

fine granular stippling seen roentgenologically. A previously rapid erythrocyte sedimentation rate became normal. Treatment has been withheld for four weeks, and shortness of breath and cyanosis have increased somewhat, the roentgenologic stippling has returned perceptibly, and sedimentation of erythrocytes again is more rapid but not to the pretreatment levels. Further hormone therapy has not been given, but she is maintaining considerable symptomatic and clinical improvement over the pretreatment status and feels satisfied that she was benefited by the treatment. She does most of her housework and does not require oxygen.

CASE 5.—D. T., a woman aged 22, did the tube filling in shop B from 1945 to 1947.² First symptoms of marked dyspnea, cough, fever and loss of weight began in April 1949 within 24 hours after biopsy of a beryllium ulcer of a finger. The patient was completely bedridden for a year. In April 1950 she had an attack of acute lobar pneumonia and almost died. Antibiotics and oxygen were given. She slowly recovered from the pneumonia and has had an almost complete remission of her berylliosis. Roentgenologically, the fine granular stippling has been markedly diminished. The cyanosis has disappeared, and she has gained 15 lb. (7 Kg.) in weight. Recent report indicates that the remission has been maintained since last May (nine months) and that she is gainfully employed on a sedentary job.

We have, then, five cases with entirely similar clinical and roentgenological findings and a common exposure to a phosphor dust containing beryllium. In all these cases there was evidence of dilatation of the heart due to pulmonary disease (cor pulmonale). All have been variously misdiagnosed as cases of miliary tuberculosis, sarcoidosis, silicosis, fungous infection and metastatic carcinoma. It seems reasonable to assume, now that there is postmortem proof for one of the cases, that all the patients have the chronic granulomatosis which has been ascribed to inhalation of beryllium dust. Although the two owners of shop A whose lungs still are clear probably have had more than 100 times the intake of those affected, the incidence of the disease from this shop is the highest so far recorded. With not over 50 persons having had any material exposure since 1938, the incidence is 8 per cent. This shop, by the way, no longer mixes the phosphor or coats its own tubing, all tubing now being bought coated from large manufacturers. Also, the beryllium in the coatings has been replaced by a nontoxic substitute, so that further cases from new exposures should not occur.

The question whether tube bending and sign making alone can give sufficient exposure to cause the disease cannot be answered conclusively. I have x-rayed the chests of over 50 such workers who had not had the heavier exposures resulting from phosphor manufacture and tube coating and have not found any cases that would suggest berylliosis. All bona fide cases which have been discovered so far appear to have been those of persons who underwent the heavier exposures, with the exception of one reported by Dr. Harriet Hardy. It seems safe to reassure tube benders working with beryllium-coated tubing that their chances of having the disease are remote.

Regarding the hormone-treated patients, it should be noted that M. C. (case 4) who had the most satisfactory improvement had the most recently acquired lesion. R. S. and R. K. (cases 1 and 3) had their first exposures in 1939 and had as a result considerable irreversible fibrosis and emphysema, which M. C. (case 4) had not. D. T. (case 5) had the most recent exposure of all, and her remission, which apparently was spontaneous or "triggered" by the attack of pneumonia, has been almost complete.

2. Dr. Dan Fisher of St. Paul contributed the information concerning this case, including follow-up reports on the clinical course.