While the type of pulmonary lesion depends on the particular dust inhaled, the underlying pathological principle is the same in all forms of the disease, namely, an ultimate fibrosis and replacement of the elastic lung tissue by a hard unyielding fibrous tissue. All occupations that expose men to large amounts of dust may give rise to pneumoconiosis but not necessarily to silicosis. It is now generally agreed that in order to produce the latter, (silicosis) inhaled silica dust must reach the lungs in a chemically uncombined condition, in very fine particles not more than ten microns (10/25,000 inch) in diameter and in sufficient amount and over a sufficiently long period of time. In relatively recent literature it appears that the proponents of the idea that only "free silica" is harmful are beginning to have some doubts and to have the thought that possibly some silicates may under some conditions be harmful. Generally speaking, the necessary length of exposure to produce silicosis is in direct proportion to the size of the dust particles and inverse proportion to the dust concentration and the amount of free silica in the dust. Silicosis is characterized anatomically by a generalized fibrosis of the lungs and clinically by shortness of breath, decreased chest expansion and a progressively lessened capacity for work which is out of proportion to the objective physical findings. The effect of exposure to dust such as silica is cumulative, the rapidity of development of the disease depending upon the time and amount of inhalation. Silicosis may develop to the point of causing symptoms only after several years exposure to silicious dust, but it may be progressive in some cases even after exposure has ceased and may cause symptoms or become disabling long after the workman has left the environment that caused the condition. Some may improve or at least remain stationary in the absence of infection. Watkins-Pitchford (J. Ind. Hyg. 9, 109 - 1927) tells of Welsh miners who passed the physical examination for enlistment in the British Army, fought through the World War, then came back to England, and died of silicosis. Unfortunately, it is not stated whether these persons died from silicosis complicated with tuberculosis. Britton and Head (J. Am. Med. Assoc. 96, 1928 - 1931) give more detailed descriptions of similar latent cases in the United States.

Silicosis in General - It is generally agreed that silicosis may be defined as

> "A chronic disease due to the breathing of air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and miliary nodules in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some of all of which may be present), and by characteristic roentgenological findings."

In other words, silicosis is a disease of the lungs in which the normal lung tissue is replaced by scar tissue due to breathing air containing silica dust. Unfortunately there is no known cure for silicosis - but SILICOSIS and nearly all other forms of dust diseases CAN BE PREVENTED.

Recognizing the wide interest and even hysteria among apprehensive employers against whom claims had been filed by alarmed workers, and the necessity of thoughtful consideration of the silicosis problem, the Secretary of Labor on April 14, 1936 called the First National Silicosis Conference attended by more than 300 persons representing workers, employers, State and Federal Agencies, Insurance companies, and other interested groups. Four Committees were organized to study specific phases of the silicosis problem; and assemble the essential facts about silicosis in a series of reports; also to present specific suggestions for silicosis prevention and straighten out other difficulties that silicosis has created. Reports made by these Committees were formally adopted at a second conference held February 2nd and 3rd 1937 in Washington, from which we quote: