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Contents

Treatment
M.D., Duluth, Minnesota 770

Coronary Disease
M.D., Saint Paul, Minnesota 775

M.D., New York, N. Y. 776

Cyst of the Thorax Simulating Empyema
M.D., and *Robert Woodruff*, Rochester, Minnesota 781

on the Occurrence and Prevention of Death
M.D., Saint Paul, Minnesota 783

with the Patient Who Is Allergic
M.D., Rochester, Minnesota 787

Facial Diplegia (Neuritis) Occurring in Children
M.D., F.A.C.P., and *M.D.*, Saint Paul, Minnesota 789

the Appendix and
Saint Paul, Minn. 791

Hyperplastic Gravis with Cure
Minn. 795

Histaminase in the Treatment of Urticaria of Pregnancy.
I. S. Blumenthal, M.D., Minneapolis, Minnesota 797

History of Medicine in Minnesota:
Diseases of the Dakota Indians. (Continued from October issue)
Thomas S. Williamson, M.D. 798

Dakota Medicine.
Rev. Thomas S. Williamson 801

President's Letter 806

Editorial:
Historical Highlights on Appendicitis 807
A Safe Safety Pin 808

Medical Economics:
The Blind Goddess 809
Minnesota First 809
National Health Program—1940 810
Medical Induction Boards 812
Privileged Communications 812
Thanks of the Council 813
Minnesota State Board of Medical Examiners ... 813

Communication 814

Of General Interest 815

In Memoriam 818

Reports and Announcements 819

Woman's Auxiliary 820

Books Reviewed 821

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interpretation may be of the utmost value, the interpreter must have a summary of the clinical history and physical findings.

Summary

1. The electrocardiograph definitely aids in the diagnosis and management of coronary disease and serial electrocardiograms are invaluable.

2. The electrocardiograph is only informative on the anatomical condition of the heart and in no way aids one in determining the functional capacity of the heart or its ability to do work and it is of almost no value in the diagnosis of valvular defects.

3. The electrocardiograph may eliminate the heart as a source of symptoms suggestive of myocardial disturbance.

4. The interpretation of the electrocardiogram must be made by a thoroughly trained individual and the findings must be correlated with the clinical history and physical findings.

5. The electrocardiograph, of and by itself, cannot make the diagnosis and prognosis for you.

PNEUMOCONIOSIS*

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PNEUMOCONIOSIS is a generic term whose use at times has been attended with some confusion. It embraces the reactions of pulmonary tissue to industrial dusts, which in some instances present clear-cut diseased conditions; in others, conditions which are not entirely clear; and in still others, conditions which have little or no clinical significance. Much water has gone over the dam since attention was first called to the importance of dust exposure in industry as a cause of pulmonary disease. A little over thirty years ago, when the campaign against tuberculosis was well under way, the statisticians began to call attention to the fact that workmen in dusty trades had a consistently high death rate from tuberculosis. It was surmised that many kinds of dust, by virtue of their physical properties of hardness, sharpness, and insolubility, produced a mechanical injury of the lungs which paved the way for a tubercle infection. With respect to certain organic dusts which did not have physical properties calculated to injure the pulmonary tissue, it was felt that these probably acted as conveyors of the tubercle bacillus. This seemed a reasonable explanation for the excess mortality from tuberculosis observed among certain groups of textile workers, tobacco workers and leather workers.

Disability and a high mortality were so conspicuous in what was commonly termed the hard rock mining industry that it was but natural that anti-tuberculosis workers should stimulate investigation of that type of underground work and the first clinical study of pulmonary dust disease was made about twenty-five years ago by the United States Public Health Service and the Bureau of Mines, to be quickly followed by others in various parts of the country. These early investigations were mostly confined to the mining industry, except for the studies made in the textile industry by a group of physicians in Philadelphia.

Investigations were made in industrial communities over long periods of time. This procedure offered the tremendous advantage of observation of industrial workers ranging from those new in the industry to those who had been employed for many years, and thus made possible a correlation between the symptoms that presented and the length of employment and the working conditions in various occupations in the same industry. There was gradually built up a comprehensive knowledge of the etiology of pulmonary dust disease which led to a clearer understanding and did away with many false conceptions. The most conspicuous fact was that where workmen were exposed to dust containing free silica in large amounts, they contracted a definite and specific type of pulmonary fibrosis that this fibrosis produced characteristic appearances on an x-ray film, and was accompanied by a consistent progressive clinical picture, in which dyspnea on exertion was the outstanding symptom.

