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ALUMINIUM THERAPY OF SILICOSIS

It was in 1937 that Denny, a metallurgical engineer, Robson, chief surgeon to the McIntyre Porcupine Gold Mine at Schumacher, Ontario, and Irwin,[1] associate professor in the Department of Medical Research at Toronto, under Sir Frederick Banting, reported that in dusting experiments they could produce typical nodular silicosis in rabbits, but that if the quartz dust was mixed with aluminium dust of particle size less than 5 μ, the proportion of aluminium in the mixture being less than 1%, nodular fibrosis did not occur. In 1939[2] they further showed that the metallic aluminium on being converted into hydrated alumina reduced the toxicity of quartz in tissues by flocculation, partly by absorbing silica from solution, but chiefly by coating the quartz particle with an insoluble and impermeable coating which is gelatinous hydrated alumina. This, on drying, forms the crystalline alpha aluminium monohydrate ($Al_2O_3H_2O$), which stains red with aurine. They also exposed eight rabbits to an atmosphere containing freshly ground finely particulate aluminium powder in a concentration averaging 7,000 particles per ml. 12 hours daily for 14 months. After cessation of the dust exposure some of the animals were observed for a further seven months. Chemical assays of the lungs showed them to contain from 270 to 1,200 mg. of aluminium per 100 g. of dried tissue. During their lifetime these animals gained normally in weight, and from the appearance and texture of their fur and general behaviour there was no evidence of any harmful effect. At necropsy there was no evidence of fibrosis of the lungs, the only abnormality being aggregates of dust cells containing irregularly shaped opaque particles. In repeated experiments in rats Belt and King,[3] however, found that the particles were treated as foreign bodies in the lungs, forming small concretions with fibrous tissue round them. Nevertheless in 1944 Leroy Gardner[4] and his colleagues confirmed with experimental evidence beyond question that aluminium and aluminium hydrate (XH-1010), probably in amorphous state, specifically inhibit fibrous reaction to quartz, and that their administration will prevent progression of silicotic lesions and cause retrogression in immature tissue responses.

Evidence on the effect of aluminium in pneumoconiosis also comes from the recent report of the experimental studies on chronic pulmonary disease in the South Wales coal-mines published by the Medical Research Council.[5] These studies revealed that the most severe reactions in lungs result from quartz, but that the pathogenicity of this substance is reduced by clean coal and is practically abolished by shale. There is little doubt that the production of silicosis depends on the solubility of silica, and it is proved that quartz particles coated with iron oxide[6] or alumina are less soluble than those which are not. King found that the shales depressed the solubility of the quartz by releasing aluminium from their aluminous components to form a protective covering over the quartz particles which could be stained with aurine. It was also demonstrated that mineral dusts containing higher proportions of aluminous components were more inert, even after being in the lungs for a long time; this is well shown by the fact that the shales are probably less dangerous to health than the sandstones. King also explained the pathogenicity of anthracite coal, which *in vitro* depresses markedly the solubility of quartz, by suggesting that the aluminous and siliceous components of steam and bituminous coal must be much more closely bound to the coal substance than is the case with anthracite.

Before applying the method for the prevention of silicosis in man it is essential to show that metallic aluminium is without harmful effects. In 1936 the Medical Research Council,[7] after reviewing the findings in a group of fifty workers exposed for many years to alumina, were unable to find any evidence that the inhalation of alumina dust had caused pulmonary fibrosis, while Hunter, Milton, Perry, and Thompson[8] were unable to find disease in the lung in 92 duralumin propeller grinders. Crombie, Blaisdell, and MacPherson[9] investigated 125 employees engaged in making aluminium powder by a stamping process: they had been on this work for periods ranging from six to twenty-three years, and they were submitted to *x*-ray examination each year. Aluminium powder did not cause lung damage, nor did it favour the development of tuberculosis or any other condition. During the war of 1939–45, however, many papers from Germany described a condition of pulmonary fibrosis with frequent spontaneous pneumothoraces in workers making alumina powder.[10-13] These papers remain an enigma. It must be pointed out, however, that there are many differences in aluminium powders. Denny, Robson, and Irwin's powder is black; it contains 95% metallic aluminium, and its particle size is under 5 μ, accounting for its black colour. Stamped aluminium powder is coated with stearine, while blown aluminium powder is coated with aluminium oxide. It is possible, therefore, that aluminium may not be completely harmless, and its use should be allowed only under the supervision of expert observers.

At the Porcupine Mine Denny, Robson, and Irwin have treated miners by blowing with compressed air metallic aluminium dust from small canisters into the rooms where

1 *Canad. med. Ass. J.*, 1937, **37**, 1.
2 Ibid., 1939, **40**, 213; *Industr. Med.*, 1939, **8**, 133.
3 *J. Path. Bact.*, 1943, **55**, 69.
4 *J. industr. Hyg.*, 1944, **26**, 211.
5 Med. Res. Cncl. Sp. Rep. Ser. No. 250, 1945, London.
6 Kettle, E. H., *J. Path. Bact.*, 1932, **35**, 395.
7 *British Medical Journal*, 1936, **2**, 1273.
8 *Brit. J. industr. Med.*, 1944, **1**, 159.
9 *Canad. med. Ass. J.*, 1944, **50**, 318.
10 Goraleswki, G., *Arch. Gewerbepath. Hyg.*, 1939, **9**, 676; ibid., 1940, **10**, 384; ibid., 1941, **11**, 106; *Dtsch. Tuber.-Bl.*, 1943, **17**, 3.
11 Goralewski, G., and Jager, R., *Arch. Gewerbepath. Hyg.*, 1943, **11**, 102.
12 Jager, R., and Jager, F., ibid., 1941, **11**, 117.
13 Koelsch, F., *Beitr. klin. Tuberk.*, 1943, **97**, 688.

the men change for about 20 minutes before they go down the mine, and after they come up. Their results are not yet published, but miners returning to this country are enthusiastic about this method of preventing silicosis. Crombie, Blaisdell, and MacPherson[8] treated men in the same way but prepared the aluminium from a mill. By this method there is a small explosive risk if the motor is close to the mill, aluminium dust forming an explosive mixture if the concentration is more than 0.02 oz. per cubic foot (0.02 g. per litre). Thirty-four men with diagnosed silicosis were treated by daily inhalations beginning with 5 minutes and gradually increasing to 30 minutes. Some men received 300 treatments, the majority about 200. Out of the 34 cases studied, 19 showed clinical improvement with diminution of the dyspnoea, cough, pain in the chest, and fatigue, though they continued to work as miners; the remaining 15 cases remained stationary. Progress of the disease was assessed by means of respiratory function tests, repeated at three-monthly intervals. Bamberger[14] also treated a group of non-ferrous metal miners with both metallic aluminium and hydrated alumina (XH-1010), and claimed that about one-third in each group showed symptomatic improvement though there was no significant change in exercise tolerance or vital capacity in any case. Aluminium dust cannot be regarded as a cure for silicosis, but these results suggest that it may prevent the development of human silicosis and may possibly arrest the progress of the disease when it is established.

These promising developments have been marred by two recent happenings. Denny and Robson[15] have taken out a patent for this method of treatment in the United States and Canada, and created a company with the McIntyre Porcupine Mines, Ltd., known as McIntyre Research, Ltd., while in Australia and Tasmania laws have been passed to compel employers to install this method of treatment in their change houses. In this country the law does not allow the patenting of therapeutic methods, and British research workers are therefore free to investigate, assess, and develop the method. There is even an obligation to carry on investigations when restrictive influences are at work elsewhere. It is also appropriate to draw attention to the recent report of the Council on Industrial Health of the American Medical Association,[16] which states that the administration of high concentrations of amorphous hydrated alumina unfavourably influences resistance to tuberculosis; and recommends that the general application of aluminium therapy in industry be delayed until adequately and impartially controlled clinical observation demonstrates its effectiveness in preventing or alleviating silicosis in man. It is evident that the real way to prevent silicosis is to prevent siliceous dust, and engineers must be encouraged to attain this end. Their achievements with wet drilling up to the present have been very great, and there seems no reason to doubt that within the next century their efforts will be rewarded with complete success. In the meantime mining must go on, and so must research into all possible methods likely to prevent or arrest silicosis in those engaged in the industry.

[14] *Industr. Med.*, 1945, **47**, 185.
[15] Robson, W. D., *Trans. Canad. Mining Inst.*, 1944, **47**, 172.
[16] *J. Amer. med. Ass.*, 1946, **130**, 1223.

DIET AND INCOME

On another page Dr. E. R. Bransby gives the results of a dietary survey, made in 1941 by the Ministry of Health, of a number of working-class families chosen at random from various parts of the country. This period, it may be remembered, was one of transition from the days of pre-war plenty to wartime austerity and food rationing. By 1941 there had been considerable changes in the economic condition of the country. Food prices had risen by about 25% and wages by about 40% above 1938 levels. The present-day figures are approximately 22% and 50% respectively, though the former figure is really fictitious, as food prices have been kept low by the Ministry of Food subsidies, which, of course, ultimately come out of the taxpayer's pocket. The extra he would pay for food owing to the rise in the cost of living is taken out of his wage or salary. In 1941 the milk-in-schools scheme and the national milk scheme were in operation, but not the vitamin schemes for expectant mothers and babies, and the school meals service was operating only to a very limited extent. The bulk of the bread consumed was made from 75% extraction flour.

The survey confirms much that was already known to the social worker and the general practitioner familiar with the conditions in working-class families. It is not surprising to read that those families with an income sufficient to spend 13s. a week per head on food averaged only one child. Based on the present-day cost of living this is not an excessive sum to provide an adequate level of nutrition without monotony. On the other hand the families with four to five children spent less than 5s. a week per head on food—a totally inadequate amount judged by any standards. The consumption of most foods, and particularly the protective ones, was notably greater among the families with few children than among the families with several children. Both groups ate approximately the same amount of potatoes, margarine, oatmeal, fats, and sugar —all foods which are cheap or rationed. These are the energy-producing foods. For the protective foods the difference in consumption in the two types of family was considerable. The intake of meat, milk, eggs, vegetables, and fruit was much greater among the families with a higher than among those with a lower food expenditure per head. On an average the consumption of these foodstuffs was 450% more in the former group than in the latter. For fruit and vegetables the figure was 1,700% higher.

Judged by the League of Nations standards of minimum requirements, and by costs calculated in 1942 by Magee and George, the intake of all nutrients, except protein, was inadequate among those families spending less than 9s. per head a week on food. Most families with more than two children spent less than this a week. Particularly deficient were the intakes of calcium, vitamin A, and vitamin C. Magee and George, at the request of the Ministry of Health Advisory Committee on Nutrition, compiled figures to show that the standards for an adequate diet, as recommended by the League of Nations Commission, cannot be satisfied if less than 9s. per head is spent on food. This figure is for adults and older children; it