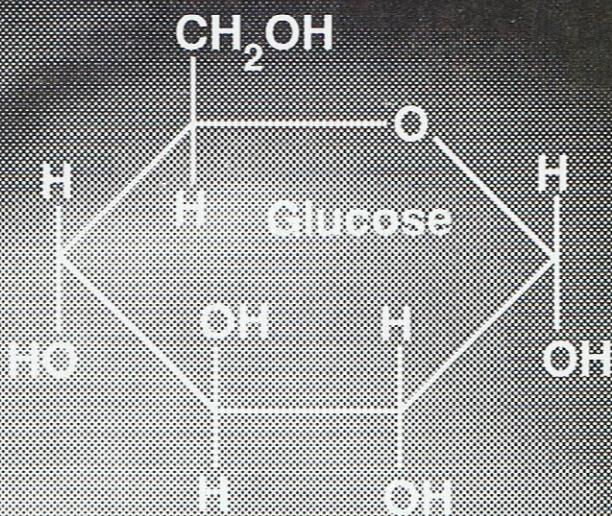


# HOPE FOR HYPOGLYCEMIA

It's not your mind, it's your liver.



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*I was privileged to have been able to thank him in person for his life, his research and his dedication to mankind."*

**Mrs. Joanne Veth**  
Teacher  
Fairfield, CT

# **HOPE FOR HYPOGLYCEMIA**

**It's Not Your Mind, It's Your Liver**

by

Broda O. Barnes, M.D., Ph.D.

and

Charlotte W. Barnes, A.M.

Revised Edition, 1989

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Second Printing, 1979  
Third Printing, 1980  
Revised Edition, 1989

**Robinson Press, Inc.**

1133 Riverside  
Fort Collins, CO 80524  
1-800-747-5395

Printed in the United States of America

Other books by the authors:

*Solved: The Riddle of Heart Attacks*  
(Robinson Press)

*Hypothyroidism: The Unsuspected Illness*

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## Foreword

Dr. Broda O. Barnes, medical pioneer, passed away on November 1, 1988, at age 82. His dedication to serving patients resulted in four books: HEART ATTACK RARENESS IN THYROID TREATED PATIENTS; HYPOTHYROIDISM: THE UNSUSPECTED ILLNESS; SOLVED: THE RIDDLE OF HEART ATTACKS; and HOPE FOR HYPOGLYCEMIA, as well as over 100 research papers on endocrine dysfunctions. He has left behind other manuscripts to be published posthumously.

Charlotte Webster Barnes passed away on August 30, 1980, after a lifetime of working with her husband in the medical field.

Following her death, Dr. Barnes married Helen T. Morgan, who currently lives in Bend, Oregon.

Dr. Barnes' work transformed the lives of many of his patients. People who had suffered for years and spent hundreds of dollars on treatment were finally able to find relief with the simple, effective, and inexpensive methods advocated by Dr. Barnes.

Some of these patients have given their own testimonials, along with some of his colleagues who saw the change brought about by his work.

Before his death, Dr. Barnes and his wife Helen established the BRODA O. BARNES, M.D. RESEARCH FOUNDATION to continue Dr. Barnes' research and work so future generations could benefit from his experience and build on it.

It exists today as a resource for people and medical professionals needing assistance. If you would like more information, please contact the Foundation at this address:

THE BRODA O. BARNES, M.D.  
RESEARCH FOUNDATION, INC.  
P.O. BOX 98  
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## Preface

Hypoglycemia like heart attacks, is a 20th century product. First recognized in 1924, it has had a stormy history. This has been due to a lack of knowledge about the cause of the syndrome and to the absence of a satisfactory laboratory test to confirm a suspicion of the condition. Now that both of these handicaps have been corrected, it is found that low blood sugar is very frequent and the physiology is very simple.

Since the introduction of antibiotics combined with better medical care, millions are now surviving into adult life creating a New Population never seen before in history. Susceptibility to infectious diseases frequently signifies a low thyroid function. In a previous book it was pointed out that patients with low thyroid function gradually deposit a glue-like material in the blood vessels which leads to heart attacks. Another effect of reduced thyroid activity is a sluggish liver which is handicapped in performing its many functions. In patients with poor thyroid activity, the liver is unable to produce sufficient sugar from protein during periods of stress, and hypoglycemia appears. Thyroid therapy restores the liver to normal function, and special diets with frequent feedings are unnecessary.

It is apparent that the escapees from early death from infections are susceptible to other diseases. Heart attacks gained the dubious distinction of killer number one due to the decline in deaths from tuberculosis. Hypoglycemia is probably more frequent than premature heart attacks, but it has been neglected since death from low blood sugar seldom occurs.

