HYPOGLYCAEMIA IN THE NEUROSES

BY

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Within the last twelve years the importance of hypoglycaemic states has come to be fully recognized. Harris,¹ estimating the fasting blood sugar level of 1,867 patients, found sixty-seven to be hypoglycaemic. His criterion was a figure below 80 mg. per 100 c.cm. He quotes Hoxie and Lisherness,² who in 307 routine blood sugar studies in nervous and mental cases found ninety-five with fasting blood sugars below 70 mg. Several workers, including Finney,³ Cammidge,⁴ Wauchope,⁵ and Roth,⁶ have remarked the striking resemblance which these states may bear to various neurotic illnesses.

There are many factors which may lead to a low blood sugar, the chief among these being excess of insulin (therapeutic injections, tumours of the islets of the pancreas, and the so-called functional hyperinsulinism), lack of opposing secretion (hypo-adrenalism, hypothyroidism, pituitary dysfunction), and abnormal excretion of sugar (renal diabetes, lactation). In no case is the diagnosis easy, and there is a peculiar liability to designate the symptoms as being neurotic. Such complaints as weakness, apprehension, trembling, sweating, vertigo, ataxia, irritability, confusion, and amnesia, which may occur without the onset of convulsions or loss of consciousness, may easily be diagnosed as being due to an anxiety neurosis, hysteria, or neurasthenia. Moreover, the actual occurrence of hypoglycaemia brings about a reflex stimulation of the adrenals, and thus, as Cannon⁷ has shown, the symptoms result largely from sympathetic stimulation. Another point is that attacks of extreme weakness, accompanied as they usually are by a sense of impending doom, tend to produce a state of panic, and in such a patient a functional nervous disorder is easily developed.

The following three cases were all sent into the Jordanburn Nerve Hospital with the diagnosis of functional nervous disorder. In no case was hypoglycaemia suspected.

Case I

A married woman, aged 36, the mother of two healthy children, was sent into hospital with a history of vomiting after every meal for the last five or six years. She was also subject to hysterical attacks, during which she became extremely weak, somewhat incoherent, emotionally unstable, and would throw articles at her husband. At these times she felt a craving for sweets, and was invariably satisfied this craving. There was marked loss of weight, the weight in hospital being 5 st. 8 lb. The pulse was usually about 50, the blood pressure 90/70, and the basal metabolic rate was found to be -25. The sella turcica was shown on x-ray examination of the skull to be perfectly normal, and the thyroid was not palpable. Amenorrhoea had been present for the past year. The skin was dry and wrinkled, but not pigmented. There was no abnormality in the distribution or amount of hair.

A glucose tolerance test, using 50 grams of glucose and testing half-hourly specimens, gave readings of 92, 85, 85, 75, and 79 mg. per 100 c.cm. That this was not due to delayed absorption was shown by x-ray examination; the stomach emptied readily, and was of good tone. Seven minims of adrenaline to the fasting patient gave a satisfactory rise of the blood sugar. The figures at half-hourly intervals were now 80, 122, 118, 104, and 82 mg. per 100 c.cm. This suggested that the liver glycogen was adequate. Five units of insulin were now given to the fasting patient, and the blood sugar figures at half-hourly intervals read 73, 49, 26, 35, and 37 mg. per 100 c.cm. During this period she sweated profusely, was excited, wept, threw a book across the room, and complained of excessive fatigue. She spontaneously remarked that this attack was exactly similar to those which she had experienced at home, and apologized for her hysterical be-

haviour. She expressed a desire for something sweet, and regained control of herself immediately after a glucose drink.

Case II

A married woman, aged 57, was admitted to hospital with a diagnosis of diabetes and a history of attacks of strange behaviour, during which she became restless, confused, and extremely talkative. These attacks were not considered to have any connexion with her diabetes, since, on one occasion, her blood during an attack was found to have a blood sugar which was within normal limits. The attacks were thought to be hysterical.

Thirty years ago her doctor discovered, on examining her for some minor complaint, that there was sugar in the urine. He considered her to be diabetic, and put her on a diet, which she adhered to for four years but gave up. She had no further trouble until the onset of the present illness. It was not known whether in the meantime she continued to pass sugar in her urine. The present illness started two years ago, when she complained of fatigue, loss of weight, and irritability. She was not diagnosed as a diabetic until February, 1934, when she was put on a low carbohydrate diet.

