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This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests, and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited. The editors will exercise care in checking on the accuracy of data printed but in all other respects articles and opinions of which expression is allowed are the opinions of their authors—the editors reserving in all cases the right to comment on the same in the current or any subsequent issues as they may be inclined.

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Pulmonary Dust Disease

A. R. RIDDELL, B.A., M.B., D.P.H.

OPINIONS concerning dusts and pulmonary disease have changed somewhat during the last few years. For a considerable period following 1918 attention was chiefly centered on silica. Dusts were classified as active or inert according as the reaction they occasioned in the lungs resembled or differed from that produced by silica. Although silica is still recognized as the most important dust concerned with dust lung injury, it has gradually become apparent that dusts other than silica and the silicate asbestos may be involved in pulmonary disorders.

A description of silicosis is superfluous at present. The disease has received so much attention and has been discussed so often that you are all conversant with its general features. You realize that silica dust inhalation, if the quantity of dust inhaled is adequate, may result in lung fibrosis of a special nature. Collagenous nodule formation is characteristic of this process, but its regular deposition may be altered often profoundly by accompanying infectious processes, or by admixture of the dusts of other materials with silica. It is general knowledge that silicosis is prone to association with tuberculosis, but the implication may not be so fully appreciated.

The late DR. GARDNER¹ is reported to have advanced the opinion that, when chronic, a combination of silicosis with tuberculosis constituted a disease quite different from either of the other two contributing conditions. Certainly it is difficult to treat. As a rule it gradually progresses in spite of what is attempted, and surgical measures are unsatisfactory. This disease may be seriously disabling, although those affected are not always obviously ill.

The importance of tuberculosis in conjunction with silicosis is illustrated by information supplied by consideration of the causes of death in a series of 436 silicotics on whom we have records. The persons concerned had been employed in various occupations carrying a silica hazard. A little more than half of them were miners. Pulmonary tuberculosis caused the deaths of 62% of the persons comprising the whole series, and was responsible for 68% in the mining group.

Estimations of disability are notoriously troublesome in silicotics. It would seem that these are too often based on radiographic appearances together with attempted measurements of breathing capacity, and that other useful information is apt to be neglected. In assessing disability the clinical picture should be viewed as a whole. Careful consideration of the information afforded by the symptomatology and clinical findings in the individual cases, may be of

considerable value in the estimate of disability.

A review of the symptoms and signs presented by 637 persons with silicosis was recently made. This offered information which may be usefully applied. The investigation confirmed the finding that dyspnea is the commonest symptom and limited expansion the commonest sign. It also disclosed, that of 203 persons with a history of having had night sweats, 117, or approximately 58%, had positive sputum. Bacilli were demonstrated either at the original examination, or later on follow up. It indicated, that 70, or 80%, of those with hemoptysis were tuberculous, and that 184, or 75%, of those whose fingers were clubbed or who had curving of the finger nails, had positive sputum, or were dead when the analysis was made.

It will be realized that for practical purposes, diagnosis in silicosis involves the differentiation of type as well as recognition of the disease. Commonly this is made on the radiographic appearance a case presents. Cases with clear cut uniform nodulation are interpreted as simple, and those with conglomerate or massive shadowing as complicated. Although this may be a useful working rule in relation to the interpretation of radiograms, there are many exceptions to it. Not infrequently the radiographically simple case will prove to be complicated, and no evidence of an infectious process can be demonstrated in the radiographically complicated, even at autopsy.

Compensation complicates the silicosis problem. With diagnosis so dependent on the interpretation of radiographic shadowing as is the case at present, it is well to remember that heavy or prominent shadowing in the lung fields or even nodulation, does not necessarily denote fibrosis or silicosis. These appearances, commonly ascribed to the effects of silica, may be simulated by lung changes related to other conditions than silicosis, in which there is often no fibrosis. When cases presenting such radiographic appearance lack a history of dust exposure the situation may not be too difficult, but with such a history they may cause considerable confusion. We have found the more important of these confusing conditions to be—heart diseases, particularly mitral stenosis, certain fungus infections, benign pneumoconioses, disseminated new growths of the lung, and sometimes pulmonary tuberculosis with a miliary distribution.

Aluminum dust employed in the treatment of, or as a prophylactic in silicosis, has received much attention in this regard during the last few years. It is administered in two forms, as finely divided metallic aluminum, and as aluminum hydroxide. The metallic powder is the more important agent, and at present many persons are receiving it both here and abroad. Its adminis-

¹Presented at the Annual Meeting of the MICHIGAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS, Lansing, Michigan, March 2, 1948.

itation has not been observed to be attended by injury, neither however, is there convincing evidence that it has been of objective benefit to silicotics. Nevertheless, many persons receiving, or who have received aluminum for silicosis claim subjective improvement.