Application of an old test for insulin sensitivity to those with symptoms of hypoglycemia should make the diagnosis much easier and remove all doubts about the existence of frequent low blood sugars. The use of thyroid therapy, where indicated, promises hope for hypoglycemia and a prolonged life for those susceptible to hardening of the arteries.

## Biography

Broda O. Barnes was born April 14, 1906, in a log cabin on the side of a rocky slope in southern Missouri. His interest in research began as an undergraduate in the chemistry department at the University of Denver. He taught physiological chemistry for two years at Western Reserve University where he received a Master's degree in 1930. He earned a Doctorate degree in physiology in 1931 at the University of Chicago; for five years he taught physiology there. During this interval many publications on research on the glands of internal secretions appeared. He finished his medical degree in 1937 at Rush Medical College, interned at Illinois Research Hospital, and began the practice of medicine. For two years he was Assistant Professor of Medicine at the University of Illinois, then became Chairman of the Health Education Department of the University of Denver. He was called to the military twice: 1943-46 and 1950-51. He became Professor Affiliate in the Department of Physiology at Colorado State University, 1963-68. In the practice of medicine his interest has been prevention rather than treatment alone. Although engaged in General Medicine for 40 years, the thyroid gland has always enjoyed the center of the stage. Over 100 publications in scientific journals and four books have summarized his endeavors.

Charlotte Webster Barnes received her A.B. and A.M. at Oberlin College in geology with graduate work at Bryn Mawr College and at the University of Chicago. From the time of their marriage in 1932 she deserted the fossils to help in the field of medicine.

## CHAPTER 1

# A SIMPLE SOLUTION TO A KNOTTY PROBLEM

My introduction to hypoglycemia came very early in my medical career. The instructor in a special class in physiology injected each of us students with a dose of insulin. He felt that if students experienced the feeling of a diabetic who had received an overdose of insulin, when the students became doctors, they would be able to detect symptoms of hypoglycemia more readily. Although this happened over 45 years ago, the feelings remain vivid in my mind. I soon became weak, nervous and very hungry; I could have eaten a fried doorknob. One member of the small group developed severe mental changes. He became belligerent, irrational, unreasonable and at the end of the experiment, refused to take the antidote—orange juice. It was necessary to restrain him and inject a strong solution of glucose (blood sugar) intravenously. In a matter of seconds his sanity returned and he was the jovial, cooperative chap with the same pleasing personality exhibited prior to the hypoglycemia. In 50 years of work in the physiological sciences I have never witnessed such a reversal of personality in such a short time. More will be said about this in a later chapter.

Before finishing the medical school curriculum, I spent several years in research. At that time, diabetes was a rapidly expanding field; insulin had been discovered just 10 years previously. This milestone was made possible by the experiments of von Mering and Minkowski in 1889 in which the pancreas was removed from a dog. Almost immediately the blood sugar began to rise, and the next day copious quantities of sugar were spilling into the urine. A model for the study of diabetes had been found. In 1931 Professor Bernardo Houssay of Buenos Aires demonstrated that when the pituitary was removed from the diabetic dog, the diabetes was markedly improved. (*Endocrinology* 15:511 (Nov.-Dec.) 1931) In other words, diabetes was affected by glands other than the pancreas. With my colleagues, Dr. Houssay's

observations were confirmed, and it was found that adrenal deficiencies would also improve the diabetic state. We also demonstrated that dogs with only the pituitary removed were highly susceptible to insulin. Likewise, if the secretion of adrenal hormones was curtailed, the animals would develop hypoglycemia with a tiny dose of insulin which would cause no visible effect in normal animals. Little did I realize at the time that these experiments would some day be important in solving the riddle of spontaneous hypoglycemia in the human. At that time, hypoglycemia was recognized almost entirely in diabetics with an overdose of insulin, although one investigator had found similar symptoms in non-diabetics after fasting.

Beginning the practice of medicine in 1937 brought into focus many problems other than diabetes or hypoglycemia. Much of my research work had centered around the thyroid gland, and I found at once that patients with thyroid deficiencies were frequent and often were not recognized by competent physicians. In the first year of practice a young lady, who had been to one of the outstanding clinics in the country, came into my office. There had been nothing found to account for her weakness, the emotional instability and her lack of energy to meet life's problems. She was told that she had "neurocirculatory asthenia" and that she should go home and get used to it; there was nothing to do for it. This is as great a shock as a life-sentence in jail. Patients, such as she, do not cease to seek relief. I had become so well acquainted with the bizarre symptoms of hypothyroidism that such a diagnosis seemed certain. Within a month, on thyroid medication, she had a new lease on life and gradually became a productive, happy member of society.

