IN DISCUSSING hazardous particles, the distinction between dust, fumes, smoke and mist is frequently overlooked. Drinker and Hatch[4] state that dust is formed by reducing earthy materials to small-sized portions, sub-microscopic to the visible, the composition of the particles being the same as that of the parent material. Common examples are the mineral dusts derived from the disintegration of rock and the organic dusts like wheat and flour.

Fumes are formed by processes like combustion, sublimation and condensation. The particle size is generally below 1 micron.

Smoke is generally of organic origin and is characterized by a particle size below 0.5 microns.

Mists or fogs are formed by the condensation of water vapor upon suitable nuclei. The particle or droplet size varies widely, depending on the condition prevailing.

The significance of these distinctions becomes apparent when evaluating the exposure in an individual manifesting signs or symptoms of pneumoconiosis. Since men working in dusty atmospheres are frequently exposed to a combination of these factors, the actual cause of fibrosis may be obscured, a fact which has considerable importance in cases involving litigation.

Like "rheumatism," the term "pneumoconiosis" covers a variety of conditions. It has been defined as a "chronic pulmonary fibrosis due to the inhalation of irritating dusts which produce a proliferative reaction" (Johnson[5]), or a condition due to "the effects upon the lungs of the inhalation of excessive quantities of dust, manifested by structural changes in the lung tissue and entirely distinct from the action of poisonous dust such as lead or mercury, in which case the lungs act merely as the point of entrance into the body without definite local influence" (Pancoast[6]). Perhaps the simplest way of stating it would be to say that the term refers to a condition of the lungs resulting from the prolonged inhalation of dust whether harmful or inert.

Pneumoconiosis includes such specific diseases as anthracosis, asbestosis, siderosis, silicosis, etc.

IN RECENT years few diseases have received more attention and publicity than those due to dust. The work of the United States Bureau of Mines in the Tri-State Lead and Zinc Mining districts (1924-1927) and the Metropolitan Life Insurance Company[7] directed attention to other industries where disabling pulmonary diseases were known to exist. Other investigations were made by the U. S. Public Health Service, and in 1933 Gardner and Cummings, of the Saranac Laboratory, began extensive studies in the iron ranges of Northern Michigan and Wisconsin. Since that time, many industries, here and abroad, have established clinics and laboratories for further research and control of the hazard.

The pathology of these diseases was definitely established by Dr. Leroy U. Gardner,[8] Director of the Saranac Laboratory, in 1934. He demonstrated the effects of various dusts on the lungs of laboratory animals and studied the part played by tuberculosis in the progress of the disease. The etiology, physical signs and symptoms, x-ray and laboratory findings were fully described and many other details of a technical nature determined. During this period also, concentration codes, representing "safe" limits of air borne dust, were recommended. These vary in different locations but, generally speaking, the following table, set up by the Committee on "Prevention of Silicosis Through Medical Control" of the National Silicosis Conference, offers a fair standard.

TABLE II.
PERMISSIBLE DUST CONCENTRATIONS IN VARIOUS INDUSTRIES

Industry	Percentage Silica in the Dust	Permissible Dust Concentration millions per Cubic Foot
South Africa*	80	4½
Ontario Gold Mines*	about 35 (in the rock)	8½
Australia Sandstone*	90 (in the rock)	6
Barre Granite*	31 to 33	00 to 20
Pennsylvania Anthracite Coal Mines**	35	5 to 10
	13	10 to 15
	5	50
Broken Hill, Australia*	10 to 17	14

*Based upon engineering practice.
**Based upon clinical studies.

Of the dusts studied up to the present time, only silica and asbestos produce definite pulmonary fibrosis. All the other types of pneumoconiosis exhibit the same general kind of tissue change with a similar pattern of shadows on the roentgenogram. Gardner states that the pattern in this instance consists of a mere accentuation of the normal branching, tree-like shadows cast chiefly by the pulmonary blood vessels. It represents a simple benign type of linear fibrosis and is difficult to distinguish from the mild accentuation of linear markings sometimes seen in x-rays of individuals with no known history of dust exposure.

Various terms are used to indicate special types of pneumoconiosis. For instance, "anthracosis" designates a condition of the lungs found among coal miners due to the inhalation of coal dust. "Siderosis" describes the tissue reaction occurring in some iron ore miners, and such terms as "byssinosis," and "tobacosis" refer to the pulmonary changes resulting from exposure to dust from cotton and tobacco. While such distinctions do not add much to our knowledge of diseases due to dust, nevertheless it is desirable to have a general conception of the important physical and roentgenological features of each for the sake of diagnosis.

In this connection it is advisable to mention the status of the silicates. The term "silicatosis" has been used from time to time to describe changes observed in the chest x-ray of persons exposed to dust from such substances as talc, soap stone, mica, feldspar, garnet, etc.; but the exact role played by these silicates in the production of pulmonary change has not yet been definitely established. They constitute a group of numerous minerals which find widespread use in industry, but with the exception of asbestos, their capacity to produce fibrosis has not been demonstrated. Petrologists warn against the potential hazard from silicates but experiments so far have produced no evidence of connective tissue proliferation as a result of their use. On the other hand, it should not be assumed that lack of such evidence indicates absolute inertness. The changes observed in the chest x-rays of persons exposed to silicate dust have been theoretically explained as a mild silicosis arising from silica left after the body fluids have leached the bases out of the silicotic molecule. A representative group of 24 silicates used in industry, listed by Gardner, appears in Lanza's book, "Silicosis and Asbestosis." The inert dusts, which include most of the silicates, are relatively unimportant because of their non-disabling character. A few of these materials are limestone, marble, talc, chalk, calcined magnesium for insulation, furnace linings, carbon dust, iron dust, tobacco dust, cement, cotton, molds, fungi, etc.

Anthraco-silicosis results from excessive exposure to coal dust which contains amounts of free silica. Since carbon is one of the inert dusts, it seems reasonable