

McIntyre-Saranac Conference on Occupational Chest Diseases



SARANAC LAKE, NEW YORK—FEBRUARY 7, 8, AND 9, 1955

FOR THE FIRST time the McIntyre Research Foundation and the Saranac Laboratory united in a shared enterprise: the "McIntyre-Saranac Conference on Occupational Chest Diseases." These two facilities are bound together in high-minded dedication to acquire and disseminate precise information on occupational diseases of the chest and notably the dusty lung diseases. Essentially, both these organizations are devoted to research investigations. The much that already has been achieved chiefly reveals the inadequacy of full understanding of the pulmonary occupational diseases and thus piques the realization that the days of such research are not numbered.

Beginning about 1933, the Saranac Laboratory inaugurated a series of Conferences entitled "Symposia on Silicosis," the seventh and last of which was staged in Saranac Lake in 1952. The published proceedings of these Conferences long have served as text and reference books on the dusty lung diseases to the point of having become authoritative. So great did the Saranac Laboratory influence become that the city of its location, Saranac Lake, New York, long has been accepted as the Mecca of occupational pulmonary disease investigation. The numbered series of symposia has given way to this, the first McIntyre-Saranac Conference, but the character of the program, the excellence of presentation, and the outstanding research work represented have not been altered except that the output of the two outstanding organizations presaged an even higher order of contribution.

The scientific status of both organizations sponsoring this conference is such that enormous responsibility rests upon all participants. What was said in the many sessions unflinchingly will touch industry, management, labor unions, compensation courts, physicians, educators throughout America and, even more, many other portions of the world. This imposed a doubly earnest, zealous and cautious approach to the tasks in hand.

The Conference program, as devised by the program committee, was guided in its coverage by Dr. G. W. H. Schepers, Director of the Saranac Laboratory and Dr. J. W. G. Hannon, Medical Director for the McIntyre Research Foundation.

Not lightly stated, the purposes of this Conference and its subsequent scientific publications amount to the determination to add to the sum

total of scientific attainments that eventually will bring all occupational diseases of the chest to praiseworthy control.

Although this Conference and all of the preceding seven Saranac Symposia offer no official pronouncements, they have provided scientific pronouncements that long have influenced medical and compensation board concepts on occupational chest diseases. There is reason to believe that this latest Conference in equal measure will influence and, to some degree, govern the thinking and the action of many persons concerned with occupational pulmonary diseases.

Major Disclosures in the Conference

SINCE this Conference is without any official status, no pronouncements were made other than by individual speakers. Still, there was tacit acceptance of many statements that in the aggregate represent the consensus of beliefs as to pulmonary occupational diseases at this time. The total embraces the following:

Silicosis as one dusty lung disease is common to all industrial countries. Perhaps in no country are recording systems sufficiently complete to provide other than an estimate of the total number of industrial workers afflicted. One estimate for the United States is that presently there may exist as many as 300,000 cases of diagnosable pulmonary occupational disease, chiefly silicosis.

While the widely accepted standards as to tolerable limits of exposure may be recognized as far from exact, no justification is known to exist at this time for any sweeping modifications. These standards, which are official in some jurisdictions are:

Substance	Million Particles Per Cubic Foot
Dust containing free silica	
high (above 50% free SiO ₂)	5
medium (5 to 50% free SiO ₂)	20
low (below 5% free SiO ₂)	50

More than ever, silicosis is to be considered both as to clinical and compensation appraisal sharply in connection with the degree or quantity of involvement. It is possible that a substantial portion of the adult population of the United States and presumably other countries harbor a few demonstrable silica nodules without industrial exposure and the number may include housewives never engaged in employment other than household duties. It follows that the mere de-

in autopsy or biopsy. A few cases are not diagnostic of occupational silicosis and possibly have no bearing on disability. Further, it follows that some degrees of silicosis detectable through x-ray examination fully may be compatible with a long work life and in the absence of any measurable disability caused by that disease.

The classical silica nodule is the primary lesion of silicosis. Atypical nodules such as the stellate form sometimes described may not be regarded as different lesions but only as modifications of the classical form.

