ACUTE SPONTANEOUS HYPOGLYCAEMIA.


Cases of severe acute spontaneous hypoglycaemia have been so rarely reported in the literature that the recognition of the condition in practice is a reasonable excuse for placing a further observation on record. Moreover, the case now to be described exhibited certain features which were different from those observed in any other so far described.

Case Report.—On the night of May 14th, 1930, a poorly-nourished woman, 27 years of age and married, was admitted to the Mater Misericordiae Hospital, in a semi-conscious condition; she was restless as she lay in bed, and was unable to give an intelligent account of herself; her speech was monotonous and slow, and her expression was dull. On the following morning she was completely unconscious and slightly cyanosed, the breathing was deep and stertorous, she frothed at the mouth, passed urine involuntarily, the limbs were so rigid that the tendon reflexes could not be elicited, and the Babinski sign was bilaterally positive. The skin was moist, there was blepharospasm and the conjunctival reflex was diminished, but the pupils reacted normally to light. The urine, obtained by catheter, contained a trace of albumen, but no sugar, diacetic acid or casts; the blood-pressure was 90/70. No other abnormal findings were noted on clinical examination.

In discussing the diagnosis the possibility of acute hypoglycaemia was considered and thought possible, but, at the time, no information could be obtained about her condition before admission. A fasting blood-sugar estimation (Folin-Wu (1) technique) of 35 mg. per 100 c.c.—the blood was drawn and the estimation made while the patient was unconscious—supported the diagnosis of acute hypoglycaemia; the blood urea, determined at the same time, was 60 mg. per 100 c.c. A confirmation of the diagnosis of acute hypoglycaemia as the cause of the condition was sought and obtained by the intravenous injection of 10 grammes of glucose in 20 c.c. of distilled water; the injection took about four minutes and before it was ended signs of returning consciousness appeared. Immediately after the injection was given the patient became quite conscious and rational, she sat up in bed and asked where she was, the rigidity and blepharospasm disappeared and the Babinski sign became bilaterally negative. We considered that the condition, now thought to be undoubtedly hypoglycaemic in origin, was probably not due to the administration of insulin, because it became progressively worse in the first fourteen hours after admission.

Three hours after the glucose injection the blood-sugar was 111 and the blood urea 30 mg. per 100 c.c. By this time we were able to learn that the patient had never had diabetes and had never received insulin; she was, therefore, put on ward diet and given glucose drinks. Glucose was not given after the second day in hospital, except once for a glucose tolerance test.
The cause of the attack for which she was admitted to hospital, in the first instance, was evidently acute spontaneous hypoglycaemia. The previous history, obtained a day or two after recovery from the acute attack, showed that her first three pregnancies were normal, but the last baby, born in May, 1926, was still-born, and this delivery was followed by a leucorrhoeal discharge which disappeared on treatment. Amenorrhoea was present since the birth of the still-born child. A year previous to her admission to the Mater Misericordiae Hospital she complained of frequent attacks of dizziness, with "an inclination to faint," especially approaching meal-time. Because of these sensations she went to the out-patient department of another hospital and there received, on February 24th, March 3rd and 24th, 1930, three intravenous injections of novarsenobillon (0.3 to 0.45 gm.) but the blood Wassermann was continuously negative. Three days before we first saw her (May 14th) she actually became unconscious for a few minutes, and on the morning of the day before admission she was found hanging out of bed, with a dull listless expression on her face and unable to recognise her husband and friends. On the morning of the day of admission she was again found unconscious—with jerky movements of the limbs and frothy saliva around the mouth—and groaning, with an expression of pain on her face; she recovered partially and was sent into hospital that evening.

The fasting blood sugar on the morning of May 16th was 56 and the blood urea 22 mg. per 100 c.c., the plasma nitroprusside test was negative and there were no symptoms then, or afterwards, attributable to hypoglycaemia; at 4 p.m. on the afternoon of the same day, after a mixed ward breakfast at 9 a.m. and dinner at 2 p.m. the blood sugar was 122 mg. per 100 c.c. On May 17th, the fasting blood sugar was 66 without hypoglycaemic symptoms and one hour after a breakfast, taken at 8.30 a.m., consisting of an egg, about 3½ ozs. of bread, butter, tea without sugar, and 5 ozs of milk, it was 116 mg. per 100 c.c.; but at noon, without further food since breakfast, it had fallen to 87. For several subsequent days, while on an ordinary ward diet, with cane-sugar allowed in moderation, but no glucose drinks, the patient was kept under observation and the following figures in mg. per 100 c.c. were obtained by blood analyses:—Sugar (fasting) varied from 72 to 76, calcium 9.75, inorganic phosphorus 1.5, cholesterol 271, uric acid 2.9, creatinin 2.3, non-protein nitrogen 37.5, and bilirubin less than 0.1. During this period no hypoglycaemic symptoms occurred. The blood sugar values obtained after the ingestion of 100 grammes of glucose, 50 grammes of starch and 50 grammes of levulose are shown in the following table.

(The comments in brackets denote urinary sugar determined on a specimen obtained at the same time as the corresponding sample of blood.)