She was admitted to hospital in February, 1935, by which time she was receiving a 2,300 calorie diet and 60 units of insulin per day. On admission she was passing sugar in her urine, but there was no acetone. Three days after admission she became restless, confused, picked at the bedclothes and pulled them about, tore her nightdress, and was extremely talkative. This attack came on just before lunch. She refused her food, and was given 20 grams of glucose with 10 units of insulin. She remained confused for four hours, but was better immediately after tea. She had a complete amnesia for this episode. A few days later she became confused, and rapidly passed into coma. Respirations were shallow and the pulse 110 per minute. The skin was bathed in perspiration, the face pallid, the pupils slightly dilated, and the intraocular tension distinctly lower than normal. The Babinski plantar response was indeterminate. A catheter specimen of her urine showed neither sugar nor acetone. (This was the only occasion while in hospital that her urine was found to be sugar-free.) She recovered consciousness fifteen minutes after an injection of 5 minims of adrenaline. On two other occasions when in coma the urine showed sugar but no acetone.

A glucose tolerance test, blood being removed at half-hourly intervals, gave 75, 126, 200, 240, and 224 mg. per 100 c.cm. During this period the urine sugar rose at one and a half hours to 5.2 grams per cent. Urines collected at two-hourly intervals over the twenty-four hours showed that the patient was never sugar-free, and on five occasions her fasting blood sugar level varied between 76 and 65 mg. per 100 c.cm. On all these occasions the urine demonstrated an appreciable amount of sugar. It was quite evident that, in spite of the diabetic nature of the glucose tolerance curve, the patient had also an exceptionally low renal threshold, and that even when in hypoglycaemic coma her urine still contained sugar.

Case III

A married man, aged 31, was admitted to hospital suffering from attacks of weakness, accompanied by ataxia, trembling, and a state of panic. He was considered to be a case of anxiety neurosis.

His present illness started three years ago, when he experienced faintness on leaving a cinema with his wife. These attacks of faintness have occurred with increasing frequency, and during the past few months he has been afraid to go out at all unless accompanied by his wife. Frequently, before an attack, he would be irritable and notice a tendency to bump into people when walking. He likened this to a mild state of drunkenness. He would feel weak, sweat, experience nausea and drowsiness, and feel afraid. He was sometimes too weak to walk home, and had to obtain a conveyance. He never lost consciousness. Usually, however, the attacks passed off on resting. He had noticed that they never came on after meals, and most commonly occurred after exertion. If feeling unwell before a meal he would immediately be relieved by taking food.

On physical examination there was no evidence of organic disease. X-ray examination showed the sella turcica to be normal. There was no evidence of endocrinopathy. A glucose tolerance test, the blood being removed at half-hourly intervals,

gave an inverted curve. The figures were 87, 75, 73, 73,* and 64 mg. per 100 c.cm. Five units of insulin given to the fasting patient resulted in the following figures at half-hourly intervals: 71, 53, 51, 56, and 62 mg. per 100 c.cm. A glucose tolerance test repeated on two subsequent occasions again gave inverted curves, but on removing blood every five minutes for half an hour an early peak reaching 123 mg. per 100 c.cm. was obtained. A rapid fall to below the fasting level then occurred within the thirty minutes.

The glycogen reserves in the liver appeared to be satisfactory, as 7 minims of adrenaline to the fasting patient gave the following curve, blood being removed at half-hourly intervals: 76, 102, 94, 92, and 85 mg. per 100 c.cm. On one occasion, before lunch, the blood sugar was found to be 42 mg. per 100 c.cm. The patient felt weak and experienced hunger pains, but there were no definite symptoms. On several other occasions when the blood was tested the level varied from 57 to 97 mg. per 100 c.cm.

Discussion

The general appearance, emaciation, low basal metabolic rate, amenorrhoea, low blood pressure, slow pulse, abnormal carbohydrate metabolism, and increased sensitivity to insulin in the first case justified the diagnosis of Simmond's disease with a resulting hypofunction of the adrenals, thyroid, and gonads. The hypoglycaemic tendency follows naturally from a relative overaction of insulin due to a lack of antagonism from the adrenals, thyroid, and pituitary. The hysterical attacks were shown to be due to a state of hypoglycaemia, and a typical attack was produced when the blood sugar was lowered by giving insulin.

