INDUSTRIAL SIDEROSIS

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IN text-books of medicine siderosis is usually defined as fibrosis of the lungs due to the inhalation of iron or steel dust. Actually the condition described is really silicosis, and this is not surprising, for many metal workers are exposed to silica dust. Siderosis used in this way merely denotes silicosis in a metal worker just as chalicosis denotes silicosis in a stone worker, schistosis in a slate worker, and so on. When iron and silica dusts are inhaled together it might be quite proper to describe any ensuing fibrotic condition of the lungs as a sidero-silicosis, but the present view is that the inhalation of iron dust alone, without added silica, does not cause fibrosis.

The inhalation of mineral dusts met with in industry result in varied and complex reactions. Some of them are locally caustic producing irritation of the upper respiratory tract, even to ulceration and perforation of the nasal septum; others irritate the bronchi or the lung parenchyma producing atypical forms of pneumonia, and some have long-term effects resulting in chronic changes in the lungs —diffuse in the case of asbestos, focal in the case of silica. Some have a carcinogenic action, some produce granulomata, and others appear to be able to lie quite inertly for long periods in the lung tissues without producing any permanent reaction by the tissues.

SILICOSIS

A description of silicosis is superfluous here. The disease has received so much attention and has been discussed so often that its general features are well known. Silica dust inhalation, if the quantity of dust is large enough and the size of the particles is small enough, results in lung fibrosis of a special nature. Nodules formed of dense fibrous tissue with a concentric arrangement of the fibres are characteristic, but the deposition of fibrous tissue may be altered profoundly by accompanying infectious processes or by other dusts which modify its action. The characteristic lesion of silicosis is the deposition of fibrous tissue in an amount which is redundant and far in excess of what is required merely to shut in the dust particles.

PNEUMOCONIOSIS OF COAL MINERS AND COAL TRIMMERS

For recent work on the etiology, pathology and the sociological effects of chronic pulmonary disease in coal miners we have to study the numerous reports from South Wales, although it is interesting to note that the earliest records came from Scotland (Gregory, 1831, Marshall 1834, Thomson 1836). Towards the end of last century and in the beginning of this century no attention seems to have been

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paid to the condition, and such authorities as Sir Thomas Oliver (1908) and E. L. Collis (1915) stated that the disease no longer existed. It was a period of obscurity and complacency—at least for everyone but the affected miners themselves. No compensation was payable to coal miners with respiratory trouble until 1929 and then only to workers who could prove that they had worked on silica rock. In 1934 the scheme was extended to cover miners working on the coal face and other underground workers, but still there continued much dissatisfaction. Increasing claims from the men and uncertainty as to the nature of the disease led to the Medical Research Council making an investigation which commenced in 1937. Reports were published in 1942, 1943, and 1945. These showed a serious incidence of radiological abnormalities, associated with symptoms and impairment of function, in coal-face workers and also, and this is important, in coal trimmers. This was interesting from the etiological point of view, for whereas it could be argued that the coal getter, working at the coal face, might inhale not only coal dust, but also dust from silica rock generated by other workmen, this could not be so in the case of the coal trimmers, who were exposed to dense clouds of coal dust in the holds of ships, but not to rock dust. The Medical Research Council investigators described an appearance in abnormal radiographs of a fine network—sometimes sharp and lacelike in appearance, more often blurred—which they called reticulation, and which was clearly occupational in origin. This condition was subsequently made compensatable by the Workmen's Compensation Act, 1943, which defined pneumoconiosis as "fibrosis of the lungs due to silica dust, asbestos dust or other dust, and including the condition of the lungs known as dust reticulation."

