FILE NAME: Hercules Chemical (HERC)

DATE: 1947

DOC#: HERC007

DOCUMENT DESCRIPTION: Book Excerpt from Introduction to Industrial Medicine - Industrial Dusts

220. Haylett's book 1947

Introduction to

INDUSTRIAL MEDICINE

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INDUSTRIAL MEDICINE PUBLISHING COMPANY
CHICAGO

SECOND EDITION

PRINTED IN THE UNITED STATES OF AMERICA

PREFACE

THE material of this book consists of the lectures given in a course on Industrial Medicine at the School of Medicine of the University of Pittsburgh. To present the content of Industrial Medicine in some degree of completeness would require a series of volumes. It is impossible to attempt exhaustive treatment within the compass of the lectures given in this course.

The lecturers participating in the course are active workers in their respective fields. They have presented their subjects in the briefest possible form consistent with clarity. The basic concepts with which these lectures deal may serve as points of departure for the student whose interest demands greater detail of presentation than is possible in the brief time at our disposal.

The value of the lectures will be greatly enhanced by the student's own notations originating either from class discussions or individual reading. The bibliography provided herewith will aid in guiding the student in his excursions through the literature.

Beyond their immediate value, these lectures may be useful as future reference material. Consequently, this means is provided for their preservation as well as for the immediate convenience of the student.

Grateful acknowledgment is made of the cooperation of the contributors who, despite the many pressing demands upon their time, agreed to prepare their notes for publication.

T. LYLE HAZLETT, M.D.

Interpretation of the significance of the dusts causing inert reactions is more difficult, but it appears logical to assume that dusts which show a tendency to remain in the tissues should be considered as potentially harmful, though not as dangerous as those causing a proliferative response. It is likewise logical to assume, and it has been proved to some extent in the laboratory, that silica mixed with an inert dust causes a modified proliferative reaction. With this biological method of classification, which, in a number of instances, has been correlated with clinical observations and industrial surveys, it is quite possible to use intraperitoneal injection methods to determine the pneumoconiotic potentialities of a dust in a relatively short time, usually 60 days.

III. Determination of Dusts

A. Necessity for Determination of Concentration and Composition.
B. Correlation with Physical Findings.
C. Standards of Dustiness for Different Conditions.
D. Methods of Determinations.

IV. Control of Health Hazards Due to Dusts

The elimination of dust hazards involves primarily the control of dust in the working environment and the periodical examination of workers engaged in the dusty trades.

The Pneumoconioses

The term pneumoconioses includes the lung changes of chronic character which are produced by the inhalation of dust over a prolonged period of time. For purposes of classification we shall divide them into the following categories based upon the nature and degree of pathology:

I. Normal Average Lung
II. Normal Average Lung+
III. Normal Average Lung++
IV. Potential Pneumoconiosis
V. Pneumoconioses
   A. Anthracosis
   B. Siderosis
   C. Asbestosis
   D. Silicosis

VI. Pneumoconiosis with Infection

Certain common dusts and their effects are briefly considered in the following pages. More detailed accounts are available in the literature mentioned in the general bibliography.

Pneumoconioses other than Silicosis

The degree of pulmonary involvement in the pneumoconioses may vary in severity from a mere phagocytic process to a condition of marked and progressive fibrosis. Silicosis, the most serious of the pneumoconioses, is due to silica dust. Among the others are asbestosis, anthracosis, siderosis, aluminosis, and byssinosis. This list is not complete; other forms exist which have not as yet been fully studied.

The reactions to dusts other than silica and asbestos are not of such character as to seriously compromise the respiratory functions. Dr. L. U. Gardner, of the Saranac Lake Laboratory, has in fact suggested that the designation "simple benign pneumoconiosis" be applied to such conditions. Among them we may mention the common uncomplicated anthracosis from coal and siderosis which results from carbon and iron. When, however, these substances are mixed with quartz the resulting condition is a modified silicosis designated, on the basis of the etiology, as anthraco-silicosis or sidero-silicosis.

Since asbestos is the only silicate which is considered to produce serious effects, it will be accorded separate treatment.