In recent decades, exposition of the causation of silicosis presumed some degree of solution of the silica within pulmonary structures which solution products were labeled as the provocative agent. From silicates investigated under experimental conditions, more free silica solution has derived than from proportionate quantities of free silica. Yet, from silicate exposure (asbestos, talc, and perhaps similar minerals excepted) no characteristic occupational disease occurs and certainly not silicosis. At the present time, many evidences are arising that the causation of silicosis in some manner is associated with particle surface action rather than simple solution, but the exact nature of this responsible surface action remains undisclosed.

As to aluminum therapy in the prevention and treatment of silicosis, the results from animal investigative work are substantial. It appears established that aluminum in more than one form but not all forms, hampers and limits the development of silicosis, but perhaps exercises no ameliorative action on silica nodules already preformed. The application of aluminum in the prevention of silicosis in human beings, and, to a lesser degree, therapy for cases already established, is attended by numerous supporting observations and investigations. The crucial appraisal of the worth of aluminum in connection with silicosis and its prevention awaits the results of fully controlled application. It is obvious that the establishment of a precisely controlled investigation is difficult and manifestly a prolonged affair involving years. No less, some uncertainty will continue to attend the use of aluminum in relation to silicosis until unequivocal results accrue from a well-planned, controlled investigation.

For practical purposes and by long precedent, silicosis does not become significant until that stage of development is reached which is characterized by modifications revealed on the well-made x-ray film. Inevitably, it must come about that prior to that time there must be indiscernible or little discernible evidence of abnormal pulmonary tract structures, but the quantity of such changes is of such order that no disability exists and compensation for the immediate situation is not warranted. Compensation on an anticipatory basis pending possible progression of the disease and plans for future compensation may be in order but as to the "then and there status," the pre-x-ray state of silicosis becomes negligible.

At this McJannet-Sarason Conference, the early change in the pneumoconiosis, i.e., that antecedent x-ray evidences of deviation from the normal was reported as in part related to vascular changes with the ultimate prospect of damage to the right side of the heart eventuating in cor pulmonale. This may not be interpreted to mean that new forms of silicosis or other pneumoconiosis have been disclosed which call for entirely new criteria for diagnosis but, in general, this newer work tends to emphasize greater cardiovascular damage than heretofore has been contemplated.

For a long time, it has been recognized that a group of silicon-bearing compounds such as talc has been the cause of pulmonary occupational disease. Otherwise this same talc as earlier used by surgeons in connection with the lubrication of operating room gloves, has led to some culpation in connection with the inauguration of adhesion formation. In explanation, there has been the practice to assert that enough free silica existed within the talc to induce these changes so that what occurred in terms of pathology was not other than silicosis or at least a modified silicosis. Now it appears that talc, which is a silicate-like asbestos and some other silicates, through its own properties, is capable of producing damage and that which takes place is not silicosis. A new primary lesion is believed to exist which lesion is not the silica nodules but more akin to the "asbestos body." If this be true, a disease heretofore regarded as silicosis becomes a separate and distinct disease entity.

For nearly four decades, enormous difficulties have attended the diagnosis and legal adjudication of claims alleging silicosis and disability or death therefrom. One of the emphasized features of this Conference was that much of this uncertainty could be removed through the removal of small portions of lung tissues followed by appropriate microscopic examination. Such examination of a properly chosen lung biopsy specimen might yield such findings as speedily and definitely to establish that the existing pathology was Boeck's sarcoid or carcinoma or silicosis or anthracosis or talcosis or tuberculosis. However it will be recognized that silicosis may exist along with tuberculosis and that asbestosis could co-exist with carcinoma. Still the potentialities of suitable microscopic examination well might reveal all such dualities as well as the single condition should singularity exist. Precise as this procedure may be, its widespread practice or application is not likely to come about since the removal of the required lung specimen in itself constitutes a major surgical operation.

Notable Investigations

WHILE all of the numerous scientific presentations were notable, some individual papers and some groups of papers stand out as exceptional. This number includes:

"Observations on the Comparative Vascular Pathology of the Occupational Chest Diseases"—G. W. H. Schepers.

"The Value of Lung Biopsy in the Diagnosis of Occupational Pulmonary Diseases"—H. S. Van Orstrand.