Our knowledge of the effects of coal dust in the lungs is due mainly to the work of Gough and his co-workers in Cardiff (Gough, 1947; Heppleston, 1947; etc.). They have shown that the essential lesion in the pneumoconiosis of coal workers is quite different from that of silicosis. In early pneumoconiosis of miners examination shows deposition of dust in the lymphoid tissue around the bifurcation of the respiratory bronchioles, but even in cases where there are X-ray changes there may be at first no tissue reaction. Later, a little fibrous tissue is laid down and the nodule retracts slightly assuming a stellate appearance. The fibrous tissue is, however, slight in degree and is not whorled. It may be said that this is merely a difference in degree depending upon the amount of silica in the dust inhaled. However, accompanying this slight retraction there occurs a well-marked emphysema in the air cells immediately adjacent to the nodule, thus forming a focal emphysema throughout the lung. This is a fundamental difference, for emphysema, for some reason that we do not know, is a relatively minor change in relation to the silicotic nodule while it is the most striking lesion in relation to the coal nodule. Curiously, also, it does not seem proportionate to the degree of fibrosis.

Inert Dusts

Certain dusts, among them iron and tin, appear to be quite inert when inhaled into the lungs. The particles are deposited in the lymphoid aggregations just as coal dust is. There they lie inertly without leading to any reaction or change in the tissues. They produce an abnormal X-ray picture because the aggregations become relatively radio-opaque by reason of their metallic content.

Electric and oxy-acetylene welders, working on iron and steel, are constantly exposed to fumes from the work. The heat of the welding arc boils the metal which vapourises and oxidises, and the fume therefore consists mainly of iron oxide particles in a very fine state of division. X-ray films of welders who have spent many years at this work may show dust reticulation, especially if they have performed a lot of work in enclosed spaces such as boilers or tanks.

The evidence that the dust which gives rise to such well-marked X-ray changes is inert may be considered under five headings :—

1. *Clinical.*—I have been interested in welders for 16 years and have examined many hundreds. In no case have I found incapacity or diminished capacity for work that could not be explained by other reasons. I have kept in touch with some welders showing dust reticulation for most of this period and find that they continue to be well and keep at work. They not infrequently admit to having cough, and often some sputum, but in my opinion these are partly due to other factors and not merely due to the deposition of iron-oxide particles in the lungs. They have no dyspnœa and exhibit no clinical evidence of fibrosis. They have a good chest expansion and exhibit a good tolerance for exercise. These opinions are amply confirmed by numerous investigators, not only as regard welders but for other workers who are exposed to iron dust (Britton and Walsh, 1940 ; Groh, 1944 ; Sander, 1944 ; Lanza, 1945 ; McLaughlin *et al.*, 1945 ; Barrie and Harding, 1947, etc.).

2. *Pathological.*—Only one report has been published in the world's literature of the post-mortem findings of a welder who showed the typical X-ray changes during life. This (Enzer and Sander, 1938) was illustrated by excellent microphotographs, which showed no fibrosis round the collection of iron dust. The man died of pneumonia complicating fracture of the spine, the result of an accident. The fact that no other reports have appeared in the literature speaks for itself and suggests that the condition does not shorten life. I have been able to obtain post-mortem material from two welders who, however, did not show the typical X-ray changes during life. Although the lungs showed small aggregations of iron particles, these were not associated with fibrosis.

Further pathological evidence of the inertness of iron dust has been produced by Harding and his colleagues who in successive papers describe the post-mortem appearances in detail of five silver finishers. These men had exposure to fine iron-oxide dust for 26 to 50 years and showed typical X-ray changes due to dust. In the first four cases careful microscopical examination showed that fibrosis was completely absent. In the last case (Harding, 1948) there was a minimal amount of fibrosis of the " reticulation " type—not at all like that produced by silica. Harding thinks that in this case individual susceptibility may have been of importance, but there is always the possibility

that the iron dust at some period of this man's long working life—over 40 years—may not have been pure and may have contained silica or other fibrosis-producing constituent.

