about 5μ in thickness was pressed against a film, and an image of the section was recorded on the film by soft x-rays filtered through 9μ of aluminum. The dust particles were in strong contrast with the lung tissue, and their structure agreed with that of the particles of the preparation used.

3. Microincineration. This technique was applied on some sections as a complement to the historadiographic procedure. The sections were incinerated at 500 C. and examined in the dark field microscope. As all organic substance was burnt away, the dust particles showed up as bright spots.

4. Spectrographic analysis. Organs from nonexposed controls were found to contain no demonstrable vanadium.

5. Testing of the elasticity of the lungs of the long-exposed animals by successive inflation and registration of the pressure in the respiratory passages according to Friberg [19] (cf. the original work by Sjöberg [12d]), which is similar to the technique employed by Van Allen and Wu.[20]

In the animals exposed to high concentrations of dust for a short period (group 1) these examinations showed the following abnormalities: conjunctivitis; acute laryngotracheitis, in some cases of membranous type; acute bronchopneumonic patches containing particles of vanadium pentoxide dust, in most cases few and not of the smallest size; fatty degeneration of the liver, in some cases more appreciable than in the controls; increased, varying content of vanadium in most examined organs; enteritis in some cases.

With another group of animals exposed as those of group 1, the thymol turbidity value was slightly increased.

The animals which were allowed to recover (group 2) showed slight chronic tracheitis, atelectatic (apparently postpneumonic) areas of the lungs and areas with dilated alveoli. In one animal emphysema was noted. No dust was found in the lungs of some of the animals, although the vanadium content of most of the examined organs, including the lungs, was high.

The animals which had been exposed to mostly low concentrations of dust for a long period (group 3) showed the following abnormalities: chronic inflammatory changes in the nasal and the tracheal mucosa; emphysema of the lungs and bronchopneumonic patches, in most cases containing a few particles of vanadium pentoxide dust; probably reduced elasticity of the lung; small round cell infiltrations of the liver (most probably of infectious origin); pyelonephritic changes in some cases; varying increased content of vanadium in the examined organs with the exception of the intestine, which contained no demonstrable vanadium.

To test the ability of vanadium pentoxide dust to produce fibrosis, the substance was injected intraperitoneally into a series of guinea pigs, according to the method of Sayers, Miller and Yant [21] (1935). No delayed peritoneal reactions were observed, which indicates that the dust has in itself no power to produce fibrosis.

From these animal experiments the following main conclusion may be drawn:

The fact that the particles were found in the affected areas of the lungs indicates that the vanadium pentoxide dust is the cause of the pathologic changes. The

19. Friberg, L.: Health Hazards in the Manufacture of Alkaline Accumulators with Special Reference to Chronic Cadmium Poisoning, Acta med. scandinav. **138**: supp. 240, 1950.

20. Van Allen, C. M., and Wu, C.: Increased Elastic Tension of the Lung in Experimental Pneumonia, J. Clin. Invest. **11**:589, 1932.

21. Sayers, R. R.; Miller, J. W., and Yant, W. P.: Response of the Peritoneal Tissue to Dust Introduced as Foreign Bodies, Am. J. Pub. Health **25**:452, 1935.