"Studies on the Prophylaxis of Experimental Silicosis by Means of Aluminum"—M. Dworski.

"The Demonstration of Aluminum in Animal Tissues"—D. A. Irwin.

One group of papers constituting a half-day program was devoted to the epidemiology of occupational chest diseases on an international basis, all furnishing evidence that no country is the sole habitat of pulmonary occupational diseases.

Prominent in this group were:

"A Review of Progress in the Conquest of Occupational Chest Diseases in South Africa"—G. W. H. Schepers.

"Accomplishments in the Epidemiologic Study of Silicosis in the United States"—H. N. Doyle, V. M. Trasko, Wm. Gafafer, S. E. Miller.

"Silicosis in Canada"—N. F. Parkinson, A. R. Riddell.

"Talc Pneumoconiosis"—M. Kleinfeld, J. Mesite, I. R. Tabershaw.

"The Dust Diseases in Great Britain"—A. I. G. McLaughlin.

"A Correlation of Tissue Reaction and the Mineral Content of the Lungs of Experimental Animals"—P. C. Pratt, T. M. Durkan.

As an exception to the general trend of discussion revealing the widespread existence of devastating occupational chest diseases was a report by Dr. Geoffrey E. Ffrench entitled "Pneumoconiosis on the Kolar Gold Field, South India." In this locale despite undenied high exposure to free silica, the incidence and severity of silicosis was not remarkable. It was recorded that one natural constituent of the total dust was a considerable percentage of aluminum oxide. In guarded fashion, the question was proposed as to the possibility of this aluminum serving as a preventive agent further substantiating the moot claims as to the protection qualities of that material.

Continuing with this recital of high merit in manuscripts, the following were notable:

"The Reaction of Chromium Compounds With Body Tissues and Their Constituents"—Anna M. Raetjer, C. M. Damron, J. H. Clark, V. Budacz.

"Biochemical and Biological Aspects of the

Surface Properties of Particulate Quartz"—L. D. Scheel, G. W. H. Schepers.

"Criteria for the Assessment of Disability in Occupational Chest Disease"—G. F. Filley.

"A Clinical Study of the Effects of Inhaled Calcined Gypsum and of Talc Mining Dusts on Human Lungs"—G. W. H. Schepers, T. M. Durkan.

"The Pulmonary Disability Associated with Coal Mining"—J. W. G. Hannon.

"The Disability Found in Persons Exposed to Certain Beryllium Compounds"—H. L. Hardy.

"The Pulmonary Disability Attendant on Asbestosis"—K. W. Smith.

"The Experimental Study of the Effects of Inhaled Hydrous Calcium Silicates, of Calcined Gypsum, and of Talc Dusts on Animal Lungs"—G. W. H. Schepers, T. M. Durkan.

"The Experimental Study of the Biological Action of Rare Earth Oxides and Fluorides, Tungsten Carbide, Cobalt, and Glass Wool on Animal Lungs"—G. W. H. Schepers, A. B. Delahant.

At the Dinner session of the Conference the Leroy U. Gardner Memorial Address was presented by Dr. Paul S. Richards, of Salt Lake City, Utah. This in turn was followed by Historical Sketches on Dr. Edward L. Trudeau and Dr. E. R. Baldwin, by Dr. Hugh M. Kinghorn, who was himself a contemporary of Drs. Trudeau and Baldwin and who participated in many of the early laboratory investigations of tuberculosis in the United States and Canada.

Distinguished Participants

THE total number in attendance including the staffs of the two sponsor organizations exceeded 250, with representation from every section of the United States. Representatives from countries other than the United States and Canada included the following physicians, industrial hygienists, and attorneys: Dr. E. Balgairies, Douai (Nord), France; Dr. Nandlal Bordia, Indore, India; Dr. Maha Nand Gupte, New Delhi, India; Dr. A. I. G. McLaughlin, London, S.W., England; Dr. Artemio Miranda, Lima, Peru; Dr. S. F. Oosthuizen, Pretoria, Union of South Africa; Dr. C. Kelman Robertson, Edinburgh, Scotland; Dr. W. Jones Williams, Cardiff Wales, England; Dr. Geoffrey E. Ffrench, Kolar Gold Field, South India.