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SILICOSIS FROM THE PUBLIC HEALTH AND ECONOMIC VIEWPOINT*

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SILICOSIS is essentially a chronic progressive disease, characterized by an extraordinary tendency to tubercle infection. It occurs only as an occupational disease, and the industrial processes associated with exposure to silica dust vary widely and are distributed over many dissimilar industries.

The time factor in silicosis is a lengthy one. This, together with its progressive nature, tends to complicate and confuse our efforts to deal with the silicotic patient either when he is an applicant for compensation benefits or the subject of litigative or legislative procedure. Obviously, the rate of development in a disease caused by the inhalation of dust is determined by several factors. There are the amount of free silica in the dust, the amount of dust in the atmosphere, and the extent of exposure of the individual. We estimate the severity of the hazard in any given case by weighing these three factors.

Where the amount of free silica in the dust is very high—80, 90, or 95 per cent—and the quantity of dust excessively large and the exposure constant, then the physician sees the severe or, as Gardner has termed it, the classical form of silicosis, which develops in from five to ten years, progresses steadily, and almost inevitably is complicated with a tubercle infection which runs its course fairly rapidly. This is the type of silicosis described in the early literature and found especially in mines where the rock was highly siliceous and drilling was done dry and no adequate preventive measures were enforced—a type of silicosis which is disappearing and will soon be relatively infrequent in this country.

Spread throughout industry are many dust hazards in which the combined weight of the three factors is less than under the conditions just described—the amount of silica may be relatively low—15 to 50 per cent—the quantity of the dust may not be so excessive, the exposure may be less constant, and more or less effective efforts at dust control may be attempted. In this situation, the clinical picture is one of very slow development, made evident only in the higher age groups, with little disability in the absence of infection, and a less pronounced incidence of tuberculosis.

In between the very severe and the mild types of silicosis are many gradations, resulting from the emphasis or lack of emphasis of one or more of the factors mentioned. I desire to stress the importance of establishing and evaluating these causative factors in every case of silicosis. The failure to do so has been largely responsible for much of the misapprehension and

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confusion that have characterized attempts to deal with the silicosis problem during the last few years.

The extent of the exposure to the silicosis hazard is greater than might be supposed. The following figures are quoted from a study of the subject by Vane and the author.¹ We estimated that there are about 115,000 miners in metal mines and coal mines exposed to silica dust—by no means all of the men employed in these industries. Workers in smelters, foundries, glass works, together with those employed as grinders, sandblasters, and vitreous enamellers, would account for about 325,000. Including also various other occupations with a known silica hazard, we arrived at a total of not less than 500,000 workers exposed to silicosis in this country and this, in our opinion, is a very conservative estimate.

At any rate, the burden of silicosis, both in sickness and in cost, is staggering. From the viewpoint of public health and preventive medicine, it is a shining target to shoot at. The situation is not new. What is new is the attention that has been focused on pulmonary dust diseases in the last five years, with the consequent tendency to include silicosis among the compensable diseases. The difficulties of applying the principles of compensation to an insidious and chronic disease are apparent. It is not my purpose to discuss them here. But it would seem that if these vexatious difficulties are to be overcome, a competent medical board should be an integral part of the compensation mechanism. It is, perhaps, too much to hope that there should be uniformity in the practice of the different States with respect to a standardized diagnostic procedure, but unless competent medical assistance can be made available in carrying out the provisions of occupational disease compensation laws, they cannot be administered with justice nor with a proper regard for the public interest.

The control and prevention of silicosis is, in the main, a medical problem. Upon the physician falls the responsibility of accurate and precise diagnosis—first, to determine whether the applicant for work, where a dust hazard may be present, is free from pulmonary or other disease which should bar him from such employment; and second, to discover as early as possible the indications of harmful effects among those employed. As a judge, deciding who should or who should not work in any given occupation, where an occupational disease hazard is involved, the physician renders a service to the public health as important and exacting as any professional function he may be called on to fulfill. I dwell upon this because it seems inevitable that the physician is going to be interjected into the industrial picture to an increasing extent. While it may be desirable to set up standards of employment in this matter of occupational disease hazards, such standards must be very general. There is no substitute for the personal judgment and experience of the physician, coupled with a first hand knowledge of working conditions. Each individual is a case in himself, and social and economic conditions may demand consideration as well as his physical condition.

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The mitigation of the dust hazards falls within the realm of the engineer but the proof of the efficiency of engineering control methods must be determined by the presence or absence of specific pathological changes in the individuals involved. In any situation where silica dust may be a hazard, the information we seek is the percentage of silica in the dust and the number of million particles per cubic foot of air in the working place. The method of sampling and analyzing dust air in working places has been standardized by the United States Public Health Service and the United States Bureau of Mines. Their technic has been generally adopted by competent investigators. The air sample is taken with the Greenberg-Smith impinger, large samples averaging about 100 cubic feet being secured on a level with the workman's face and at a rate approximating that of normal breathing. The sample is analyzed for free silica, and from microscopic preparations the particles are counted and the size-frequency determined. Under most industrial conditions, about 90 per cent of the particles are under ten microns in size. While dust determinations do not come within the physician's duties, he should be able to evaluate the information obtained, otherwise he cannot determine who should be employed and who should be rejected, nor who should be transferred to another job, and who retained.

