HYPOGLYCEMIA

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Introduction

The physician practicing Homœopathy today must be a "better than average" doctor. He must be continually refreshing his materia medica and keeping abreast of a rapidly expanding front of modern diagnosis and therapy. As a contribution to this and to the understanding of the basic mechanisms involved, I would like to discuss some experiences with functional hypoglycemia.

History

Early in the 1920's Seale Harris, Professor of Medicine at the University of Alabama, knowing that in disorders of other endocrine glands there are syndromes connected with overactivity as well as underactivity, wondered if there wasn't syndrome of overactivity of the Islands of Langerhans of the pancreas in contrast to diabetes mellitus. He went to Montreal to ask Banting who had never considered this point and knew of nothing written on it. In 1924 Harris first described several cases of overactivity of the islets of Langerhans, in many ways the opposite of diabetes.

In 1927 Wilder and his coworkers related hyperinsulinism and hypoglycemic shock. They were able to produce hypoglycemia in rabbits by the injection of an extract of an adenoma of the islets of Langerhans removed at operation. A year later Joslin clarified the mechanism of the many mental symptoms found in patients with hypoglycemia when he wrote "by lowering the blood sugar, certain oxidative processes become depressed to such a degree that the brain cells are effected in the same manner as in asphyxia." Portis and others have made significant contributions in the psychosomatic aspects of this condition. With little or no storage of glucose in the nervous tissue, it is not surprising that hypoglycemia produces very marked psychic symptoms, but "several writers have made the important point that the threshold at which psychological and mental changes occur varies greatly in

different individuals" and "the nature of psychiatric symptoms also depends on the previous organization of the personality."

Diagnosis

The diagnosis of hypoglycemia must be considered in any case of physical or mental symptoms which are partially relieved by eating. Unless one can obtain a blood sample for analysis during the initial phase of the symptom complex, a modified glucose tolerance test is essential to establish the diagnosis. Even the customary glucose tolerance test may not be helpful and in all cases the test must be continued to a five or six hour period in the oral method or three hours in the intravenous method. Edwards and Lummus on the other hand, use intravenous insulin correlating the symptoms produced with the symptoms of the case. I have found that a fasting blood sugar and a determination 4, 5 and again at 6 hours after 100 grams of glucose by mouth to be usually satisfactory and easiest on the patient. The level of 70 mg % or below is diagnostic.

Etiology

There are many causes of a lower than normal sugar level in the blood. Jerome W. Conn has presented an extensive classification which has more recently been simplified by Komfield of the Mt. Sinai Hospital in 1954. The latter estimates that 80-90% of cases of hypoglycemia are due to islet cell tumors, liver disease and idiopathic causes. The tumors are frequently multiple and only 50% of cases give clinical symptoms. That is, at autopsy they are not infrequently found in patients who died from other causes and have had no clinical symptoms referable to the islet adenoma.

Table I

Classification of Hypoglycemia (after Komfield)

I. Organic Hypoglycemia

1. Endocrine causes
   A. Pancreas (hyperinsulinism)
      a. Islet cell adenoma or adenocarcinoma or adenomatosis
      b. Islet cell hypertrophy or hyperplasia

B. Hypopituitarism (anterior lobe) or Simmonds' disease
C. Adrenal cortical insufficiency, atrophy or destructive granuloma or neoplasm
D. Hypothyroidism

2. Hepatic disease
A. Glycogen diseases (von Gierk's)
B. Fatty metamorphosis
C. Diffuse intrahepatic cholangitis
D. Toxic hepatitis
E. Diffuse carcinomatosis

3. Central Nervous System disease
A. Hypothalmic lesions
B. Brain stem lesions

II. Idiopathic hypoglycemia

1. Autonomic nervous system imbalance (increased vagal tone)
2. Alimentary tract disturbances (diminished absorption due to enzymatic disfunction or hyperperistalsis)
3. Renal glycosuria
4. Post-operative hypoglycemia
5. Lactation
6. Exhaustive muscular exertion
7. Starvation

