wednesday, 27 August 1930, 10 a.m.**Chairman: Dr. L. G. IRVINE*

The Chairman: The minutes of the previous Sittings, with the corrections made by members, would be taken as adopted. Any further corrections which members had to make could be sent to the International Labour Office in Geneva.

Mr. Phelan: The Conference might hold an informal conversation on the next steps to be taken in the question of silicosis.

The Chairman: The Government Departments concerned should maintain libraries on the question of silicosis.

Dr. Badham: The text of Recommendation IV (6) was incomplete. It was decided that the corrected text should be distributed immediately.

Sir Spencer Lister: The Conference might make a recommendation regarding the safe storage of X-ray films.

After some discussion, it was decided that this suggestion should be mentioned in the Minutes.

(At this point the representatives of the Press were admitted.)

Mr. Phelan: On behalf of the International Labour Office, he thanked the delegates for the work which had been brought to a successful conclusion. He realised that they had sacrificed their time and convenience in coming to this Conference. The special thanks of the Office were due to Dr. Russell and Dr. Gardner, who had recorded yet another proof of the unstinted scientific collaboration of the United States of America. The thanks of the International Labour Office were also due to the Chamber of Mines, and in particular to Mr. Limebeer and the Staff, without whose help the small Geneva staff could not have coped with the work. The thanks of the Conference were due to Messrs. Owen Jones, Ltd., for the loan of epidiascopes and microscopes; to the South African General Electric Company for the loan of X-ray stands, and to Messrs. Kodak (South Africa) for the album which they had presented to members. This was the first Conference which the League of Nations had convened outside Europe, and a certain risk was involved, for Conferences had been known to fail in Geneva. This Conference, however, was an unqualified success, largely due to the hard work of the delegates, and in particular to the Chairman, who had conducted a highly technical discussion with charm and fairness.

He presented the Chairman with a photograph of the members of the Conference.

Professor Hall moved a vote of thanks to the Chairman, to whose great clinical experience and to whose advice and help it was entirely due that the Conference had performed its work punctually and unanimously.

Professor Böhme seconded the vote of thanks and said that all the members of the Conference had admired the Chairman's expert knowledge and tact.

The vote of thanks to the Chairman was unanimously adopted.

The Chairman: He thanked the Conference for the honour which they had done him. His original trepidation at taking the chair had been replaced by a feeling of increasing pleasure in his task because of the extraordinary spirit of goodwill and co-operation which the Conference had shown throughout. He was much encouraged by the spirit of good fellowship shown between scientific workers of many scattered nationalities.

South Africans did not always realise that the problem of silicosis was world-wide and ramified into many other industries. The Conference had largely recognised and accepted South African experience, but equally valuable contributions had come from other countries. A large measure of agreement had been reached on the causation and nature of silicosis, but they had also been led to realise how much material was not yet known. For these reasons, the Conference had laid down lines for future investigation and research. He hoped the International Labour Office would keep the spirit of international co-operation alive. On behalf of the Conference he expressed his thanks to Mr. Phelan and the Staff of the International Labour Office, to the Chamber of Mines, to Dr. Orenstein, and to the various reporters.

Dr. Middleton moved a vote of thanks to the Vice-Chairmen.

Professor Hall seconded this vote of thanks.

The vote of thanks to the Vice-Chairmen was unanimously adopted.

Sir Spencer Lister thanked the Conference. Their duties had been light because the Chairman had borne the greater burden.

Dr. Russell also thanked the Conference and the International Labour Office for inviting him to be present. He was especially grateful to the Chairman and the South African members for their hospitality and friendship.

Professor Loriga: He had been more an observer than a collaborator. He had thought that some of the conclusions in the papers submitted to the Conference were exaggerated, but he was now convinced that South African scientists had a very profound and accurate knowledge of silicosis.

Dr. Kranenburg: The thanks of the Conference were due to Dr. Orenstein, to Dr. Carozzi, and to the members of the Staff of the International Labour Office.

The Chairman declared the Conference closed.

(The Conference adjourned sine die at 11.50 a.m.)