Workmen exposed to a silica hazard should be examined periodically. An annual examination may be sufficient but the judgment of the physician must be the deciding factor and he should have the right to reexamine those under his care as often as he deems advisable. When a workman is found to have silicosis, he should be protected from further dust exposure—if necessary transferred to another job. When a silicotic becomes tuberculous, he should be placed upon complete disability, both in his own interest and that of his fellow workmen.

From the point of view of diagnosis the value of the roentgenogram has been stressed. While it is true that a roentgen-ray film of the chest is essential, it should be recognized that there is a tendency to disregard other necessary and important diagnostic information. It is a common occurrence for films to be sent to our office for confirmation of a diagnosis with no case history or clinical data whatsoever. A complete occupational history, as well as a clinical history, and a thorough physical examination are necessary if the film is to be correctly interpreted. It is also vitally important that the physician who attempts the diagnosis of silicosis be familiar with the general appearance of chest films of working men so that he may be conversant with the various appearances that are compatible with what has been aptly termed "the healthy chest."

The attention that has been centered upon the various pneumoconioses in the last few years has revealed the tendency to disregard occupation as one of the important factors in the clinical history of patients who come from the wage earning class. It is unusual to find an accurate occupational history in either private or institutional records, which is one of the reasons why occupational diseases are not diagnosed more frequently. Espe-

cially where the disease is essentially chronic, as in silicosis, a careful building up of the work history of the patient, from the time he left school to earn his living, may be the only key that will unlock a diagnostic puzzle.

I would also like to point out that the clinical picture of silicosis among negroes is not the same as among whites. The negro is not as resistant to silica dust, he becomes tuberculous more readily and he succumbs more quickly. Where the silica dust exposure has been very severe, the clinical picture among negroes may approach the acute in type whereas among white men, working on the same job, it will be characteristically chronic.

An important phase of the silicosis problem is the incidence of tuberculous infection among the families of silicotics. In the early literature, especially that relating to the mining industry, the widow who had buried several husbands with miner's consumption was much in evidence. From this arose the general belief that there was not much spread of infection from silicotics with tuberculosis to their families. Undoubtedly, the situation varies locally and is influenced by race, general hygienic conditions and the severity of the hazard.

As a rule, the silicotic does not live long after his tuberculosis becomes open. Cummings of Saranac, in a study made in the Picher, Oklahoma District (unpublished) found evidence of tubercle infection in over 50 per cent of the children under 16 living in contact with silicotic patients having a positive sputum, but the infection was entirely of the primary type and there was no progressive adult tuberculosis. The whole subject of infection needs further study. We do not know what is the nature of the affinity between silica and the tubercle bacillus.

It is, unfortunately, a characteristic of silicosis that it tends to progress even when there is no longer an exposure to silica dust. The workman with a considerable degree of silicosis has an unfavorable prognosis even though he is removed from further dust exposure. He will probably succumb to tuberculosis. Where the damage to the lungs is slight, or moderate, he may go on for many years with little or no disability, but the overall mortality from respiratory diseases, particularly tuberculosis, among the silica dusty trades bears ample witness to the harmfulness that lies inherent in exposure to silica.

One of the difficulties that is constantly present in dealing with individual cases of silicosis is the estimation of the degree of disability. We would expect this in any chronic disease. The roentgen-ray film is not a satisfactory criterion. There may be evidence of a considerable amount of pathologic change in the film and yet the patient may look fairly well and be conscious of little impairment of his working ability. Chest expansion gives us no clue and little can be estimated from the patient's statements concerning dyspnea except in the advanced cases. Furthermore, it is not always possible to be certain of the presence or absence of infection—either from the symptoms or the roentgen-ray film. As a result, there has been a definite tendency to award compensation or other benefits on the basis of

a diagnosis rather than on actual disability—a most unsatisfactory condition of affairs. Some promising research has been started for the purpose of estimating impairment of pulmonary function without as yet producing results that can be converted into procedures for the clinician.

To sum up, silicosis is a widespread disease and is responsible for much tuberculosis among wage-earners. Its detection and control depend upon the accuracy and timeliness of diagnosis. As with other phases of the public health, successful achievement rests with the physician.

REFERENCE

1. LANZA, A. J., and VANE, R. J.: The prevalence of silicosis in the general population and its effects upon the incidence of tuberculosis. *Am. Rev. Tuberc.*, 1934, xxix, 8-16.