With pancreatic adenoma as one cause of an abnormally low blood sugar, every case which presents this finding must be thoroughly studied to rule out islet cell tumor as the cause or a contributing cause. Typically, the blood sugar levels with pancreatic adenoma are extremely low, even 20mg% or below with attacks, and are relieved by the administration of sugar. Exclusion is not always easy, but by supplementing physical examination, blood analysis and response to therapy by a Sensitive Blood Crystallization Test, I have not had to subject any of my small series to retroperitoneal air injection or to exploratory laparotomy.

The lowest level of blood sugar in liver disease occur when fasting, and the glucose tolerance curve has a high plateau and then falls, while in the so-called functional hyperinsulinism, the lowest levels are several hours after eating, then rising somewhat before the next meal. Liver disease usually has other manifesta-

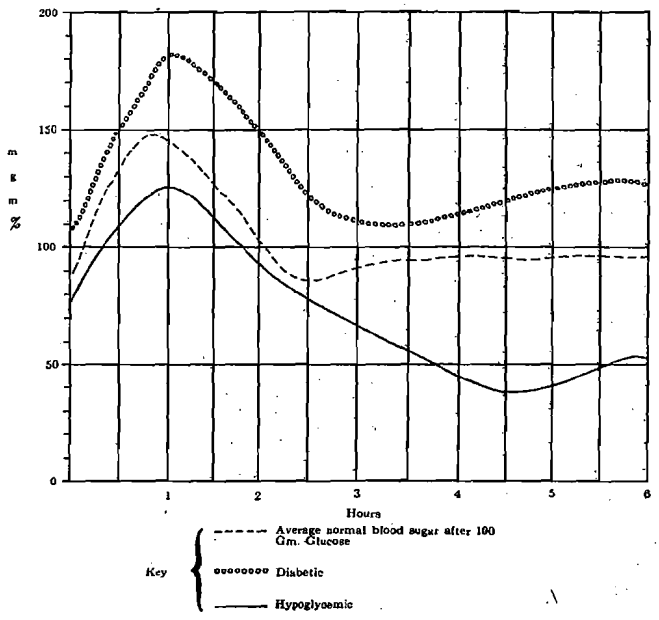
tions, while in functional hyperinsulinism we usually deal with a vagotonic individual who is hyperreactive in many ways. That the condition is not solely nervous but also chemically regulated is suggested by experiments of Anderson, Long and Sutton which showed that a rise in the sugar level in the blood entering the pancreatic artery results in an increase in the blood-sugar-lowering (or insulin) effect of the blood leaving the pancreas. This is undoubtedly the mechanism of the hypoglycemia seen following gastrectomy where we are dealing with the response of a normal pancreas to the dumping of only slightly digested carbohydrates into the small intestine.

Functional Hyperinsulinism

But let us return to the so-called functional hyperinsulinism. Here we are dealing with patients with a relatively normal G.I. tract and, to all appearances, a normal pancreas but with "insta-

Figure I

TYPICAL BLOOD SUGAR CURVES



bility of the autonomic nervous system which is normally responsible for adjustments of the blood sugar level." Conn likewise states "it represents a disturbance in the nervous regulation of the blood sugar level." "The vagus (parasympathetic) carries secretory fibers to the islets which control the finer regulation of insulin secretion." There is no evidence beyond this for the condition to be considered as representing an "hereditary metabolic disease."

Symptoms: Of itself, hypoglycemia can cause a variety of symptoms. Lack of ambition and easy fatigability may progress to extreme depression with suicidal tendencies and extreme weakness aggravated by every physical or mental effort and partially and temporarily relieved by eating or drinking stimulating beverages.