The status of aluminum as a therapeutic agent has not yet been determined. It seems to offer possibilities as a prophylactic, but as considerable time is still necessary to establish its usefulness, no laxity in regard to recognized methods of dust control should be countenanced.

Early in 1942 an occupational lung disease new to this continent was discovered by DR. C. G. SHAVER, Superintendent of a sanatorium situated near St. Catharines, Ontario. A description of the disease and the circumstances which revealed it has already been published.² The details need not be repeated. In the process involving bauxite, aluminum ore is converted to corundum, a hard metal used in the abrasive industry. While the process is in operation, the attendant workmen are exposed to dense fumes from the materials being treated.

The process is not new. It has been carried on for upward of 20 years in some of the plants concerned, yet it was not previously known to be injurious. This may be explained on the basis, either that in the past it did not occasion lung injury, or the disease was not recognized because its accompanying signs and symptoms were ascribed to other causes. The latter is the more probable. Early cases are symptom free, the signs, exclusively radiographic, are often so slight that they would be readily missed by one not conversant with the situation and on the look out for them. Later cases as characterized by attacks of sudden extreme dyspnea and chest pain which have, on occasion, been interpreted as indicating heart disease or pneumonia.

The radiographic characteristics of the condition may be divided into "early" manifestations and "well established" disease. The early manifestations are extremely slight. They consist of an indefinite granular appearance in the apices with slight rather lace-like shadowing which appears in the sub-apical area and which may extend to the first interspace. As the condition advances the lace-like shadowing is found extending lower in the lung fields. A further extension is accompanied by the loss of the lace-like character of the shadowing. Eventually the whole lung field may be occupied by shadowing which has a rather granular, or even an irregular nodular appearance.

Coincident with the peculiar lung shadowing the diaphragm is generally markedly distorted, and the mediastinum is widened. The "well established" case is characterized by the occurrence of spontaneous pneumothoraces, very often bi-lateral. These occurrences are associated with the rather dramatic appearance of desperate dyspnea and severe chest pain.

The fume is the source of this lung damage. It contains considerable quantities of very finely divided alumina and silica, and small amounts

of many other substances. Which of these is responsible has not yet been determined. In the presence of so much silica, this material cannot be absolved, on the other hand rather similar lung damage has been reported from Germany¹ in exposures in which aluminum at least figured. MIDDLETON⁴ also reported a case in England in which exposure to aluminum was associated with lung changes.

The inhalation of cadmium or cadmium compounds, either as fume or dust is attended by respiratory damage of a serious nature. In an Ontario exposure 15 workmen from two plants were injured in this way in 1935—two died.

Dust is produced when the metal is being ground or polished. Fumes are evolved during welding operations, or when the metal is subjected to high temperatures by accident, or for the purpose of removing cadmium plate.

When small quantities of cadmium are inhaled, dryness of the throat, cough, a sense of restriction in the chest, headache and vomiting are produced. Heavy exposure is followed by severe lung changes, accompanied by marked dyspnea, persistent cough, cyanosis and prostration, which may be prolonged. The disease has the characteristics of a chemical pneumonia, and this is confirmed by postmortem findings. Convalescence is apt to be protracted, but they eventually return to work without disability.

In one instance radiographs were prepared during the acute illness. In this, the lung fields were seen to be occupied by intense nodular shadowing, closely resembling that to be observed in certain cases of silicosis. Radiographic examination of the survivors of the affected was carried out some years later. In none was there evidence of any permanent lung injury.

It was during the war years that beryllium poisoning was drawn to our attention.⁵ Exposure to beryllium or to certain of its compounds is attended in certain individuals by skin troubles. It is also attended by lung disease, often of a serious nature. The disease has special features: Symptoms may develop during exposure, or they may be delayed, perhaps, for months or years after exposure has ceased. Two types of the disease are recognized, an acute type and a delayed. The acute form has characteristics which suggest a chemical pneumonitis. It results from exposure to high concentrations of beryllium compounds. It may be severe and even fatal, but in those who survive, the condition has a tendency to clear. The delayed form appears to have little relation to intensity of exposure. It is more apt to occur where relatively light, but more prolonged exposure has been sustained. A feature of the delayed form is the occurrences of scattered granulomatous areas in the lung tissue which may undergo organization. In some cases the radiographic appearances closely resemble silicosis, it was therefore thought at one time that the disease might be a modified silicosis. The etiology is still obscure. While investigating this matter, it was DR. GARDNER'S⁶ opinion that some other factor than beryllium