Satisfied patients have never ceased talking about their improvement, and they have encouraged friends with any complaints to try their new doctor. A practice was early established from the "washouts" of disappointed patients at the hands of other doctors. Two unsolicited tours of duty in the military, and a change in location for the health of a member of my family did not alter the stream of tired people looking for relief.

The years of research on hypoglycemia were put on the back burner, but by 1973 a controversy had arisen in the medical profession about the incidence of hypoglycemia. On one extreme were physicians specializing in the new disease, and hence seeing many such patients

who migrate to a place in which they can find help, while other specialists in internal medicine were denying that many hypoglycemic patients existed. Those with symptoms of this disorder were called "psychosomatic" — the wastebasket for physicians' ignorance.

Hypoglycemia came off the back burner suddenly when a secretary came into the office with a 12-year history of repeated disappointments. She had passed out in the office where she was employed as a legal secretary. This had to be "heart attack," and she was rushed into intensive care. After three days of hospitalization, and hundreds of dollars of sophisticated tests, she was dismissed with a diagnosis of "hysteria." Thousands of dollars were spent in repeated hospitalizations and tests. Finally some help was found in a private office when hypoglycemia was uncovered. The usual high protein and low sugar diet gave considerable relief, but she knew that she was far from being well. At someone's suggestion, she came to my office and unraveled her story. She had so many of the symptoms of hypothyroidism, there was no doubt that this was her major complaint. Suddenly a light dawned. If she was suffering from hypothyroidism as well as from hypoglycemic syndrome, might the two occur together? For 40 years I had treated thousands of cases of low thyroid and not one of them had suffered from hypoglycemia during the thyroid treatment. This series of patients began before hypoglycemia was understood, and patients were not questioned about it. Only in recent years have patients been aware of hypoglycemia and have mentioned it. They have been treated for their hypothyroid symptoms, and there have been no more complaints about hypoglycemia. This would suggest that hypoglycemia might be one of the symptoms of hypothyroidism.

My early work, years ago, on animals with glandular deficiencies clearly showed that they were quite sensitive to insulin. A review of the physiology of the various glands and their influence on sugar metabolism left no doubt that the cause of functional hypoglycemia was at long last clearly understood. The following chapters will explain how thyroid deficiency may lead to the hypoglycemic syndrome.



## CHAPTER 2

### Definition and Brief History

Hypoglycemia means a blood sugar low enough to cause symptoms. Since most of the symptoms involve the central nervous system, Professor Marks in England prefers to call the condition "Neuroglycopenia" which is a jaw breaker meaning a lack of sufficient sugar within the cells of the brain. Technically he is correct, for the nervous manifestations are caused by the low sugar content within the brain cells. However, the only way a low sugar content of the brain cells can occur is to have the blood sugar fall, resulting in a lack of glucose to replace that used up by the brain. In the past, some have contended that hypoglycemia is not a disease but only a condition of the blood. Even the Hypoglycemia Association, Inc., Ashton, Maryland 20702, in their pamphlet "Hypoglycemia and Me" states that hypoglycemia is not a disease but a symptom that something has gone wrong. Hypoglycemia has come to mean far more than a symptom. The time has come when Hypoglycemic Syndrome would fit the facts far better. Webster defines syndrome as "a group of signs and symptoms that occur together and characterize a particular abnormality." The low blood sugar reading is only a laboratory finding that signifies the hypoglycemic syndrome may be present. The two are not always found together since some diabetics taking insulin are occasionally found with a low blood sugar, but entirely free from any discomfort. Undoubtedly if the blood sugar remains low long enough, the hypoglycemic syndrome follows. So much for the definition of hypoglycemia; now for a brief history.

It seems likely that the hypoglycemic syndrome has been present for centuries since some of the diseases associated with the syndrome have been known in the past. It was not, however, until the introduction of insulin in the treatment of diabetes in 1921 that hypoglycemia was recognized. If an overdose of insulin is administered, some hours later the patient may become nervous, break out in a sweat, become weak, and hungry. If untreated with food of some kind, he may

become mentally confused and even go into convulsions and coma. The physicians treating diabetes soon learned to watch for the early signs of low blood sugar and to relieve them at once with orange juice or intravenous sugar if necessary.