Treatment: Conn states that at least 70% of cases of functional hyperinsulinism are relieved by diet, the basis of which consists in 1) radical restriction of all concentrated carbohydrates and increase in protein and 2) frequent feedings. This is best outlined by Abrahamson and Pezet. Many authors recommend the full atropinization of all patients (1/150 gr. or less before each meal). But it is in those cases that do not respond satisfactory to diet, with or without atropine, that provoked my interest in this condition. For once a patient has inadvisedly left his diet and had a recurrence of his symptoms, it is literally months, even with diet plus atropinization, before he regains any sense of well being. Though the reasons for this seem obscure, when we consider the basic autonomic nervous imbalance that underlies this condition and the disrupting effect of worry and uncertainty on this imbalance, we get a glimpse of the reason and also the type of therapy that is required. The psychosomatically oriented physicians routinely employ psychotherapy. In my own experience, mechanoneural therapy offers a dependable and satisfactory method of direct approach to the autonomic nervous imbalance and relieves this lag in recovery that is both characteristic and distressing. In both of my most recalcitrant cases, two or three weeks was enough to bring them back to their previous state of health.

Case Reports

E.B., aged 62, came complaining of extreme fatigue, long

periods of wakefulness during the night, exhaustion from even slight exertion, pain in and around the right eye, irritability, feeling of faintness partially and temporarily alleviated by eating. His fasting blood sugar was 74mg% and three, four and five hours after a dose of 100 grams of glucose the blood sugar ran 52, 79 and 86. He was put on diet and gradually began to feel better. After four months, he left the diet and returned to chocolate and sweets. For 10 months of semi-retirement he felt well but consumed more and more chocolate and let his wife do the major portion of the work around the farm. Eventually, however, the old symptoms returned, and return to the diet gave no improvement over a 6 weeks period. A fuller investigation was carried out including a blood count of 17.5 grams of hemoglobin; 4.88 million RBC; 7,050 WBC with 54 segmented neutrophiles, 2% non-segmented neutrophiles, 42% lymphocytes and 2% eosinophiles. Urinalysis was normal. The protein bound iodine was 4.0 mgm%. Roentgen studies of the sella tursica showed no abnormality. Sensitive Blood Crystallization Test showed a weakness suggestive of enzymatic efficiency in the pancreatic area and sclerotic and spastic forms in the thyroid area indicative of a probable low level of function. After the second mechanoneural treatment he began to feel better and after the third he felt a return of his vigor and interest in life.

C.A.R. born 7/9/06, was first seen in the fall of 1954 with the complaints of lack of vital heat, shooting pains in the chest, lachrymation and, especially, a lack of all interest and ambition together with great weakness and exhaustion after any physical work.

This condition dated back about nine years but has increased in severity. He originally found that eating often and drinking considerable coffee during the day enabled him to keep going but more recently this has not given him any benefit.

His physical examination was not unusual, except for a deep healed wound from a gun shot in childhood involving a large part of the left side of the neck. Laboratory analysis showed a normal fasting blood sugar but three, four and five hours after 100 grams of dextrose by mouth, his blood sugars were 77, 59 and 67 milligram percent, respectively. He was put on a diet eliminating all

concentrated carbohydrates and coffee and providing at least six meals a day. He was also given a course of mechanoneural therapy and soon began to feel better and to take interest in his machine shop again.

During the winter of 1955, he gradually started to feel his old symptoms returning in spite of staying on the diet. He woke frequently at 4 A.M. with paresthesias of the forearm. In spite of the rather characteristic glucose tolerance curve of functional hyperinsulinism, with the return of symptoms while still on the diet, it was felt important to have a Sensitive Blood Crystallization Test which gave evidence of considerable congestion in the whole metabolic system, greatest towards the head and chest, an apparent overloading of the nervous system with metabolities. There also seemed to be some functional liver damage on the basis of passive congestion. The area of the pancreas seemed dense and heavy but no definite adenoma or tumor forms showed up. After another series of mechanoneural therapy treatment he felt better. The use of cold sitz-baths and two doses of *Phosphorus* 30x and later when effects seem to lag 200x brought back his sense of well being. The Sensitive Blood Crystallization Test taken in October showed general improvement with some metabolic congestion still present and a localized area of irritation in the left lower quadrant. There seemed to be also an irritation form in the heart area such as found in strain of the heart muscles. "The form is of inflammatory character which would be quite typical for the action of *Phosphorus*." The general vitality seemed somewhat reduced.

