30 million particles per cubic foot would give 0.10 times 30 million, which equals 3 million (good practice); a dust containing 30 per cent with an average total concentration of 50 million particles per cubic foot would equal 0.3 times 50, or 15 million (unsatisfactory). This formula not applicable to any dust containing less than 5 per cent free silica.

The Division of Occupational Health, U. S. Public Health Service,[38] has suggested that an attempt should be made to have highly siliceous dusts kept at a concentration below 4 million particles per cubic foot since considerable silicosis occurred in the pottery industry even with low dust concentrations.

Dust may also be simply a nuisance. It is considered good practice to control even relatively harmless dust sufficiently to prevent concentrations in excess of 50 million particles per cubic foot (by light-field count) in the workroom air.

c. Particle Size in Relation to Development of Fibrosis. We have already mentioned the fact that large particles settle more rapidly than small ones and that particle size is important as a determining factor in the actual amount of dust in air. Particle size is even more important in determining the chemical activity of the dust and physiological responses to it. Chemical activity is, of course, increased in small particles because of the increased surface area. McCrae[39] found that 70 per cent of the particles in silicotic lungs were less than 1 μ in diameter and that the largest were not greater than 10.5 μ. An upper limit of 10 μ as the maximum size that will produce silicosis has been suggested because larger particles are probably collected by mucus in the upper respiratory tract and moved out of the lungs by ciliary action. The importance of small particles in the production of fibrosis has been well demonstrated by Tebbens, Schulz, and Drinker.[40] These investigators produced liver fibrosis in experimental animals by intravenous injections of suspended silica and found that particles less than 0.6 μ in diameter caused much more fibrosis than larger particles.

Briefly, silicosis is caused by the inhalation of silica particles less than 10 μ in diameter. Recent work suggests that particles of less than 0.6 μ may be of greatest importance in this respect.

d. Individual Predisposition. Detailed discussion of the role of individual predisposition in the etiology of silicosis is hardly necessary. Race and sex seem to play little part, although race may be a factor in the development of tuberculosis just as financial status may be a factor. Certainly it is known that of two persons working in the same dusty exposure one may contract silicosis in a few years while the other may require many years. We have already mentioned the possible influence of differences in nasal filtration as a factor in causation of silicosis. It is likely that there are sound explanations for the many variations in individual susceptibility, but clarification is lacking. Many of these variations undoubtedly are related to differences in adrenal cortical hormone activity.

[38] R. H. Flinn, W. C. Dreessen, T. I. Edwards, E. C. Riley, J. J. Bloomfield, and R. R. Sayers, *U. S. Pub. Health Bull.* No. **244**, 1939.

[39] J. McCrae, *Pub. S. African Inst. Med. Research*, Report No. 3 (1913).

[40] B. D. Tebbens, R. Z. Schulz, and P. Drinker, *J. Ind. Hyg. Toxicol.*, **27**, 199 (1945).

4. Pathological Anatomy and X-ray Findings

a. Benign Pneumoconiosis. All dust other than that containing free silica and asbestos generally is inert and produces little or no fibrosis in the lungs.

X-ray examination. There is simply an accentuation of the normal, branching, treelike shadows cast chiefly by the pulmonary blood vessels. As an occasional variation in this picture there may be superimposed fine reticulations. These reticulations are thought to be caused by thickening of the sheaths of the pulmonary arteries and thickening of the interlobular septa (see Figures 4 and 5).

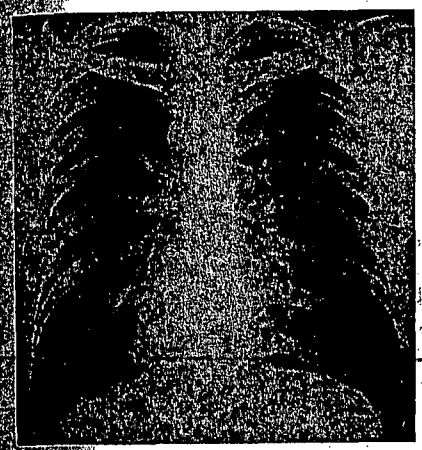

Figure 4. *Normal adult chest.* Note that root shadows and vascular markings are not prominent. Underexposed films may cause exaggeration of the vascular pattern and suspicion of dust retention.

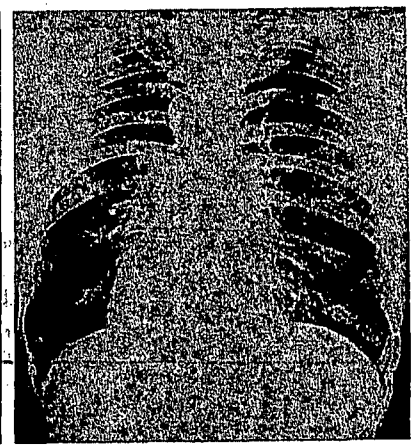

Figure 5. *Increased linear markings.* These usually are due to increased vascularity of the lungs for one reason or another and are not diagnostic of silicosis or other fibrosis. They may, however, be due to early inert dust retention in the lymphatics of the lungs.

Dusts with higher atomic weights, such as iron (56), tin (119), and barium (137), are at times retained in the lungs to such a degree that the perivascular and peribronchiolar collections of the dust actually cast nodular shadows on chest x-ray films. This was first shown by Enzer and Sander[41] in 1938 in the postmortem tissue studies of an electric arc welder, and was subsequently confirmed by animal-experimental studies by Hamlin and Vorwald.[42] Such iron deposits

[41] N. Enzer and O. A. Sander, Chronic lung changes in electric arc welders. *J. Ind. Hyg. Toxicol.*, **20**, 333–350 (May, 1938).

[42] L. E. Hamlin and A. J. Vorwald, Siderosis: a benign pneumoconiosis due to the inhalation of iron dust, *Ind. Med. and Surg.*, **19**, 151–180 (April, 1950).