hospital admission film in 1948. There were no respiratory symptoms. Vital capacity was 85 per cent of normal. A history of being exposed to dust and fumes from a detinning furnace for a period of eighteen years was obtained, this exposure having terminated in 1930.

Permission for postmortem examination was limited to thorax and abdomen. The microscopic appearance of the lungs was that of moderate anthracosis. There was no necrosis, nor nodular fibrosis, of parenchyma or lymphoid tissue. On a roentgenogram of a thin slice of lung there were dense perivascular, peribronchial and subpleural shadows. Microincinerated sections examined under polarized light disclosed compact accumulations of bright yellowish brown particles at these sites. The particles were soluble in hydrochloric acid in the presence of powdered zinc and gave a positive spot test for tin. Spectrographic analysis disclosed abnormally large amounts of tin in the lungs. By roentgen ray diffraction analysis of dried lung tissue powder, a pattern identical with that of stannic oxide (SnO_2) was obtained. The lungs contained 1.46 per cent stannic oxide dry weight, and the ash was composed of 13.4 per cent stannic oxide. The silica content of lungs and lymph nodes was normal.

The authors arrive at the following conclusions: 1. Stannic oxide when inhaled for long periods is productive of benign, symptomless pneumonoconiosis with no significant pulmonary fibrosis. 2. Stannic oxide is radiopaque. Particles accumulated in the regions of perivascular, peribronchial and subpleural lymphatics are productive of sharp shadows on the chest roentgenogram. These shadows are not cast by foci of fibrous tissue. 3. Tin deposited in the lungs in this form is nontoxic systemically. 4. Analytic and microincineration studies are necessary to differentiate this condition from simple anthracosis.

THE ROENTGENOLOGIC ASPECTS OF ANTHRACOSILICOSIS. W. J. CORCORAN, Pennsylvania M. J. **53**:505 (May) 1950.

According to Corcoran, the term "anthracosilicosis" is applied to a form of pneumonoconiosis commonly called miner's asthma. It is a chronic disease due to breathing air containing dust generated in the various processes involved in the mining and preparation of anthracite coal. It is characterized anatomically by generalized fibrotic changes throughout both lungs with the presence of excessive amounts of carbonaceous and siliceous material, usually by compensating emphysema and often by cardiac changes in the later stages of the disease.

The first stage consists of a bilateral peribronchial and perivascular fibrosis. A diagnosis cannot be made in the first stage. The second stage consists of nodule formation, and this gives the most characteristic appearance of the disease. The nodules are uniformly distributed throughout each lung. When the nodules begin to coalesce and there are conglomerate areas in the lungs, the third stage has been reached. The author believes that infection plays a part in conglomeration and is the major cause of disability. It has been Corcoran's experience over 25 years that when a person has been exposed to a silica hazard, from five to seven years elapse before any distinctive changes can be seen on the roentgenogram.

While changes are going on in the lung there are changes coincidentally in the mechanics of the chest. The accompanying emphysema gives a darker appearance to the lungs, the interspaces are wider, the costophrenic angles are deeper, and because the chest is approaching a state of fixed inspiration, the heart shadow is smaller than average in the majority of cases.

Carcinoma of the lung is no more common in patients with anthracosilicosis than in people who are not afflicted with this disease; likewise tuberculosis is not more common in these patients before the age of 50, but after 50 years of age patients with anthracosilicosis have a higher incidence of tuberculosis than has the general population.