

INTER-ORGANIZATION CORRESPONDENCE

UNITED STATES STEEL CORPORATION

TO All Participants

DATE September 1, 1960

FROM R. B. O'Connor, M. D.
Medical Director

SUBJECT Transactions, Sixth Semiannual
Administrative Medical Meeting

Enclosed is a copy of the Transactions of the Sixth Semiannual Administrative Medical Meeting held in Pittsburgh on June 21 and 22, 1960.

There was so much clinical detail involved which appeared to be of special interest that I took rather copious notes. From them I compiled this summary, which you may want to review and keep in your files for reference.

R B O'Connor M.D.

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enclosure

Drs. R. N. Armen	Drs. A. M. Edwards	Drs. J. H. Murry
L. H. Botkin	G. V. Foster	J. F. Novak
R. J. Bennett	Louis Hamman	J. C. Painter
D. C. Braun	J. C. Hayes	W. J. Ralston
C. B. Bryce	W. J. Helzlsouer	W. K. Riland
Merle Bundy	W. J. Lace	R. W. Saul
E. W. Caughey	B. J. Larsen	D. J. Smith
H. H. Clapper	R. M. Maher	J. T. Taylor
J. B. Clement	C. P. Markle	Mr. W. W. Turner
H. E. DeWalt	H. E. Mathay	Drs. J. R. Weddell
J. C. Donchess	Mr. K. M. Morse	C. R. Zeiss

cc: E. B. Robinson, M. D.
F. J. Schaeffer
C. T. Spivey

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TRANSACTIONS OF THE U. S. STEEL
~~SIXTH~~SEMIANNUAL ADMINISTRATIVE MEDICAL MEETING
115 William Penn Place Building, Pittsburgh, Pennsylvania

June 21 and 22, 1960

U. S. ~~Sixth~~Sixth Semiannual Administrative Medical Meeting was convened at 9 a.m., ~~June 21~~. Those in attendance are listed in Appendix A. The program is listed in Appendix B.

The entire first morning was devoted to pulmonary disease.

I. The first speaker was E. D. Robin, M. D., Associate Professor of Medicine at the University of Pittsburgh. As an aside at the beginning, he mentioned that he would be giving a talk to UMWA doctors on the same subject, namely, Pulmonary Function Tests in the following week.

A. At ~~the~~outset, Dr. Robin said there were four things that should be recognized as a backdrop to his talk:

1. All things being equal, the best pulmonary function test still is a careful clinical history, a thorough physical examination, and properly and carefully taken x-rays of the lung.
2. Current pulmonary function tests almost never give data that is etiologically precise, i.e., they tell the degree of involvement but not the cause of it.
3. Dr. Robin emphasized that he was unfamiliar with our specific problems and hoped to learn from us.
4. As presently understood, pulmonary function is complex, thus current tests are complex; there is no single adequate test, but rather subdivided, isolated functions are tested with different presently available tests.

B. Four reasons were given for doing pulmonary function tests:

1. To pick up subtle gradations of abnormality. For example, in asbestosis the patient may have considerable symptoms but there may be no abnormal physical findings except in a pulmonary function test.
2. These tests are the only way to get specific figures representing objective measurements.

In reply to the question as to whether the Timed Vital Capacity will pick up early emphysema, he said there is an early stage which is not ordinarily detectable but will be picked up by this simple test. He stated that pulmonary function tests cannot distinguish focal emphysema from diffuse obstructive emphysema.

A glossary of terms, abbreviations, definitions and normal values, supplied by Dr. Robin, is in Appendix C.

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II The second speaker was K. M. Morse, Director of Industrial Hygiene, U. S. Steel, and the title of his talk was "Measurement of Dusts."

Mr. Morse said there are four major items of concern to the industrial hygienist and the physician regarding dust and its effects on the lungs:

A. Chemical composition. In the past, one was concerned almost wholly with silica and asbestos. If these were not present, then the dust was considered merely a nuisance. Although free silica content is still important and silica is the most hazardous of the common industrial dusts, we now know that any dust regardless of silica content, in great enough quantities, can overload the defenses of the lungs and cause trouble.

B. Particle size. We are concerned only with those under 5 microns. It is felt that maximum retention of dust in the lung occurs with particle size between 1.5 or 2 microns.

C. Concentration. Maximum Allowable Concentrations (MAC) have been set up by the Conference on Governmental Industrial Hygienists for all the substances commonly met in industry. Some of these have been well substantiated, but some of them are mere guesses. For example, the MAC of 5 million particles per cubic meter of air for silica has been well validated, whereas a commonly accepted MAC of 50 million particles for "nuisance" dusts is not at all well validated. In U. S. Steel, we are aiming below this latter amount and are recommending 20 million as a maximum in any dusty situation.

D. Duration of Exposure. In speaking of pulmonary dust diseases, one is speaking of conditions that take twelve to fifteen years to develop.

Mr. Morse then discussed various techniques of measurement of dust and the instruments used:

A. Sedimentation. This is now obsolete for in-plant studies, but is still used in some studies of community air pollution. Settled dust can be used for chemical analyses; however, it is apt not to be truly representative of the dust in the air.

Two psychiatrists tried unsuccessfully to get rid of the fear reaction. A Workmen's Compensation Claim was filed. He said he was so disabled that he didn't even have the stamina to drive a taxi cab, a job which he had tried. His wife was working to support him and their three children. He was awarded compensation. Subsequently he was killed in an auto accident and at autopsy his lungs were proven to be perfectly normal except for iron retention which produces no disablement.

Dr. Sander said that the best distinction on x-ray between a benign siderosis and an actual pulmonary fibrosis was that in siderosis the root shadows were within normal limits. Also the nodulation was fine, small and evenly distributed. Of course, sometimes one can have both, as in welders who work in a foundry cleaning room, exposed to both iron oxide fumes and to silica dust.

To show the difference, x-rays of a case of silicosis were used to demonstrate the larger general silicotic nodulation with dense root shadows and increased root markings, compared to the siderosis, uniform fine nodulation and normal hilar shadows. These are sometimes combined in a grinder, although now most grinding wheels are Silicon Carbide or Aluminum Oxide. Earlier studies from Saranac suggested that Silicon Carbide might make a person more susceptible to tuberculosis; Sander has doubted this, and Dr. Gross has shown overgrowth of fibrous tissue in animals dusted with Silicon Carbide but no increase in incidence of tuberculosis. Sander has successfully contested claims of this kind in Workmen's Compensation. With the grinding wheels presently in use, he indicated that grinders can develop pure iron retention, i.e., siderosis.

He then showed x-rays of a case of tin oxide retention, which presented a brighter, harder appearing nodulation.

Discussing 35 claims for "damaged lungs" in burners in ship yards, said to be due to smoke, iron oxide and soot from coal, he reviewed the question of the possible occupational origin of emphysema. While he is not prepared to say that emphysema is any higher in occupational exposures than in the general population, he has found it very difficult to contest a case in men exposed to irritants who develop emphysema. However, if there is well-developed emphysema in a man who has been a heavy smoker all his life, who has a heavy cough and recurrent upper respiratory infections, such a case should be vigorously contested. Sander and many others felt that heavy smoking is a major cause of emphysema. As men grow older and smoke longer, developing chronic bronchitis and emphysema, those that also have dust exposure at work he feels will probably succeed in a compensation claim.

He next showed x-rays of a case of asbestosis caused by installing asbestos bats around piping. This affects the alveolar walls more than silicosis, and in this case the man died of cor pulmonale 20 years after the last exposure to asbestos. He contrasted this with simple silicosis which does not produce such disablement because the alveolar walls are not so blocked with the fibrosis.