3. *Experimental.*—Various experiments had been made subjecting the animals to the inhalation of iron-oxide dusts. Harding, Grout and Lloyd Davies produced X-ray changes in such experimental animals but examination of the tissues showed no fibrotic or other reaction. Cappell too found that "no ill effects follow massive accumulation of iron in the liver cells." Other workers have found that iron oxides injected subcutaneously (Von Haam and Groom) or intraperitonially behaved inertly. The late Professor Kettle's experiments might also be mentioned in this connection. He found that not only did iron alone produce no tissue reaction but that iron when mixed with silica prevented silicosis.

4. *Statistical.*—The Registrar-General in his Occupational Mortality Supplement shows that in 1931 in the group of welders and burners, numbering 11,542 in England and Wales, there were only 123 deaths compared with 161 expected on the basis of age-rates of all males. Unfortunately no more recent figures are available. I have, however, collected from various factories information regarding the sickness absence of welders in relation to other workers and find that welders are favourably placed compared with other groups, not only for total sickness, but also for respiratory illnesses. I have been asked to consider these figures as confidential and therefore unfortunately they cannot be reproduced here.

Collen and his colleagues (1944) give reliable evidence about pneumonia in welders in an analysis of the sickness rates of the 90,000 workers in the Kaiser Richmond shipyards in America. They found that there was no increased incidence in welders. Collen, in a later article (1947) confirms his earlier findings saying that the annual incidence, death rates and case fatality rates for welders were similar to all other shipyard workers, and that the cases were similar in severity, required the same number of days for treatment and showed no difference in the incidence of complications.

5. *Radiological.*—The presence of pulmonary fibrosis in and around dust aggregations cannot be diagnosed from an examination of single X-ray films; the film of a welder may be indistinguishable from that of a sandblaster with silicosis, or a miner with pneumoconiosis. The small densities forming reticulation or nodulation may be due to a variety of conditions of which nodular fibrosis is only one. However, the X-ray changes in silicosis, asbestosis, and pneumoconiosis are permanent; if they change at all it is in the direction of progression, and this is common. McLaughlin and I, however, recently described two of our original welders whose X-ray appearances have changed for the better (Doig and McLaughlin, 1948). We showed that one man, who exhibited definite X-ray changes of the dust inhalation type in 1934, and who ceased welding on being told of these changes, now has an X-ray picture within normal limits. The pneumoconiosis has resolved, and no one could guess at its previous existence. The other man who had marked changes in 1933, and who continued to work as a welder, became a welding instructor in 1940, with consequent great diminution in his exposure to fume; his X-ray film now shows partial clearing of the abnormal shadows.

Other dusts which are considered on present evidence to be inert include tin, calcium, and barium, but much more knowledge is

required before we can be sure that this is so. Tin is much more radio-opaque than iron, therefore deposits of tin in the lungs cause particularly dense shadows. Pendergrass and Pryde (1948) show an X-ray film of this type, discovered on routine examination, the subject being a man aged 45 years who had bagged tin oxide for fifteen years. There was no disability.

I have said no word about hæmatite miners, although there are a number of reports in the literature about the effects on the lungs of iron-ore dust. There is no doubt that hæmatite miners may get a disabling pneumoconiosis, but this is to be expected for iron ore is a rock with considerable silica content, and so the disease is really a silicosis or at least sidero-silicosis. Some hæmatite miners working in mines with soft ore may develop X-ray changes without incapacity, and in such cases it may be assumed that the siderosis element predominates over the silicosis.

Although I have referred only very briefly to the evidence, I think I have said enough to show that industrial siderosis is one of the benign pneumoconioses and that iron is one of the inert dusts. One of the practical lessons to be learned is that by looking at an X-ray film one cannot diagnose silicosis; one requires in addition to the X-ray evidence a complete industrial and clinical history and the results of a clinical examination. The radiologist must primarily describe what he sees in the film. He does not see pathological changes. He must not assume the presence of fibrosis when X-ray examination shows reticulation or nodulation. If he does so there are liable to be unfortunate repercussions on the patient, on his family, and on industry.

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