He gradually improved and was on the diet for one year with a minimum of weakness and tiredness and able to do his full job and enjoy his recreational time. Towards the end of 1957 he began drinking more frequently and more heavily than he had previously and early in 1958 he suddenly began to feel very poorly again. He eliminated alcohol and became very strict in his diet but did not feel greatly improved. He came under my care again in April complaining of weakness, tiring easily, impotence, insomnia and so forth. Because of the persistence and return of these symptoms, a full investigation was carried out. His fasting blood sugar was found to be 67 with a post-prandial blood sugar

at 1½ hours at 93. His blood phosphorous was 2.6 mg percent, calcium 11.2 mg percent, urea nitrogen 21 mg percent, chlorides 98.8 milli-equivalents per liter, basal metabolism plus 5, plus 6; CO_2 capacity was 65. The blood count showed 16 grams of hemoglobin, with 5.1 million red cells, 8,850 white cells with differential of 55% segmented neutrophiles, 41% lymphocytes and 4% eosinophiles. Urinalysis was normal. The seventeen Ketosteroids were 9 miligrams per 24 hours. The eosinophile count before the administration of 20 units of ACTH intravenously in 500 cc of water over an eight hour period was 88 per cubic millimeter. Eight hours later the count had dropped to 11 per cubic millimeter. X-rays of the abdomen showed a rather large opaque shadow low down in the abdomen which was believed to be a calcified mesenteric node of no clinical significance. No other abnormalities were found in this region. An x-ray of the pituitary fossa showed no abnormalities in size or contour. An incidental finding was the presence of many opaque shot in the upper cervical area and scattered throughout the soft tissue. A Sensitive Blood Crystallization Test was done which showed considerable disturbance in the stomach area of a type of gastritis but no clear forms were seen indicating degeneration to ulcers or malignancy. The forms were a combination of fibrotic and hole forms which would certainly tend more towards ulcer than any malignancy. There seems to be some functional thyroid weakness and imbalance together with a considerable loss of vitality and loss of body reserve. The pattern of general exhaustion had no obvious organic defects otherwise. Heart and liver showed no abnormalities at this time and the pattern was very different than the previous one. A chromatographic Analysis of the urine for amino acids was performed showing that tryptophan was absent, arginine and threonine were both lowered. This was interpreted as a tryptophan deficiency related to a niacin deficiency. The arginine is significant for a lowered hormone level to which testicular as well as adrenal and thyroid factors contribute. This patient was started immediately on a series of mechanoneural therapy treatments, this time without the use of any homœopathic remedy and after a few days began to notice definite improvement and was able

to go out and solicit business and felt after two weeks as good as he had at any previous time.

This case brings out two additional points. First, the typical glucose tolerance curve for functional hyperinsulinism with a normal fasting blood sugar, a drop to well below 70 (in this case 59) four to six hours after the test dose of sugar and a rise again in the six hour test. The second point is the lag in return to normal vitality on diet alone whereas our homœopathic remedy and, in my expérience, mechanoneural therapy reduce the convalescence period markedly.

Summary

The clinical aspects of hypoglycemia have been reviewed with special emphasis on the mechanism and treatment of functional hyperinsulinism.

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"HAHNEMANN"

Thanks to thee! gallant Knight!
Immortal be thy name
Shining bright as heavenly light
With sweet perfume of fame.

Suffering humanity crying so wild
Helpless wretched and poor
Finds relief so soft and mild
At the similimum's door.

We bow to thee oh blessed dear
So sublime yet ever so near.

DR. NARENDRA NATH BANERJEE, CALCUTTA