In the second case the patient was predisposed to hypoglycaemia because of the abnormal excretion of sugar resulting from the low renal threshold. It is probable that the diabetes treated thirty years ago was in fact a renal diabetes, and that only within the last year or two had a tendency to hyperglycaemia been added. There can be no doubt as to the presence now in this patient of a condition of diabetes mellitus, but any use of insulin must be controlled by the sugar level of the blood and not of the urine. A diabetic whose blood sugar has for some time been at a high level may show hypoglycaemic symptoms when this level is reduced to what would normally be an ordinary figure. This, together with the continued presence of sugar in the urine, led to the attacks being considered functional rather than hypoglycaemic. Probably two factors contributed to the state of hypoglycaemia in this case—the excessive loss of sugar resulting from the low renal threshold, and also the excessive use of insulin.

The third case is more difficult to understand, as no apparent cause can be found to explain the tendency to hypoglycaemia, and physically the patient appeared to be in good condition. Gammon and Tenery⁸ describe a state of alimentary hypoglycaemia in which the fall in blood sugar, after the ingestion of food, has been sufficiently marked to cause slight symptoms of hypoglycaemia. In this case there appears to be an excessive insulin response to the ingestion of glucose, and in the absence of endocrine, hepatic, or renal abnormality one must consider the possibility of a simple hyperplasia of the islets of Langerhans. It is highly probable that several attacks, originally due to hypoglycaemia, especially if they occur outside, will predispose to future attacks of anxiety. The patient came to dread going outside alone, fearing the occurrence of his extreme weakness. As already remarked, the initial hypoglycaemic symptoms are largely sympathetic in nature, and the distinction between such a condition and an anxiety state is very difficult to make.

In the second case the patient was on a strict diabetic diet, while in the first and third the patients, who were under observation for a prolonged period, were on the ordinary hospital diet.

The blood sugars were measured by the Hagedorn-Jensen method.

Summary

Three cases of hypoglycaemia are presented, and the aetiology is distinct in each case. The difficulty of distinguishing between such cases and functional nervous disorders which they may closely resemble is discussed.

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THE INCIDENCE OF ERYSIPELAS

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In the literature of erysipelas there are many varied statements concerning the aetiology. While agreeing that the causal organism is a streptococcus, writers on the subject differ widely in their other views. Whether it is a specific organism or merely a variant of the streptococcus of scarlet or puerperal fever remains undecided.

The factors increasing susceptibility to infection are very vaguely stated. Beyond the necessity for a lesion of the epithelium most writers refuse to commit themselves, and take refuge in various possibilities such as alcoholism, poverty, overcrowding, and unhealthy conditions of life. That age plays an important part all are agreed, but very often wide limits are given. That sex plays an equally important part is also agreed, but apparently sex incidence varies from place to place. The season of maximum incidence is another moot point. Lord Horder¹ favours the spring, but in Ker's textbook *Infectious Diseases* the autumn is definitely stated. Similarly with other factors.

The subject, therefore, is well worthy of further investigation, and this article, which deals with the 686 cases of erysipelas notified in the landward area of Stirlingshire between July 1st, 1919, and June 30th, 1934, inclusive, is written in the belief that it is only by the pooling of many such local researches that a true knowledge of the disease may be gained.

Annual Incidence

In view of the close relation with the streptococcus of scarlet fever, it might be expected that the annual variation of erysipelas would show some relation with that of scarlet fever. Table I gives the number of cases of erysipelas and of scarlet fever in the yearly periods from July to the following June.

The only possible connexion which might be deduced is that there is an increased incidence of erysipelas in the year following a scarlet fever epidemic—an occurrence that might be expected theoretically as the result of the increased number of virulent streptococcal carriers present in the community during this period. The figures show no periodic increase and decrease in the incidence of erysipelas such as is found with scarlet fever. That the disease is practically stationary as regards total incidence is shown by the fact that in the three quinquennial periods the total number of cases is respectively 233, 229, and 224.