

The authors do not rule out the possible pathogenicity of certain silicates, pointing out that in earlier reports they coined the concept of silicogenous dust as a collective term for the etiological factor in silicosis. They believe that any dust which contains free silicic acid, either in the form of quartz or of other modifications of silicic acid or silicic acid bound in silicates, is likely to cause silicosis.

As regards the size of grains in pathogenic dusts the authors say that it has been generally accepted that large-grained dusts are less dangerous than fine-grained dusts. Grains which exceed a certain size are not inhaled but are prevented from entering the deeper air passages by physiologic defense mechanisms in nose, pharynx and trachea. Most investigators are of the opinion that particles measuring less than 10 microns are harmful.

Describing their method of examining lung tissues, the authors say that ashing of lung tissue is not advisable when the grain size is to be determined, since incineration causes sintering or baking together of particles. The dust material was therefore isolated from the lung tissue by dissolving out fat and by wet oxidation of the insoluble tissue portions. After giving detailed descriptions of the analytic methods employed, the authors evaluate the total ash content of the lung, the chemical composition of isolated samples and the distribution of the various sizes of grains in the dust. The sizing of the isolated dust samples was done in the small elutriation apparatus of Gessner and Bühler. The dust grains in the lungs from silicotic patients were mostly in the range from 0.9 to 1 micron; grains larger or smaller were present in lesser quantities. The largest grains had a diameter of 20 microns. Those ranging in size from 10 to 20 microns were present in only small percentages of the total.

The authors also compare the grain size composition of the dusts found in the lungs with those of the dusts resulting from such operations as the drilling of granite (either wet or dry) or from the crushing of sandstone. From the results of these comparisons they deduce pointers to be followed in ascertaining dust exposure and in combating dusts. In ascertaining dust exposure not only the total concentration of dust must be determined but the particle size distribution must be ascertained. Further, the chemical and mineralogical composition, particularly the content in free silicon dioxide, should be determined. They show what factors must be considered when counting methods are employed to determine dust densities.

In combating the dust hazard, the small grain sizes of dust must be eliminated. With this factor in mind the authors discuss ventilating devices, the use of water for settling of dusts and air washing.

ROENTGENOLOGICAL EVOLUTION OF ASYMPTOMATIC SILICOSIS. G. LISCHI, *Ann. radiol. diag.* **22:27** (no. 1) 1950.

Lischi studied two silicotic patients in whom roentgenography revealed advanced lesions, although clinical symptoms were lacking. His explanation of this phenomenon is that silicosis, in spite of invading wide areas of the pulmonary parenchyma, may present a mild form which does not impair pulmonary functions. Thus a fair working capacity may be the result of the fact that some areas of the pulmonary parenchyma are not involved in the fibrosis and are not subjected to other morbid influences which may alter the blood supply and the metabolism of the organ, thus creating conditions favoring emphysema, which is responsible for progressive respiratory insufficiency. Lischi believes that the fibrotic localization produced by the inhaled silica does not cause an acute stage of a definite disease but establishes a predisposition toward morbid processes which may aggravate the silicosis.

METAL DUST PNEUMONITIS. S. G. SJÖBERG, *Nord. Med.* **43:117** (Jan. 20) 1950.

Sjöberg is attached to a copper works in Sweden, the only industrial concern employing vanadium in the form of its acid (V_2O_5) in that country. Of late years he has observed several cases of illness due to inhalation of dust in this works, and in 1947 he observed as many as five cases of pneumonia in the staff of only 36, some of whom were only casual employees engaged on building operations. There were also two employees suffering from chronic laryngo-tracheitis followed by bronchopneumonia after being exposed to vanadium. All the five patients with pneumonia recovered. Sjöberg has also studied the effect of vanadium dust on experimental animals and has succeeded in demonstrating extensive changes in their lungs in which particles of vanadium and leukocytic infiltration could be detected. He notes that certain diseases due to metal dust are not yet included in the Swedish Industrial Diseases Act.