asbestosis does not tend to progress after exposure is stopped or markedly reduced. Advanced asbestosis usually is associated with considerable emphysema and commonly leads to right heart strain and failure.

C. BAUXITE PNEUMOCONIOSIS (SHAVER'S DISEASE)

A new occupational fibrosis of the lungs was recognized for the first time in 1942 by Dr. Cecil G. Shaver,[92] Niagara Peninsula Sanatorium, St. Catherines, Ontario. It is essentially a diffuse interstitial fibrosis and marked associated emphysema, with a complete absence of any nodular fibrosis. It has been found only in workers exposed to the fumes resulting from the fusion of bauxite in the manufacture of artificial aluminum abrasives.

Bauxite is a natural hydrated aluminum oxide (Al_2O_3) containing small amounts of silica and iron. When it is fused by heating at a high temperature in open electric furnaces, a crystalline artificial corundum results. This process gives off dense white fumes containing from 40 to 60 per cent aluminum and 30 to 45 per cent amorphous silica. Particle size ranges of the fume are remarkably small—from 0.5 μ down to .02 μ—and can be visualized only with the electron microscope. Workers with the heaviest exposures have been the overhead crane operators and those who shovel the mix into the furnaces.

In the early stages there are no symptoms or clinical signs. The early x-ray pattern is characterized by indefinite granular or lacelike shadows, particularly in the upper lobes, resembling early tuberculous infiltration. As the pathological changes progress, involvement of the lower lobes occurs. The shadows may remain granular or may assume an irregular nodular pattern. The root shadows usually are enlarged and increased in density. Emphysema usually is present and may be extreme, with large emphysematous blebs. Spontaneous pneumothorax has occurred in numerous cases and resulted in the death of several of these workers. The main symptom of those with advanced disease is shortness of breath. This may be sudden and extreme at the time the pneumothorax occurs. Most cases have developed in a rather short period of exposure: from two to five years.

Approximately 50 cases had been discovered in the Niagara Falls area up to about five years ago. No tuberculosis was found in any of them. Since better fume control measures have been instituted, no new cases have come to light. It is of interest that no cases of the disease were recognized until 1942, even though the fusion process had been used since 1914. The only explanation for this is the tremendous increase in production of the artificial abrasives during the war years.

The precise causative factor in the fume has not been positively identified. Heretofore, aluminum and amorphous silica had been thought to be innocuous. Also, extremely fine particles of any dust, less than 0.5 μ in size, had been thought to have no significance in the development of lung fibrosis. It was believed that

[92] C. G. Shaver and A. R. Riddell, Lung changes associated with the manufacture of alumina abrasives, *J. Ind. Hyg. Toxicol.*, **29**, 145 (1947).

such fine particles were not retained in the lungs. However, Hatch[93] has calculated that alveolar retention of particles below 0.5 μ is as great as of larger particles, and may even be greater below 0.1 μ because of Brownian motion. If this is true, then the fineness of the particles alone is an adequate explanation for the rapidity of development of this disease. Whether the silica particles alone or the aluminum particles alone are responsible, or whether the combination is necessary, has not been determined. German investigators, particularly Goralewski,[94] believed that aluminum alone was the offender and they still speak of the disease as Aluminosis. Opinion in this country, however, favors the silica fume as the toxic agent, probably modified by the aluminum particles. This seems the most logical explanation for the fact that the fibrosis does not assume a nodular character.

Interest in this disease extends well beyond the artificial abrasive industry because of the implications associated with the rapid development of fibrosis due to ultramicroscopic particles, which are harmless when larger. Is it possible that other innocuous materials may be harmful in the fume size ranges? This possibility seems unlikely for iron because of the experience with welding fumes, which contain upwards of 95 per cent iron oxide particles in size ranges below 0.1 μ and in Brownian motion. The evidence is clear, at least for this fume, that no such reaction has occurred, even in welders who had such concentrated exposures that the iron collections in their lungs could be seen by x-ray. Such exposures caused not even a trace of fibrosis in the lungs. This evidence suggests that it is not the submicroscopic particle per se that is the toxic factor in Shaver's disease. More extensive research with the fumes of many materials is needed before this interesting problem can be resolved.

D. DIATOMACEOUS EARTH PNEUMOCONIOSIS (DIATOMITE SILICOSIS; DIATOMITE PNEUMOCONIOSIS)

The first epidemiological study in the diatomite processing industry in California was reported by Legge and Rosencrantz[95] in 1932. Of 118 employees who were x-rayed, 68.5 per cent were said to have shown evidence of pneumoconiosis. No correlations were made between the degree of dust exposure and the clinical findings. There were no other American reports for the next 20 years, but a major study in Italy was reported by Vigliani and Mottura[96] in 1948. For the first time, it was shown by way of x-ray diffraction that crude diatomaceous earth contained no crystalline form of free silica, but that after flux calcining at

[93] T. F. Hatch, J. H. Brown, K. M. Cook, and F. G. Ney, Influence of particle size upon the retention of particulate matter in the human lung, *Am. J. Pub. Health*, **40**, 450 (1950).

[94] G. Goralewski, Aluminum lung, *Arch. Gewerbepathol. Gewerbehyg.*, **11**, 102–116 (1941).

[95] R. T. Legge and E. Rosencrantz, Observations and studies on silicosis by diatomaceous silica, *Am. J. Pub. Health*, **22**, 1055–1060 (1932).

[96] E. E. Vigliani and G. Mottura, Diatomaceous earth silicosis, *Brit. J. Ind. Med.*, **5**, 148–160 (1948).