Valle[1] has pointed out that more than 50 per cent of industrial dusts are greater than 1 μ in size while in atmospheric dust the median size is approximately 0.5 μ. Bloomfield[3] found that only 20 per cent of the particles found in air in fifty industries were smaller than 1 μ and that the median size was 1.3 μ; he suggested that most of the particles of smaller size found in industrial atmospheres were those normally present in air. In certain industries, however, such as pneumatic rock drilling, large numbers of particles well below 1 μ in size are produced.[4]

II. Classification of Dust Based on Its Effect in the Body

The industrial hygienist is interested in dust because of its effect on the human body. Therefore, a limited classification of dust from this viewpoint may form a basis for relating the chemical composition of dust to the anatomical and physiological reactions that occur.

Pneumoconiosis is defined as: *Any dust particles retained in the lymphatic depots of the lungs.* The term generically carries no implication of fibrosis or other reaction and no implication of disturbed function of the lungs. Nor is it specific with relation to the type of dust.

If we accept this generic definition of "Pneumonokoniosis," which Zenker coined in 1866 (the term was shortened by the International Labor Office in 1932), then all humans have some degree of pneumoconiosis. In city dwellers it is the anthracotic pigmentation from air pollution and in farmers the dust from the field and from the grains. The only reason such deposits are not generally thought of as pneumoconioses is that they usually are not visualized by x-ray. Even where the soil is fairly high in free silica, insufficient silicotic nodulation results to be seen by x-ray. Also, most dusts in the general atmosphere have a low grade of radiopacity so that such deposits in the lungs do not cast direct shadows. Therefore, our chest roentgenograms are read as showing "healthy lungs," even though we know that we all have some degree of retained dust or pneumoconiosis.

There are those who, for medical-legal reasons, would limit the meaning of "pneumoconiosis" to the fibroses resulting from industrial dust exposure. They argue that ordinary dust retention should not be dignified with such a word. In addition, a number of state compensation laws list "pneumoconiosis" as a compensable occupational disease. However, there are others who feel that such a strict limitation of meaning of the term is artificial and that it disregards the occupational dust deposits which are benign and do not cause fibrosis (the so-called "benign pneumoconioses"). To the authors it seems more sound scientifically to think in terms of the generic definition and to consider that all humans have pneumoconiosis. When a doctor says that so-and-so has pneumoconiosis, the reply ought to be: "We all have. Please specify. Is it occupational? What type of dust?" Certainly the word has no place in occupational disease laws where the compensable diseases should be carefully and specifically defined.

[3] J. J. Bloomfield, *U. S. Pub. Health Repts.*, **48**, 961 (1933).
[4] T. Hatch and C. L. Pool, *J. Ind. Hyg.*, **16**, 177 (1934).

A useful classification of the pneumoconioses, based on the effect of dust in the lungs, is as follows:

A. *Inert Dusts* ("benign pneumoconioses"). Little or no fibrous tissue reaction; dormant deposits in pulmonary lymphatics and root lymph nodes, which are visualized by x-ray when the material is radiopaque. They do not appear to have a predisposing effect on tuberculosis or other infection and do not cause impaired lung function.

1. Carbon (smoke, soot): Most anthracosis due to coal deposits also benign, but excessive deposition over many years may lead to disabling emphysema (see below).

2. Calcium (cement, marble, gypsum): Generally not visualized by x-ray. Cases called "calcicosis" in past probably were endogenous calcification due to fungous diseases such as histoplasmosis.

3. Iron (confined welding, grinding, steel cutting, and burning): Radiopaque deposits visualized by x-ray as discrete nodulation. "Siderosis" is a perfectly proper term for such iron pigmentation, but there should be no implication of fibrosis in the term.

4. Artificial abrasives (carborundum, emery, aluminum oxide): Have replaced sandstone in grinding wheels.

5. Aluminum (or hydrated aluminum): Used in some areas in prevention and treatment of silicosis.

6. Barium and tin (radiopaque deposits): Baritosis and stannosis.

B. *Proliferative Dusts* ("fibrotic pneumoconioses").

1. Silica (crystalline free silica, SiO_2, as quartz, flint, etc.): Typical fibrous nodules spread throughout both lungs and visualized by x-ray. Usually normal alveolar walls. Simple silicosis. (*a*) Most silicosis today modified by other components in dust, such as Ca, Al, Fe, and C. (*b*) No disability unless conglomerate or complicated with tuberculosis or advanced emphysema. (*c*) Relationship of silicosis and tuberculosis: Primarily reactivation and spread of pre-existing tuberculous foci. Combined lesions frequently result in chronic massive progressive fibrosis. (*d*) Relationship with heart disease: Right heart strain (cor pulmonale) only with advanced stages of silicosis, silicotuberculosis, and emphysema.

2. Asbestos (hydrated magnesium silicate): Mechanical plugging of bronchioles by long fibers and peribronchiolar fibrosis. Fibrosis may result from breakdown of asbestos bodies. Asbestosis.

3. Other silicates? (talc, kaolin, soapstone, mica, feldspar): Reported fibrosis with some silicates may be due to quartz impurities.

4. Bauxite processing (Shaver's disease): Diffuse fibrosis with marked emphysema and frequent spontaneous pneumothorax. Due to silica fume modified by aluminum fume?

5. Diatomaceous earth: Amorphous and only slightly reactive before calcining; then crystobalite formed, causing diffuse fibrosis.

6. Coal dust (coal workers' pneumoconiosis): Focal emphysema and minimal