

INFORMATION GAINED IN PRETHERAPY AND POST-THERAPY PULMONARY FUNCTION STUDIES

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I SHOULD like to review briefly the experience that we have had on the Columbia Service at Bellevue Hospital and at Presbyterian Hospital with the use of cortisone and corticotropin (ACTH) in a variety of diffuse pulmonary diseases. All the patients have had, in addition to the usual clinical and roentgenological observations, serial studies of cardiopulmonary function. We have treated and studied six patients. We have studied one patient who had scleroderma with diffuse pulmonary involvement; two patients who had a granulomatous disease of the lung in which the lesions had a similar and uniform histologic pattern characterized by a large number of foreign body giant cells, birefractive crystals and epithelioid cells—in a moment I will tell you of one of these in more detail—one patient with a granulomatous lesion clinically and histologically characteristic of Boeck's sarcoid; one with chronic beryllium poisoning, and one for whom a diagnosis could not be established by a history of exposure to toxic substances or by a biopsy but who was believed clinically to have diffuse granulomatous disease of the lung.

As you see, we have had little experience in the treatment of patients with chronic beryllium intoxication. However, four of these six patients have had lesions which are histologically similar to the lesions seen in chronic beryllium intoxication, and all of them have had physiological defects which are remarkably similar to those which have been described in persons exposed to beryllium compounds. Characteristically they have had hyperventilation at rest and during exercise, normal or nearly normal arterial oxyhemoglobin saturation at rest, marked arterial oxyhemoglobin unsaturation after exercise and some elevation of the pulmonary artery blood pressure. In all these cases we have measured the oxygen-diffusing capacity of the lung and found it markedly reduced and believe that this interference with the diffusion of oxygen across the alveolar-capillary membrane is the fundamental defect in these cases. Because of the physiological similarity of these cases to those of beryllium intoxication, we hope that our observations will cast some light on the mode of action of ACTH and cortisone in chronic beryllium poisoning. Our experience with these cases has convinced us that cardiopulmonary physiological studies

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