SILICOSIS*

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SILICOSIS is purely an industrial disease, affecting the lungs of persons whose occupations expose them over long periods to the dust of hard rock. The disease has received a great deal of attention in Africa, where for many years it has been a serious economical problem in the mining industries. The South African Institute of Medical Research and the Miners' Phthisis Bureau of Johannesburg have published the most important contributions to the literature on the subject, and much of our knowledge of the disease is derived from this source. The largest silicotic community in America centres around the granite-cutting industry of the New England States, and to the investigators in this field we are also indebted for much valuable information.

Silicosis became a public health problem in Ontario only within the last six years. The first case was compensated in 1924, on the medical evidence of Dr. C. D. Parfitt. In the same year Dr. J. H. Elliott published the first Canadian contribution to the study of the disease. His report dealt with a survey of the Porcupine mines made at the request of the Ontario Government. The examination was limited to 11 men who had worked underground continuously for five years or more in the same area. Four of them were found to have definite silicosis; an additional 5 presented evidence of early fibrosis; only 2 were considered entirely free from lung involvement. Dr. Elliott's investigations established the presence of definite health hazards in the gold mines of Ontario. As a result, the Provincial Department of Health quickly assumed a lively interest in the matter, and organized surveys of all the important mining camps. Diagnostic clinics were established, means of preventing the disease instituted, and the mechanism of compensation set in motion. Up to January of this year 182 miners have been granted benefits by the Workmen's Compensation Board, on the grounds of total or partial disability from silicosis. In addition, a few granite cutters and others employed in various hazardous trades other than mining have received compensation for the same condition.

With an ever-increasing number of cases under clinical observation, autopsy material has, from time to time, been available for laboratory study. In the Department of Pathology of the University of Toronto, we have had the opportunity of examining the lungs of some 17 proved cases. The majority of these were handed over to us by Dr. A. R. Riddell, of the Department of Health, and to him belongs the credit for obtaining the autopsies. Under the direction of Prof. Oskar Klotz these cases have been studied from the pathological point of view, and the present communication is mainly concerned with the pathological findings.

Certain general considerations must, however, be taken into account in order to obtain an accurate appreciation of the nature of the disease. In the first place, it is important to realize that silicosis is by far the most serious type of dust disease met with. It stands by itself when compared with the less serious conditions produced by coal or iron dust. Indeed, many authors believe there is really no such disease as anthracosis or siderosis; that when appreciable fibrosis appears in the lung associated with dust inhalation, it is always due to silica. But, whether this be true or not, it is evident that the injury induced by other dusts is trivial compared with that due to silica.

One must bear in mind, too, that silicosis is characterized by an extreme chronicity. It never develops in less than four years' exposure, and massive inhalations are necessary to produce it in so short a time. If the dust risk is not intense more than twenty years of

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exposure may be endured before the characteristic lesions develop. The reserve power of the respiratory system to take care of inhaled dust particles is enormous, and it is only when the natural resources are taxed to the utmost that injury supervenes.

The disease runs entirely to fibrosis of the lung. So far as is known, no other part of the body is directly affected. It is a disease, which, in its uncomplicated form, practically never of itself causes death. Even in a pronounced case the affected person may appear robust, feel well, and be able to work, except that he gets rapidly out of breath on exertion. None the less, silicosis is a very serious affection because it renders the victim particularly vulnerable to pulmonary infections.

The offending dust is of one variety only, namely that containing appreciable quantities of free silica. In order to produce its deadly effect, silica must be present in uncombined SiO_2; if it exists as a silicate, *e.g.*, in clays, shale, slate, or limestone, the dust is not harmful. Silica is a crystalline substance, one of the hardest compounds of the element silicon, and it constitutes a large part of all hard rock. Quartz is made up almost entirely of free silica. Thus, any industry which has to do with the fracture of hard rock is a hazardous trade from the standpoint of silicosis. When fractured the rock liberates myriads of tiny fragments which float about in the air. The smaller the fragments the more harmful they are to the lung; in fact the whole of the mischief is caused by particles under 10 microns in size. It is very difficult to control such fine dust. Even under the most favourable conditions with sprinkling and running water the dust escapes into the air in an invisible form.

Chemically, silica is rather inert. It is insoluble in water and in all the acids except hydrofluoric. It can be dissolved by boiling the pulverized form in alkalies, for example, sodium carbonate solution, and this is how water-glass is made.

It has never been determined why silica alone, of all the inorganic trade dusts, should produce such a serious pathological condition of the lungs. Many theories have been advanced to explain its pathogenicity. Obviously, the fibrosis is something more than a foreign body reaction. The earlier workers considered that silica produced a mechanical injury in the lungs by virtue of the hardness and sharpness of its fragments. Plugging of the lymphatic vessels of the lung by dust-laden cells has been held by some authors to be the reason for fibrosis. The South African workers believe that the silica, when ingested by phagocytes, takes on the fixative properties of water-glass and virtually mummifies the dust-laden cells, thus giving rise to fibrotic induration. A more modern view is that particulate silica undergoes a slow change in the pulmonary tissues, becoming slowly converted into a toxic substance which by chronic irritatation produces a fibrous response. There is some experimental evidence to show that certain compounds of silica are protoplasmic poisons, and it is considered likely that the tissue juices may so act upon silica dust within the lung as to slowly produce such a toxic substance. Histological evidence bears out this conception. Recent dust collections seen under the microscope contain an abundance of visible silica particles, but in the older deposits where fibrosis is under way, the fragments disappear. It only remains to conjecture that slow dissolution of the siliceous particles is coincident with the development of fibrosis.

