Dr. Parkes Weber said that the skilogram of the skull showed a change in the sella turcica, which had been claimed as characteristic of this rare disease in children,1 namely, a very tall posterior clinoid process. R. G. Karshner (Amer. Journ. of Röntgenology, 1926), who called this disease "osteopetrosis," also laid great stress on the transverse metaphysial striation of long bones, which, however, was better known in connection with celiac rickets and other irregularities of the growth of bones in children.

Muscular Atrophy of Doubtful Origin.—Neill Hobhouse, M.D.

William C., aged 11 years and 9 months. Brought to hospital in July, 1931, because he had had a fit. The mother did not appear to have noticed that the feet were abnormal, but she admitted that he had been inclined to stumble since he had had diphtheria in 1928.

Family history.—There are five children in the family. One other has fits. No paralysis or deformity in any relative.

Present condition.—There is general wasting below the knee in both legs. Left calf half an inch smaller than right. Pes equinus with marked cavus. No fibrillary twitching has been seen. Knee-jerks and ankle-jerks absent. Superficial reflexes normal. Wassermann reaction (blood), negative.

The condition was suggestive of an early stage of the Charcot-Marie-Tooth type of atrophy, but the history showed that the patient had been in hospitals with diphtheria for seven months, and the possibility of the deformity following severe diphtheritic neuritis seemed worth considering. By the kindness of Dr. Rolleston and Dr. Borthwick I have been able to obtain evidence of his condition while in hospital. He was admitted to the Western Fever Hospital in January, 1928, and had a severe attack of diphtheria with palatal paralysis. He was unable to walk when first allowed up, but was noted as fit for transfer to the Northern Convalescent Hospital, March 15, 1928. At this hospital he was never kept in bed, and there is no note of absent reflexes, wasting, or deformity. There is a definite note that on May 30 there was no paresis of any kind, and he was discharged on June 1, 1928, without any diphtheritic sequelae.

The question is whether this may be an instance of abiotrophy being precipitated, or conditioned, by infective disease in those particular neurones. Dr. J. D. Rolleston tells me that he has known a case of Friedreich's ataxy following diphtheria. I have not obtained evidence of the condition being progressive, though I have been watching the case for three months.

Dr. J. D. Rolleston said that he had seen this boy in his own hospital. Though there had been some paralysis of the palate, there had not been either there or in the convalescent institution any obvious paralysis of the legs. Chronic diphtheritic paralysis was extremely rare. There were only about six such cases on record, and even in those it was difficult to eliminate some co-existing taint. The case of Friedreich's disease, to which Dr. Hobhouse had alluded, was one which Dr. James Taylor had shown before the Section of Neurology in December, 1918.2 The patient had had diphtheria in April, 1907, and was admitted into the Queen Square Hospital in December of the same year, when the condition was diagnosed as post-diphtheritic ataxy. He was re-admitted six years later as a case of Friedreich's ataxy. That was the only case that he (the speaker) knew in the literature which was comparable with the case now shown.

1 The primary (congenital-developmental) condition of "marble bones" or "osteopetrosis" must be distinguished from the secondary condition, which still better deserves the name, caused by metastatic hyperplastic carcinomatosis of bones and bone-marrow. This carcinomatous osteosclerosis or osteopetrosis is due to millions of minute metastatic emboli washed out from a primary prostatic carcinoma, which may be small enough to escape detection during life (cf. F. P. Weber, Trans. Med. Soc. Lond., 1929, lii, pp. 99-108).