It was learned in time that the symptoms of

*Address at the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, April 23, 1940.

sented by any group of workmen bore a definite relationship to the severity of such silica exposure. In other words, the symptoms and progress of the disease were directly proportionate to the amount of silica which invaded the pulmonary tissue. It was conspicuously apparent that in communities where men were exposed to a severe silica hazard, the incidence of and mortality from tuberculosis was extremely high, in some instances so high as to be hardly credible. This tuberculosis mortality, however, was in sharp contrast with that of surrounding communities and even with that of the workers' families.

There was built up a concept of an occupational disease resulting from the inhalation of free silica dust, manifested primarily as a fibrosis evenly distributed throughout both lungs, which, dependent upon the circumstances of exposure to silica dust, would progress with the gradual development of disability. Secondarily, especially where the silica damage to the lungs was of a considerable extent, tuberculous infection was apt to appear. Infection with the tubercle bacillus was more than merely a terminal episode and these two conditions reacted upon each other, definitely increased disability, and frequently led to a fatal termination. When death occurred as a result of pulmonary involvement in the great majority of cases, infection with the tubercle bacillus was present.

There came to my attention within the past month the report of the Miners' Phthisis Prevention Committee of Johannesburg, South Africa, for the three-year period 1935-1938. The authors summed up the experience of thirty years' study of silicosis in the gold mining industry and it was interesting to note how largely the observations concerning the etiology and clinical course of silicosis made in the early studies in this country had stood the test of time. Laboratory researches which have been carried on in this and other countries have modified our earlier ideas as to the manner in which silica affects the lung and have greatly increased our knowledge of the pathology of dust disease. Modern roentgenology has made possible early and accurate diagnosis and also has enabled the physician to watch the progress of affected individuals.

I might state here that organic dust has not been shown to produce a reaction of the pulmonary tissue and the pronouncement of Landis some twenty years ago that organic dusts played no part in causing pulmonary fibrosis has held good.

Attention was naturally directed to other industries which involved exposure to dust containing silica in many forms—that is to say, combined silica—as well as exposure to other types of non-siliceous inorganic dust. Here investigators found that the picture of disability and disease seen among those exposed to free silica was conspicuously absent. Various terms such as chalicosis and siderosis fell into the discard and other designations, such as potters' rot, grinders' rot, miners' phthisis, and miners' consumption were found to relate to identical conditions and were supplanted by the term silicosis.

Field studies of various industries went hand in hand with animal experimentation in which were employed dusts similar to those found in industry. Animals were exposed to the inhalation of these dusts in specially prepared dusting chambers and Gardner, at the Saranac Laboratory, was the first to demonstrate silicosis in experimental animals. The method of intraperitoneal injection, with the technique of Miller and Sayers, was also used. The underlying motive for all this was to determine which industrial dusts were harmful, that is, would cause a pneumoconiosis of such a type or degree as to produce disability.

Among the dusts investigated both in industry and in the experimental laboratory were limestone, calcium carbonate, gypsum, cement, feldspar, soapstone, talc, precipitator ash, artificial abrasives, sericite and others. None of these, whether they contained silica in combined form or no silica, produced disease in animals, nor was evidence of disease found among workmen exposed to these dusts except in those instances where free silica was also present.* That the failure to find evidence of pulmonary disease among workmen was not due merely to lack of a sufficiently severe exposure was indicated by the uniformly negative results in animals where the conditions of exposure were known and controlled and similar to those of silica exposures. X-ray films of individuals exposed for a number of years to these inorganic dusts, which contained no free silica, may show peribronchial thickening or accentuation of the bronchial tree. The same appearance can be found in individuals who have never been employed in dusty industries. Occa-

*Talc is one exception. See U.S.P.H.S. Bull. 1935.

sionally x-ray films of some of these workers will show a diffused haziness or increased density that might be construed as due to the action of dust. However, these workmen are healthy and neither symptoms nor disability can be ascribed to this x-ray appearance. Hence, my opening comment that pneumoconiosis, as a generic term, has a limited importance from a clinical standpoint.

Two specific and distinct types of pneumoconiosis are important, namely silicosis and asbestosis. Asbestosis is the exception to the general rule in that it is a silicate, a form of combined and not free silica, which does produce a characteristic fibrous reaction in the pulmonary tissue and which may produce disability and death. In animal experimentation, intraperitoneal injection of asbestos dust produces an inert reaction—that is, there is no cellular proliferation as is observed with the various forms of silica. Inhalation experiments produce a pulmonary fibrosis similar to that in human beings, with the curious anomaly that fibrosis did not result when the finer dusts were used. This has led Gardner to believe that the action of asbestos in the lungs is probably mechanical and due to the peculiar shape of the asbestos spicule and the constant movement of the lungs. Further support is given to this theory in that, unlike silica, asbestos will not produce its characteristic reaction in animals in other than the pulmonary tissue.

