hormone. It should be noted, however, that his data were obtained by a method of unprecedented sensitivity. They show an excretion about 10 times smaller than it is possible to determine by any of the published methods. Since at present we have no information of the nature and precision of the method employed, and since we have no control data on individuals who had not been exposed to beryllium, we have to postpone evaluation of these data.

The data given by Dr. Hardy show an apparently significant increase in beryllium excretion following the administration of cortisone (or ACTH?). In this case one should note that the analytical figures of beryllium excretion are about 100 times greater than those ever reported in the literature or found by us at the Saranac Laboratory in a rather large number of cases with pulmonary granulomatosis. Because methodical errors are an ever present possibility in the determination of beryllium, I hesitate to put too much weight on such data unless they are obtained by a known method of proved reliability.

Our own experience about the excretion of beryllium during cortisone therapy is limited to one patient whose urine showed no detectable amounts of beryllium before and during treatment.

This question whether beryllium can be mobilized by hormone treatment may be of great practical importance and will probably be solved in the near future. It appears difficult to believe that a compound which, for all practical purposes, seems insoluble can be made soluble by administration of a hormone. If, however, it should be possible to eliminate beryllium from the body, then there would be some hope that prolonged treatment might actually effect a more permanent cure in this disease.

ADDENDUM

Since this paper was given, I have had occasion to determine the urinary excretion of beryllium in a patient with pulmonary granulomatosis before and during cortisone treatment. For the analyses a chemical procedure was employed, the reliability of which has been established.[4] The patient, who had been exposed to various beryllium compounds in 1947 and 1948, began to experience fatigue, dyspnea and loss of weight about November 1950. In February 1951, administration of cortisone was followed by prompt improvement in all symptoms as well as clearing of the roentgenographic shadows. The daily excretion of beryllium, as shown in the accompanying chart, remained constant during the experimental period and was not influenced by cortisone. The excretion of creatinine, which according to Sprague and co-workers[5] is not altered by the administration of cortisone, was determined as a check on the completeness of the urinary collections.

DISCUSSION

DR. HARRIET L. HARDY, Boston: I think Dr. Klemperer has just filled us with questions and ideas, which I expect is all we can ask him to do. Dr. Grier worked here, in Dr. Aub's group, with beryllium intoxication before he went out to New Mexico.

4. Klemperer, F. W., and Martin, A. P.: Determination of Traces of Beryllium in Biological Material, Anal. Chem. **22**:828, 1950.

5. Sprague, R. G.; Power, M. H.; Mason, H. L.; Albert, A.; Mathieson, D. R.; Hench, P. S.; Kendall, E. C.; Slocumb, C. H., and Polley H. F.: Observations on the Physiologic Effects of Cortisone and ACTH in Man, Arch. Int. Med. **85**:199 (Feb.) 1950.