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United States Steel Corporation

TRANSACTIONS

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A high power view of the silica nodule showed the concentric structure, with an a-cellular, hyaline center, and a cellular periphery indicating progression. An ash pattern superimposed showed that there was considerable variation with distribution of silica, some in the center of the nodule, some in the periphery; most, however, was in the periphery, not the center. Emphysema was also noted around the nodule.

Another slide demonstrated that if the silica is associated with other dusts, the nodule is modified. Instead of being round it is stellate with processes projecting out. This is a microscopic, not macroscopic, change. It is seen when silica is combined with silicates or coal dust and the like.

Of the common forms of free crystalline silica, i.e., quartz, crystobalite and tridomite, pathogenicity occurs increasingly in that order, with tridomite the worst. Diatomaceous earth is a silica, but it is amorphous, not crystalline. It produces a cellular reaction with alveolar wall thickening and no collagen formation. In this it is like the quartz-iron combination. It may produce considerable alveolar distortion, but without scar formation. However, if the diatomaceous earth is calcined, some crystalline silica is formed and this then can produce large plaques with serious collagen formation.

Asbestos produces a diffuse pulmonary fibrosis with some scarring. The thickening of the alveolar walls is especially noted at the bases of the lungs. There is often an associated severe obliterative fibrous pleuritis. The slide also showed the presence of "asbestos bodies," elongated, straight or curved, club shaped structures. The severe fibrinous pleurisy, scarred alveolar walls and emphysema mean that an asbestosis usually dies of cor pulmonale.

A slide of talc pneumoconiosis showed thickened alveolar walls, in this instance with desquamation into the alveolar spaces and secondary infection.

A study, conducted under Iron and Steel Institute auspices by Dr. Gross, has demonstrated the mobility of dust deposits in the lungs. The first slide showed a young silicotic nodule, a reaction to quartz dust, with no collagen as yet. A superimposed ashed specimen showed the silica throughout the nodule but the air spaces free of it. If ANTU is then given to produce pulmonary edema and pleural effusion, at the level of an LD 50 dose, the section then shows outpouring of edema fluid. When the ash pattern is superimposed, it shows that the dust has been mobilized out of the nodule and is now dispersed through the edema fluid. Reabsorption would then produce further damage and scarring, the progression of silicosis. (This phenomenon also explains why expectoration of coal or iron dust can occur for many years after cessation of exposure.)