

(*St. Bartholomew's*):—1020, acid, no albumin, no sugar, no blood. Diastase in urine.

Blood-count 30.7.30.—R.B.Cs., 4,730,000; Hb., 70%; C.I., 0.74; W.B.Cs., 5,600.

Differential count on 4,800 cells.—Polys. 41%; lymphos., 50%; large monos., 5%; eosinos., 2%; basos., 2%.

Blood Wassermann reaction ++

Hæmochromatosis, with Diabetes Mellitus, Hepatic Cirrhosis and Chronic Ascites.—F. PARKES WEBER, M.D.

The patient (A. P.), aged 54, an English picture-frame-maker, was admitted to hospital on November 4, 1930, in a somewhat drowsy state. The skin of his whole body was rather dry, and showed slight "slaty" pigmentation. There was moderate ascites and the liver and spleen both reached down to umbilical level. His urine was of specific gravity 1035; acid; free from albumin; it contained 4.6 per cent. sugar, much acetone and diacetic acid, but was free from indican and excess of urobilinogen. Brachial blood-pressure: 115/70 mm. Hg. The blood-serum showed no lipid turbidity; contained 150 mgm. per cent. cholesterol (within normal limits); and gave a very slightly positive indirect (negative direct) Hijmans van den Bergh reaction and negative Wassermann and Meinicke reactions. Blood-sugar (November 11): 0.230 per cent. Nothing abnormal by ophthalmoscopic examination. The blood sedimentation rate is slightly accelerated as compared to the normal average. The blood-picture (November 21) shows nothing special, excepting that in the differential count the lymphocytes are 31 per cent. and there are 7 per cent. eosinophils. The treatment is by insulin and tapping the ascites when required. It seems that the present illness was first noticed about four months before admission, when the patient commenced to pass more urine than he was wont to, and some darkness of his skin was observed. He had been under insulin treatment before admission, but nevertheless for two months he had been troubled with thirst. Previously he seems to have enjoyed good health, but had malaria 12 years ago, and pneumonia once when younger. Has apparently been a moderate beer-drinker.

Dr. Weber thinks that visceral without cutaneous hæmochromatosis may be present in some cases of hepatic cirrhosis with diabetes mellitus, even when there is no cutaneous pigmentation.

Discussion.—Dr. PARKES WEBER added that his case was very similar to that shown by Dr. Evans, but the slaty colour of the skin was slightly less. It resembled one of the cases shown at the last meeting of the Medical Section of the Society (at Guy's Hospital). Moreover, the cirrhosis of the liver in his case had already caused ascites, which had constantly to be tapped. The speaker desired to emphasize that the typical coloration in hæmochromatosis was usually not bronze as the name "bronzed diabetes" would imply, but slaty, and there might sometimes be a blue tinge in it. One of the earlier carefully-examined cases was that described by Dr. Maude Abbott, and the patient had been known in the ward of the hospital as "Blue Mary" on account of her slaty-blue appearance.¹

Dr. BERNARD MYERS asked whether there was any appearance of carotinæmia in these cases.

Dr. PARKES WEBER (in reply) said that cases which had shown no obvious pigmentation during life had sometimes been diagnosed post-mortem by the presence of hæmochromatosis in the liver and viscera. Clinically, when marked diabetes mellitus was found associated with obvious cirrhosis of the liver, hæmochromatosis should be suspected even if the skin showed no pigmentation. In answer to Dr. Bernard Myers, there was nothing in the present case to suggest any excess of lipochrome or carotin-like pigment (no xanthosis of the palms of the hands or soles of the feet).

¹ M. E. Abbott, *Trans. Path. Soc. Lond.*, 1900, li, p. 66.