Asbestosis

The number of workers exposed to asbestos is small when compared to those exposed to silica dust. A few years ago Lanza estimated the number to be approximately 10,000.

The elements of asbestos dust are in the form of fibers which vary in length up to 50 or 60 microns although some have been found to have a length of 200 microns. These bodies tend to remain in the terminal bronchioles but some of the shorter ones are carried by the dust cells into the walls surrounding the air spaces and there give rise to fibrosis. There is thickening of the alveolar septa and the alveolar structure is finally destroyed and replaced with fibrous tissue. The fibrosis is ultimately seen as diffuse, peribronchial and basal. It progresses into all parts of the lung tissue. Within the fibrotic areas bronchiolectasis and bronchiectasis may be seen. There is a thickening of the pleura,

particularly in the basal areas, and, in fact, there may be complete obliteration of the pleural sac.

The action of asbestos is believed to be of mechanical nature and not chemical as in the case of free silica. Irritation by the stiff fibers is believed to produce the tissue injury in the disease. If the length of the fiber is reduced to about two microns, its irritating quality is destroyed.

In affected lungs asbestos bodies are found in both diseased and healthy areas. They are cylindrical and often have rounded ends which give them a dumb-bell form.

The roentgenological findings are largely seen in the lower one-half or two-thirds of the lung fields; in silicosis the upper areas also show evidence of pathological change. The pleural thickening is apparent. There is no nodulation and the general appearance of the picture has been described as resembling "groundglass." Early roentgenological diagnosis is difficult.

Among the significant signs and symptoms are dyspnea, which is the first to appear, and nonproductive cough. The chest expansion is diminished. There is, frequently, pallor, weight loss and cyanosis. As the process advances, fine crackling rales may be heard.

The time required for the disease to develop is said to be seven to nine years if the concentration is fairly high. The disease is progressive. In evaluating the degree of disability it is necessary of course to consider any coexistent pulmonary infection, particularly tuberculosis.

The treatment of the disease remains symptomatic; reliance must still be placed upon the elimination of exposure through adequate atmospheric control.

Carbon

Carbon is relatively harmless. From work done by Cummings and Sladden on the examination of many coal miners there would not be any serious impairment of health if there were no damage from silica already present. Dr. Haythorn, of Pittsburgh, in a series of 2000 autopsies has shown that with an increasing amount of carbon in the lungs there is a decrease in the number of tubercles found. Therefore, an atmosphere containing a high percentage of carbon such as we have in this district may account for our low mortality from tuberculosis. From our own experience with foundry workers one cannot help but

feel that carbon mixed with silica has a retarding effect on the action of silica.

Haldane in England has suggested that carbon be blown in the air as an antidote for silica.

Coal dust in the lungs or anthracosis has received more attention than silicosis but according to our present knowledge the following facts may be stated:

1. A fibrosis not typical of silicosis and not sufficient to cause disease may be shown on an x-ray film following inhalation of pure coal dust.
2. Silicosis may occur when siliceous rock overlaps the coal.
3. Brundage, of the United States Public Health Service, states that tuberculosis is low for soft coal miners but not for anthracite.

Iron

None of the oxides of iron in themselves have been shown to produce any serious effects on individuals although the lungs of men who have been exposed to iron dusts are characteristically colored and are called siderotic, the condition itself being termed siderosis. Oftentimes, however, the ore contains quartz which is silicosis-producing and causes a varying degree of fibrosis, depending on the proportion of quartz present.

Tuberculosis is rare in workers in oxide of iron.

Dr. Gardner believes that iron oxide retards the onset of silicosis and also modifies its progress. He and his associates have made a most comprehensive study of men employed in the iron mines in the Lake Superior region and have conducted animal experiments without discovering evidence of disability due to uncomplicated nodular fibrosis. There were 6,000 men examined.

Cement

The manufacture of Portland Cement, although one of the dustiest operations, has held the enviable reputation of being free from tuberculosis. On the basis of several surveys, cement dust has not been found to be a producer of silicosis, tuberculosis or pneumonia.

Cement is made by crushing limestone with clay or shale, roasting this mixture and then pulverizing it. The constituents are silica, silicates and calcium carbonate.

In a study by the Public Health Service, diseases of the skin,