Asbestosis first came into prominence in this country about 1930, since when a number of industrial studies have been made. The symptoms are largely similar to those of silicosis but the pathology and the appearance on an x-ray film are entirely different. The x-ray shows a diffuse haziness or ground glass appearance, especially in the lower half of the lungs; nodulation is absent, and the factor of infection appears to be much less important than in silicosis, at least that is our experience in this country. The total number of workmen exposed to asbestos dust inhalation is very much smaller than for silica and strenuous efforts have been made by asbestos plants to control their dust hazard. Asbestos fabrication is a factory industry, largely textile in character, and the control of dust is less difficult than the control of silica dust in mines and foundries. Consequently, there has not been and probably never will be, the opportunities for the clinical study of asbestosis such as have been made possible by the wide variety and extent of silica occupations.

A small number of deaths from asbestosis have been reported and not all of these have been followed by postmortem. Mostly other organic disease was also found or diagnosed, which tends to confuse the rôle of asbestos in these cases, but in some cases death undoubtedly resulted from uncomplicated asbestosis.

There has not been sufficient opportunity in field surveys of asbestos plants to acquire enough information to correlate the production of asbestosis with varying degrees or intensities of dust exposure nor have the conditions of dustiness, as evidenced by particle counts, run as high as in the more severe types of silica dust exposure. We have had under observation for ten years a small group of workmen with asbestosis. Some of these have shown a progression on their x-ray films. They are still working and in seeming good health. However, in the meantime the plant has been cleaned up and the dust controlled so we do not know what would have been their history had they continued to work under the former, presumably hazardous conditions.

Our original concept of silicosis was based on studies of industries in which there was generally a more or less severe exposure to dust containing a high percentage of free silica. As industrial investigations came to include a wider variety of industries, our concept broadened and underwent some modifications. The data gained in industrial surveys, when correlated with laboratory findings, cleared up a number of apparently conflicting and puzzling observations. Not only did we learn that the incidence and progress of silicosis in any location depended upon the amount of silica dust in the air and the length of exposure, but it was demonstrated that in many instances other substances were present in the dust with the silica and that these substances modified the action of silica. Furthermore, it was shown that the action of silica was not due to its physical properties but to its chemical properties. Also, where certain substances had been accused of causing a pneumoconiosis, the offending agent was in fact the silica which was also present. Metallic dust, for instance, which in the early writings had been considered as more dagerous than silica, was found to be relatively harmless. There came a better understanding of what caused the variations in the clinical appearance

and course of silicosis as observed in different locations and in different industries. Men engaged in iron ore mining and in quarrying and cutting granite are exposed to dust containing silica and other substances as well, which tend to retard the action of silica upon the lung tissue. The pathologic process is altered and the appearance on the roentgenogram is altered sufficiently for the experienced physician to distinguish one type of exposure from another. With the mixed dusts, the progress of silicosis is usually slower and when infection occurs, it tends to be more chronic but the amount of tuberculosis found among granite workers and iron ore miners is nevertheless inordinately high. Cases have been reported of workmen exposed to dust containing silica and caustic alkalies which produced a rapidly developing and fatal type of silicosis with early infection.

On the other hand, certain industries have been studied in which the silica hazard was found to be of a degree which would not produce disabling silicosis within the average working lifetime. After forty or more years of such exposure, many workmen would show typical nodulation on the roentgenogram, but their symptoms were few or absent and they were still working every day, many of them being well over sixty years old. All of this emphasizes how necessary it is to study the etiological factors in order to arrive at a correct solution of the silicosis hazard in a given location or in a particular industry. It also explains the apparently conflicting conclusions that are sometimes drawn from isolated cases of silicosis when the work conditions, that is, the nature and type of exposure, have not been or cannot be determined.

The pneumoconiosis of coal miners illustrates very well the modifying action of one dust upon another. Miners' asthma had for years been recognized by physicians in the anthracite industry as a definite clinical entity. It was difficult to believe, however, that coal, an organic substance, could produce a disabling pneumoconiosis. The Public Health Service made a thorough study of the anthracite industry in 1933 and the results of this confirmed what many observers had suspected, namely, that disabling miners' asthma was, in fact, a silicosis; a form of silicosis modified by the combined action of silica and anthracite dust, and to which the term anthraco-silicosis might justly be applied. The incidence and progress of disability was, as in other silica dust hazards, proportionate to the extent of dust exposure. The silica was accounted for by the fact that the veins of coal lie between rock formations which frequently have a fairly high silica content, and, also, that there was some silica in direct association with the coal itself. Consequently, the workers had some silica exposure. Anthracite mine dust varied in its silica content from about 11 per cent for miners working directly in coal to about 60 per cent for men working in rock, with various types of exposures falling between these two extremes. Pulmonary tuberculosis was associated with anthraco-silicosis to a very considerable extent.

