schedule, the lowest dosage has been 8 mg. every 12 hours. Up to now I have followed a plan of observing the patient for a time after the primary or initial course of treatment prior to placing that patient on a maintenance schedule. Two of the four patients have maintained the level of improved status for three and six months, respectively, after a course of treatment, while the other two relapsed two weeks after the therapy, to require a maintenance program immediately.

In arriving at our dosage schedule, we watched for and attempted to (1) avoid glycosuria and hyperglycemia, (2) significant alterations of electrolyte metabolism (as measured by shifts in sodium and potassium) and (3) signs of Cushing's syndrome, including plethora, striae, purpuric spots, hypertension, muscle weakness, osteoporosis, hypercalciuria and hirsutism. We have constantly tried to use as a guide in our therapy the fall in circulating eosinophils, which, as you know, should be more than 50 per cent. We have not found this test uniformly helpful, however, with our patients or controls.

Before closing I should like to mention a few therapeutic hints that have been reported to me referring to this therapy in other conditions. The colleague treating a considerable series of patients for lupus erythematosus has been able in some cases to manage with orally taken cortisone, prescribing amounts only slightly larger than the parenteral dose he had administered to the same patient. He has also found the addition of 1 to 2 grains (0.06 to 0.13 Gm.) of thyroid extract to enhance the effectiveness of the therapy. It is reported that the subsequent administration of insulin injection U. S. P. (e. g., 60 U. S. P. insulin units with 20 mg. of cortisone) much increases the desired effect. Compound F is reported to be just as potent orally as parenterally.

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DISCUSSION

DR. HARRIET L. HARDY: Dr. Van Ordstrand, do I understand you that it probably isn't sound to treat these people that are nondisabled?

DR. VAN ORDSTRAND: Well, I wonder if with our present knowledge we're justified in doing that. I am interested in Dr. Frawley's statement that their experience has indicated that the individual will still make the basic recovery and wonder if we are working on the right line in treating those whose disease is progressing unrelentingly and are trying to stop that death. Actually, I think we all feel that in beryllium poisoning we are using this until something better comes.

DR. HARDY: It is very tempting, when we see these people not disabled or before potential disability, which we cannot prophesy, to see whether or not we can do something about their reactivity.

DR. JOSEPH DE NARDI: I am interested in knowing what patients with berylliosis to give ACTH to, or cortisone—in what state of the disease, that is, whether in a severe state or whether in the incipient state. I have at the present time a group of patients here that are in the status quo, most of them doing light work. Some have shown a remarkable improvement in the last two years without any therapy, that is, specific therapy, as ACTH. I would hesitate to give those people ACTH or cortisone for the simple reason that we do not know exactly what is going to happen to these patients perhaps a year from now, and since these therapeutic agents are new to us, we have to be very careful. My present feeling is that the use of cortisone or ACTH should be decided on the basis of severity; that is, it is wise to use the drugs in very severe cases, when it is more or less a last resort.

DR. HARDY: Dr. Kennedy and Dr. Wright are in favor of it.

DR. B. J. KENNEDY: I see no reason why you can't curtail the disease in its early stage. Dr. Benning, of Montreal, has shown that by measuring the corticoids in the urine—if you