

oxygen, according to the method of Riley, Lilienthal and associates.² Catheterization of the right side of the heart was performed simultaneously, and from these data the venous admixture, the physiological dead space and the oxygen-diffusing capacity were calculated.³ These studies late in therapy showed an increase in the oxygen-diffusing capacity. The venous admixture was reduced from its pretreatment level, and this indicates, we believe, a decrease in the flow of pulmonary blood to areas where no oxygen is transferred and where a significant carbon dioxide gradient exists. It seems likely that this change is brought about by improving diffusion in some part of the lung rather than by any large change in pulmonary blood distribution. The mild pulmonary artery hypertension which the patient had before treatment persisted after he had been given cortisone, and there was no change in the slightly elevated cardiac output.

Three weeks after therapy had been stopped, the studies were repeated, and a return to the pretreatment level or lower was found in nearly every measurement.

The only patient with beryllium poisoning whom we have treated thus far is a 39 year old man who was an electrician and who during the preceding five years removed and broke many fluorescent light bulbs. He did not know there was any hazard involved in this activity and took no precautions. He was known to have had an abnormal roentgenogram two years before he entered the hospital. His symptoms, however, developed only in the nine months before admission, and they developed quite rapidly and progressively. When admitted to the hospital he had severe, disabling dyspnea, abundant rales throughout both lungs, conspicuously clubbed fingers and obvious tachypnea. A roentgenogram of the chest showed a fine nodular infiltration of both lung fields. Before being treated with cortisone this patient had decreased lung volumes, normal maximum breathing capacity, normal oxygen saturation of the arterial blood at rest but a fall in saturation to 75 per cent after exercise, large alveolar-arterial gradients, reduced oxygen-diffusing capacity and slightly elevated pulmonary artery blood pressure. While being treated with 100 mg. of cortisone a day he had no improvement in his symptoms. In fact, at times it seemed that he was in worse condition. After four weeks of therapy pulmonary function studies revealed no significant change. In fact, the values were even a little worse. He had larger alveolar-arterial gradients, both at the high and at the low level of oxygen.

From our observations it seems that in some cases the course of pulmonary granulomatosis may be altered by cortisone. In one of four cases this change was manifest histologically in the disappearance of the epithelioid cells observed in the granulomas and physiologically in the improvement in the oxygen-diffusing capacity of the lung. We believe that in pulmonary granulomatosis after exposure to beryllium the primary physiological defect is an interference with the diffusion of oxygen and that physiological studies of the effect of therapy should include tests which measure this defect.

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