Correlative Clinicopathologic Studies in Polymyositis. By ROBERT O. BECKER *(by invitation)* and J. ALBERT SCHAEFER, *Departments of Orthopedics and Pathology, Veterans Administration Hospital and State University College of Medicine, Syracuse*.

The clinical entity of polymyositis was first described in Eaton (Neurology, 1954, *4*, 245) who characterized it as a disease of gradual onset occurring, in his series, in adults, and manifested by weakness, atrophy, and hyperirritability of large proximal muscle groups. Other authors (Shy and McEachern, J. Neurology, Neurosurgery, Psychiatry, 1951, *14*, 101; Garland. In: Modern Trends in Neurology, N. Y., Harper, 1957, p. 229) have described clinically similar processes under different names, and discussed histopathologic changes "characteristic" of the condition. The monograph of Walton and Adams (Polymyositis, Balti-

more, Williams and Wilkins, 1958) discusses the pathologic features of polymyositis in some detail, and depicts degeneration of muscle fibers, interstitial infiltration of inflammatory cells, extreme variation in size of muscle fibers, regeneration of muscle cells, and fibrosis as the principal changes. This in general agrees with the descriptions of other authors, but Greenfield *et al.* (An Atlas of Muscle Pathology in Neuromuscular Diseases, Baltimore, Williams and Wilkins, 1957) question the real value of muscle biopsy, and state that although the criteria may be roughly true, in many cases there is a large discrepancy between clinical and histopathological findings.

A series of eight cases observed during the past year at the Syracuse Veterans Administration Hospital were analyzed by clinical and laboratory methods, including the modalities of the electromyograph, the glucose tolerance test, and histopathologic technics. The following observations have been made: There has been great diversity in the histopathologic picture ranging from the "classical," described previously, seen in two cases, to one of minimal fat infiltration and no other significant pathological changes. In one case, the picture is more that of a "distal muscular syndrome of probable neurogenic origin" than that of polymyositis. From our present experience, we must agree with Greenfield *et al*. in their questioning of the value of histopathologic technics in establishing a diagnosis of polymyositis.

Clinical pathologic aids, however, are apparently more rewarding. Electromyography is a valuable diagnostic aid in distinguishing other diseases from polymyositis, with particular reference to anterior horn cell disease and muscular dystrophy. The electromyogram in the involved muscles in cases of polymyositis is characterized by polyphasic action potentials and bursts of high frequency discharges (Lambert, Trans. Amer. Neurol. Ass., 1954, *79*, 64). It is felt that the high frequency burst discharges reflect in some way the hyperirritability of the involved muscles. We have essentially substantiated this observation in that such activity has been found in all eight cases in this report. In addition, we have found that such bursts of high frequency discharges can be evoked by passive stretch of the muscle, and further, if the stretching procedure is repeated ten times at a rate of five seconds for each cycle, potentiation of the discharge will occur with each succeeding stretch. This aspect has become of considerable diagnostic value.

Three of the eight cases had been diagnosed as diabetes early in the course of their disease. Because the differential between polymyositis and diabetic amyotrophy is of importance, fasting blood glucose levels and glucose tolerance tests were done in all cases. In no case was the fasting blood sugar abnormal, but in four of the cases, the tolerance curve was displaced, being higher and more prolonged than normal.

Since the "classical" pathology of polymyositis resembles that reported in rats on a diet deficient in vitamin E, the patients were all placed upon a high dosage of this vitamin as synthetic alpha-tocopherol. Four cases have been under treatment for from six months to one and a half years, and in all of them,

clinical improvement has been marked. The remainder of the cases have been under treatment for less than six months and results are not yet definite. Other investigators using cortisone preparations or alpha-tocopherol in low doses have reported much less successful results.

It is important to stress the fact that this disease is more prevalent than previously thought and that the differential diagnosis from anterior horn cell disease is possible. Muscle biopsy may be helpful, but at this point is not conclusive. Clinical pathologic aids in diagnosis are most helpful at present. Some gratifying results have been obtained with therapy.

DR. WEINBERG: What is the age group of these people? What is the age of the youngest, particularly?

DR. BECKER: The youngest was thirty-five and the oldest was about fifty-five. None were over sixty; none under thirty.

DR. WACHSTEIN: If one had only a muscle biopsy, could one make the diagnosis?

DR. SCHAEFER: Of our eight cases, only two have fulfilled the criteria for polymyositis given by Greenfield or Walton and Adams on muscle biopsy.

DR. BECKER: One of our cases fulfilled the criteria for the diagnosis of primary neurogenic atrophy, and yet this patient has lived for over a year; biopsies have been taken and the clinical condition has reversed itself considerably.