This conception of the pathogenesis of silicosis has an important theoretical bearing on the clinical progress of the disease, for it follows that silicosis must be progressive after a man leaves his dusty occupation, for the reason that much unaltered silica is already fixed in his lung, and it will continue to stimulate slow fibrosis for a long time. Dr. J. G. Cunningham tells me this theory is borne out in practice. Many cases progress markedly after retiring from the hazardous trades.

So much for the consideration of uncomplicated silicosis. There remains the all-important matter of the relationship between it and tuberculosis, which we may now discuss briefly.

The majority of sufferers from silicosis eventually develop tuberculous infection of the lungs, and the combination almost always proves rapidly fatal. The mortality from phthisis amongst hard rock miners and granite cutters is strikingly high. Eleven of the 17 cases we have examined died of tuberculous bronchopneumonia or miliary tuberculosis, primary in the lung.

It is only in the experimental field that one can hope to explain the unfortunate vulnerability of silicotic tissue to tuberculous infection, but unhappily researches in this direction have shed little light on the problem. It is known that silica does not stimulate the growth of tubercle bacilli *in vitro,* but *in vivo* tuberculous infection is profoundly influenced by the presence of silica. The infection runs wild and the organisms multiply inordinately, but the reason for this is a complete mystery. Some authorities believe that the silica, by exerting a toxic action, shrouds the colonies of bacilli in a protective medium of devitalized tissue into which phagocytes do not readily penetrate. Others believe silica *in vivo* acts as a specific stimulus to the reproduction of tubercle bacilli. A very recent theory postulates that silica, by virtue of its colloidal properties, absorbs antibodies and so prevents the organisms from being attacked.

Our examinations tend to show that it is not the fibrosis of silicotic lungs which favours a tuberculous infection, but simply the presence of siliceous dust. Fibrotic tissue is naturally inimical to tuberculosis and seldom are the indurated portions of lung attacked by the infective process. Three of our cases were in the early stages of silicosis, having considerable dust in their lungs, but with no appreciable fibrosis; these three died, each with a massive bilateral caseous involvement, and there is little doubt but that the silica was in some strange way contributory to the fatal outcome.

If there is much fibrosis present when tuberculosis becomes superimposed, the reaction to the infection takes the form of widespread connective-tissue proliferation, which leads to massive induration, almost like an irregular fibroid tumour growth. If, on the other hand, infection takes place early in relation to the pneumokoniosis, caseation is the rule. From the radiologist's point of view, it is often very difficult to determine if and when tuberculosis has become superimposed on a silicotic lung. In this regard, autopsy findings may facilitate the interpretation of the roentgenogram. The lesion of simple silicosis runs to a finely nodular type of fibrosis, involving both lungs more or less uniformly and symmetrically. When, therefore, the x-ray shadows indicate that the nodules are matting together into irregular, symmetrical areas of induration, this may be taken as certain evidence of an infective process.

In consideration of the relationship between silica and tuberculous infection, it is interesting to speculate as to whether silica, which is a ubiquitous substance, may not play a part in the etiology of tuberculosis in the ordinary walks of life.

TREATMENT OF SEA-SICKNESS.—P. Vogt-Moller has studied the problem of sea-sickness during twenty-four crossings of the Atlantic in 1928 and 1929. He found that more than three-quarters of his patients were women, whose sea-sickness was more severe and protracted, as a rule, than that of the male patients. The best purely mechanical device proved to be keeping them as near to the centre of the ship as possible, since at this point the movements of the ship were less than anywhere else. The drugs tested were morphine, veronal and its derivatives, bromine salts, adrenaline, cocaine (used as a local anæsthetic for the stomach), strychnine, camphor, caffeine, chlorotone, benzyl-benzoate, sodium nitrite, and atropine. Neither the severity nor the duration of the sea-sickness was influenced by any of the drugs in this list when they were swallowed; even atropine by the mouth proved quite ineffective, though it had some effect when given by subcutaneous injection. The combination found most successfuly was a 0.1 per cent solution of atropine sulphate with a 0.1 per cent solution of scopolamine bromide, given subcutaneously or in suppositories. The dose of the atropine was from 0.5 to 1.25 mg., and that of the scopolamine from 0.25 to 0.5 mg.; no ill effects other than slight dryness of the mouth, easily relieved by water, were observed. Even when the temperature of the air was tropical there were no discomforts from interference with the secretion of sweat. Although the results of this treatment were described as amazingly good, the effects of a single injection or suppository were not necessarily immediate, but after three or four injections, distributed over a couple of days, most of the patients could leave their berths and felt well. In severe cases the average duration of the sea-sickness was reduced by three days by this treatment. In many cases only one or two injections or suppositories were necessary in order to restore the patients to a complete sense of well-being, even though the sea remained rough.—*Ugeskrift for Laeger,* p. 700, July 17, 1930.