the alveolar air is the same as that in the room air, with due correction for temperature and humidity. With the more soluble vapors, alveolar air concentration, at any point during accumulation, may be used to estimate the average room-air concentration, given the length of exposure, the saturation curve for the particular vapor, and its coefficient of distribution. An interesting variation, of much practical usefulness, is met in the case of radon: its concentration in the alveolar air may be used as an indication of radium storage or deposition, providing sufficient time has elapsed for relatively complete desaturation (8 hours or more after the end of an inhalation exposure).

The physical laws governing the rate of gas exchange between the blood and the respired atmosphere are the basis of a proposed method[13] for measuring pulmonary functional capacity in terms of lung ventilation, gas transfer by diffusion across the alveolar wall, and pulmonary blood flow. The authors suggest possibilities of determining the nature and magnitude of respiratory impairment.

D. DUSTS AND FUMES

1. *The Absorption of Particulate Matter*

Dusts, fumes, and smokes represent solid particlulate matter differing in quality, form, and particle size. The process of their accumulation, distribution, and elimination is much more complicated than the same process with gases. Brown[14] showed that with a number of dusts having a particle size range of 0.25 to 6.0 μ, approximately 55 per cent of the inhaled dust was retained by men. The extremes of retention reported, however, ranged from 15 to 95 per cent. Retention was found to be inversely proportional to the rate of respiration and minute ventilation for rates below 20 inhalations per minute, with no apparent change above 20. Retention was directly proportional to the size of particles or their aggregates, to density, and to wettability. Of this retained dust, a part is retained in the nasal filter and part by contact with wet surfaces in the respiratory passages, while some reaches the respiratory tissues. The upper respiratory passages are subject to brief periods of low velocity, or static air, between alternately directed flows of rather high velocity air. Because of sharp changes in direction, some dust, especially the larger particles, 5 to 20 μ in diameter, is impinged and arrested in these passages by contact with moist surfaces. In the alveoli the air must necessarily be static or moving slowly during respiratory movements, so that ample opportunity is presented for settling and for contact with the moist alveolar walls. If particles are caught before they enter the alveoli, they are continually swept toward the mouth by the ciliated epithelium,[8] which extends from the lower pharynx to the respiratory bronchioles. These ciliated cells are abundant in the trachea, and even more abundant in the large bronchioles, but are sparse in the respiratory bronchioles and absent in the alveoli. The motions of the cilia are wavelike and their efficiency

[13] T. F. Hatch and K. M. Cook, *A.M.A. Arch. Ind. Health*, **11**, 142 (1955).
[14] C. E. Brown, *J. Ind. Hyg. Toxicol.*, **13**, 293 (1931).

in propelling mucous and foreign material toward the oral cavity is of a high order. The individual cilium moves toward its goal with whiplike motion, then slowly resumes its former position. It is evident, then, that dust particles trapped anywhere short of the alveolar ducts are subject to removal from the lungs through ciliary action. Upon arrival at the oral cavity they are expectorated or swallowed. Dust particles (silica) reaching and remaining in the alveoli are believed to be largely limited to the sizes ranging between 3 and 0.1 μ,[15] 3 being the largest to arrive at the alveoli in significant quantities. The majority of particles below 0.1 are thought to be too small to be trapped in the alveoli and are exhaled much the same as is an undissolved gas. The particles caught in the alveoli may be absorbed through the alveolar wall, or they may be engulfed in phagocyte cells and be carried into the blood capillaries, or enter the lymphatics and be concentrated at the tracheobronchial lymph nodes. Particles of silica 1 μ and under are the most active and dangerous, not only in their ability to remain suspended in the air, but especially in their activity in tissue.[16]

2. *Action of Suspended Particulate Matter*

The toxic dusts may dissolve and enter the circulation by absorption from the respiratory tract, or they may be absorbed after being swallowed. The action of these is discussed under the material involved. "Pneumoconiosis"-producing dusts may exert their effects after lodging in the alveoli. Other particulate matter may cause allergic reactions or, in the case of disease germs, infections. The possibility of the sorption of gases and vapors by particulate matter should not be overlooked as a source of significant exposure of the respiratory tract to these gases and vapors although they may not be present in significant concentrations in the *gaseous* environment. Also, it has been demonstrated that the normal locus of action of water-soluble gases may be transferred from the upper to the lower respiratory tract through sorption on particles, thereby increasing their physiological effect. Conversely, when gases less soluble in water, such as nitrogen oxides, phosgene, and ozone, are mixed with aerosols, their effects may be lessened because of sorption. For a complete discussion of the role of dusts and the evaluation of disability arising therefrom see Chapter XII.

E. MISTS

Mists may be inhaled to reach all parts of the lungs and from there may be absorbed. Of a slightly volatile material, concentrations far above those possible from partial pressures of the gaseous phase at room temperature may be encountered. For that reason a solvent, the vapors of which may be considered harmless at ordinary temperatures, can be very dangerous as a mist.

[15] L. U. Gardner, *Ind. Med.*, **9**, 45 (1940).
[16] H. D. Tebbins, R. Z. Schulz, and P. Drinker, *J. Ind. Hyg. Toxicol.*, **27**, 199 (1945).
[17] C. W. LaBelle, J. E. Long, and E. E. Christofano, *A.M.A. Arch. Ind. Health*, **11**, 297 (1955).