sharpness of the particles. However, Gardner[27] found that no fibrosis was produced by experimental inhalation of aluminum oxide and diamond dusts which are even harder and sharper than silica. Thus, the action is clearly chemical, but the exact chemicophysiological reaction that takes place is still unknown.

The mixed reaction sometimes produced by dusts of the inert group in which silica is mixed with other minerals emphasizes the possible effect of mixed dusts on the development of silicosis. The probable importance of concomitant exposures has also been indicated by clinical experience. Chapman,[28] MacDonald and his associates,[29] and Kilgore[30] have reported cases of rapidly developing silicosis caused by breathing air with high concentrations of silica and alkali dust. Kettle[31] and McCord[32] failed to demonstrate such action experimentally. McCord, on the basis of extensive investigations of workers in six plants where there was exposure to silica and alkali dusts, found no evidence of an accelerator action by alkalies. In fact, the absence of silicosis in this group suggested an inhibitor action which he believes may have resulted from the marked increase in solubility of silica in the presence of alkali. Peritoneal injection in animals yielded no results to prove either an accelerator or inhibitor action of alkali in the formation of silica nodules. Alkali did, however, cause the silica to spread from the point of injection and increased its primary toxicity.

The absence of silicosis in ganister-brick manufacturing was thought at first due to an inhibiting effect of accompanying dusts; more recently it has been found that the concentration of free silica in the work atmosphere was too low to cause fibrosis. Silicosis as found in coal miners (anthracosilicosis) and in foundry men may be quite different in x-ray appearance and in its course from that observed in quartz grinders.

b. Number of Particles Inhaled in Relation to Production of Fibrosis. It is apparent that the type of dust may play an important part in determining the number of particles in the workroom atmosphere. Not only may some components of admixtures flocculate and thus change the character of a dust, but silica itself, being a hard material with a tendency to form large particles as compared with those formed by softer minerals, may settle out more quickly than other components of a mixed dust.

However, at this point we are somewhat more concerned with the concentrations of dust that are harmful than with the factors that have produced any given concentration in the air. Determinations of free and total silica in the lungs of patients who have died after fibrosis developed and in those without fibrosis have been made by incinerating the lungs and chemically analyzing the ash for its silica content. Although there have been some variations in published data on the

[27] L. U. Gardner, *Am. Rev. Tuberc.*, **20**, 883 (1929).
[28] A. M. Chapman, *J. Am. Med. Assoc.*, **98**, 9 (1935).
[29] G. MacDonald, A. P. Piggot, and F. W. Gilder, *Lancet*, **2**, 836 (1930).
[30] E. S. Kilgore, *J. Am. Med. Assoc.*, **99**, 1414 (1935).
[31] E. H. Kettle, *Proc. Inst. Mining Metallurgy (London), 43rd Sess.*, 1934.
[32] C. P. McCord, *Ind. Med.*, **5**, 17 (1936).

amount of silica in pulmonary tissue necessary to produce silicosis, Sladden,[33] McNally,[34] Badham and Taylor,[35] and Fowweather[36] found fairly comparable amounts. Drinker and Hatch[2] sum up the present knowledge somewhat as follows: As a rough guide, a total silica content as high as 0.2 per cent of dried lung can be considered normal. A content over 1 per cent is definite evidence of dust exposure, and this amount is usually accompanied by fibrosis. There are too much overlapping of data and variation in technique to be sure from published data of the significance of quantities between 0.2 and 1 per cent of ashed lung. This fact is of especial significance, as pointed out by Cummings.[37] Since there is usually no evidence of silicotic reaction in lungs containing less than 1.5 to 2 g. of silica (1 per cent of weight of dried lung), it may be inferred that the contraction of silicosis does not necessarily follow the inhalation of silica. Silicosis occurs only after the inhalation of amounts in excess of a minimum.

In any control program we are of course concerned primarily with the amount of dust in the air that will cause fibrosis. The methods of determining atmospheric dust concentrations are discussed in Chapter VII. Cummings suggested that atmospheric concentrations of silica dust should be considered in terms of two thresholds: the primary threshold, a level at which a healthy man can be employed for his lifetime without harm, about 5 million particles per cubic foot (light-field count); and the secondary threshold, a level at which a healthy man will inevitably develop silicosis, about 100 million particles per cubic foot (light-field count). The National Silicosis Conference summarizes the situation as follows: "There is evidence that for prolonged exposure a concentration of more than 5 million particles per cubic foot, of a highly siliceous dust, is dangerous. Therefore it is now considered good practice to hold concentrations of highly siliceous dust at 5 million particles per cubic foot, or less," as based on light-field counting methods.

Since standards of safe atmospheric dust concentration based on medical findings have been suggested tentatively for only a few industrial dusts, and since considerable study is necessary to form a basis for such standards for other industrial dusts, a tentative arbitrary measure of what is good practice may be used. This should be within the limits of good engineering practice and yet low enough to control the silicosis hazard for most industrial exposures. The following formula is frequently used to express the maximum permissible concentration of silica in the air:

Multiply the percentage of free silica by the total dust particle count per cubic foot (light-field technique). If the result is over 5 million, the concentration may be considered too high. For example: a dust containing 10 per cent free silica with an average total concentration of

[33] A. F. Sladden, *Lancet*, **2**, 123 (1933).
[34] W. D. McNally, *J. Am. Med. Assoc.*, **101**, 584 (1933).
[35] C. Badham and H. B. Taylor, *Med. J. Australia*, **1**, 511 (1933).
[36] F. S. Fowweather, *Chem. Industries*, **53**, 713 (1934).
[37] D. E. Cummings, "The Etiology of Silicosis," in B. E. Kuechle, ed., *Fourth Saranac Laboratory Symposium on Silicosis*. Employers Mutual Liability Insurance, Wausau, Wis., 1939.