Below the age of thirty-five, hard coal miners showed an incidence of pulmonary tuberculosis slightly less than that found among male workers in general. In the age group of thirty-five to forty-four, the tuberculosis incidence was twice as great, and from forty-five to fifty-four, five times and above fifty-five about ten times the rate for all adult male workers. Or, to express it in another way, clinical pulmonary tuberculosis was found in 15 per cent of those with early anthraco-silicosis and in 43 per cent of those in the more advanced stages. The x-ray appearances, while similar to the silicosis of hard rock miners, can usually be differentiated from the latter.

With respect to bituminous coal mining, the silica factor is apparently less in extent or lacking entirely. A number of studies have been made by the Public Health Service but have not yet been published. Death certificates of soft coal miners were analyzed for a five-year period and showed no appreciable difference, with respect to deaths from tuberculosis, between the coal miners and the farmers living in the same counties.

Inhalation experiments with bituminous and anthracite coal (1.76 per cent SiO_2) have produced a very slight reaction and anthracite dust proved inert on intraperitoneal injection. We may conclude that in the absence of silica, coal will not produce a specific fibrosis associated with disability.

The relationship between pneumonia and silicosis is not entirely clear. The data obtained from various studies and from animal experimentation are not always consistent. The high incidence of pneumonia in some industries with a silica hazard can not be disassociated from ex-

posure to extremes of heat or cold or both. Coal miners have a high incidence of pneumonia and it is a common cause of death among the older men with anthraco-silicosis. My own impression is that individuals with any considerable amount of silicosis have a more unfavorable prognosis in pneumonia than would otherwise be expected but this experience is not very recent. It remains to be seen whether silicotics respond to specific serum treatment or chemotherapy as well as do those who do not have dust fibrosed lungs.

Summary

There is still much to learn about the action on the lungs of many kinds of dust. The effects of pure silica (SiO_2) have been demonstrated under many conditions in this and other countries with a high unanimity of agreement among investigators. Where the silica damage to the lungs is extensive, the incidence of tubercle infection is high. Where the silica exposure has been extreme, a tuberculous infection is almost inevitable, but every physician who has had wide experience with these cases has seen exceptions to this rule.

Today in American industry, severe exposures are uncommon. They occur mostly as isolated instances as when a sandblaster has been working with faulty protection. Tuberculo-silicosis may run a very chronic course, but if the silica damage is extensive, the progress may be fairly rapid, but many exceptions will be found to the general rule.

We are acquiring a constantly broadened knowledge of the action of mixed dusts, that is, dusts containing other substances along with the silica, but the more intense the silica exposure, the more these cases run true to type. Naturally the clinical picture of silicosis is changing as industrial dust control becomes more and more perfected so that we may expect to see more and more of the type or degree of silicosis which is accompanied by little disability. I am convinced, however, that where the reaction has proceeded to cause nodulation, even though moderate in extent, a definite, increased susceptibility to tuberculosis takes place.

Finally, we must consider the criteria upon which the diagnosis of silicosis can be based. Undoubtedly, the early manifestation of silica dust reaction is peribronchial thickening with an increase of the hilium shadows, as seen in a roentgenogram. The same may be seen in workmen exposed to dusts other than silica and in individuals who have never had any sort of dust exposure. Consequently, those of us who got together several years ago under the leadership of Dr. Pancoast, felt that for practical purposes, a diagnosis of silicosis was not justified unless the specific reaction of nodulation, as found in experimental animals as well as in human subjects, was present.

In addition to a characteristic film, there must be a history of exposure to silica dust adequate to produce disease. Where an industrial investigation is being made, and the working conditions previously determined, it is usually not difficult to separate the silicotics from the non-silicotics and usually the presence of infection can be recognized or ruled out, careful consideration being given to the physical signs and the symptoms as well as the x-ray film. The isolated case that may turn up in hospital or clinic service may present considerable difficulty. Such patients are apt to be suffering from a combination of illnesses. They usually cannot give a sufficiently informative work history and even with a characteristic film, it may be difficult to say that a patient has silicosis (or even more difficult, asbestosis) and to what extent his pulmonary condition may be responsible for his symptoms or his disability.