A very astute physician, Dr. Seale Harris of Birmingham, Alabama, was the first to recognize the hypoglycemic syndrome in a nondiabetic not taking insulin. A fellow physician complained of weakness, hunger and nervousness about three hours after the last meal. He could not continue with his duties unless food of some kind was ingested; thereupon he felt normal again. Dr. Harris checked the blood sugar during the periods of weakness and found that indeed the blood sugar was as low as that found with an overdose of insulin. In 1924 (*Journal of the American Medical Association*, volume 83, 1924, page 729) he reported this and two other cases showing the hypoglycemic syndrome without any insulin administration. They were relieved with five small meals daily about three hours apart. Dr. Harris reasoned that since the hypoglycemic symptoms were occurring during fasting between meals, an overnight fast might show more reactors. In some 200 patients given a blood glucose test before breakfast, 12 showed low readings and some of the symptoms of hypoglycemic syndrome. It is apparent that even before the hypoglycemic syndrome was recognized as such, 6 percent of the tested individuals had signs and symptoms of hypoglycemia.

A few years later Dr. E. M. Abrahamson had a very puzzling case of rapid heart beat. This would occur in the middle of the night and nothing could be found upon examination. The patient was referred to a heart specialist who could find nothing wrong and who made a diagnosis of "cardiac neurosis." This is a polite way of saying that it is all in one's mind. Finally an attack occurred in the afternoon, and the patient came in demanding that something be done. Thinking something wrong might be found, Dr. Abrahamson drew some blood. To his amazement the blood sugar was very low. The observation aroused an interest in this new syndrome; Dr. Abrahamson went to visit Dr. Harris and thereupon embarked on a career in this field. It culminated 16 years later in a book, *Body, Mind, and Sugar*, published by Holt, Rinehart and Winston, New York 1951 (A. W. Pezet, co-author).

This classic has undergone 25 printings, the last in 1973. Many facts about hypoglycemia are recorded in the book, but newer knowl-

edge may give a different interpretation to some of the facts. Thus, like Dr. Harris, Dr. Abrahamson felt that all cases of hypoglycemia were due to the presence of too much insulin. Later information has clearly shown that there are several causes of the syndrome. The important part of Abrahamson's contribution was the prevalence of the syndrome and its association with many other established diseases. Hypoglycemia was accused of causing allergies, hay fever, asthma, rheumatic fever and ulcers by Dr. Abrahamson because he found the hypoglycemic syndrome frequently among these diseases.

It is not unusual for a fellow traveler to be accused of a crime. Circumstantial evidence would convict many an innocent bystander if additional facts were not presented to prove him innocent. Such was the case in heart attacks. Circumstantial evidence convicted cholesterol as the villain in heart attacks for many years. Finally the truth emerged when it was shown that indeed a high cholesterol is frequently present in heart attacks. However, evidence has been accumulating for 100 years indicating that the real culprit is a thyroid deficiency, and cholesterol, which is usually increased in hypothyroidism, is only an innocent bystander.

Just because the hypoglycemic syndrome is present in several other diseases does not prove that the hypoglycemia has caused the various ailments. In the chapter on mental diseases, Dr. Abrahamson showed that hypoglycemia was often present. His own work and likewise that of others have clearly shown that hypoglycemia may be present. His inference that the low blood sugar caused the mental disease is not justified. No one will doubt the association, but we shall find later that "guilty by association" is a dangerous conviction. Likewise, in the case of alcoholics, do they drink because of a low blood sugar or is the hypoglycemia an accompanying condition of some other disease? Although cases of suicide, murder and epilepsy may show a hypoglycemia, it may be only Mark Twain's "cussedness of coincidence," and it may signify that a search should be made for other diseases.

The most significant contribution for treatment was made by Dr. Jerome Conn at the University of Michigan in 1936 (*Jour. Clin. Invest.* 15, 673, 1936) when he recommended a low carbohydrate, high protein diet for cases of hypoglycemia. About 58 percent of the protein can slowly be converted to glucose without a rise in the blood sugar

which would cause a discharge of insulin. This management of hypoglycemia has persisted until the present. From the standpoint of nutrition in hypoglycemia as well as nutrition in most other diseases, Dr. Carlton Fredericks' name stands out above the others.

## CHAPTER 3

### The Importance of Glucose

Why does a drop in glucose in the blood stream cause such profound mental changes? It is because glucose is almost the only fuel used by the brain and central nervous system. It is the combination of glucose with oxygen that keeps the brain ticking. The absence of either one will lead to unconsciousness at once. This was clearly demonstrated during World War II in the training of high altitude flyers. I worked for nine months in one of the low-pressure-chambers used for indoctrination of the flyers. Air was pumped from the chambers simulating 38,000 feet altitude. It was necessary to wear oxygen masks at all times. To impress upon the men the seriousness of fouling the lines, thereby cutting off oxygen, a trainee would be asked to remove his mask. In less than 30 seconds he would slump over, unconscious, and some one else would have to replace the mask. If one could suddenly cut off the supply of sugar to the brain, unconsciousness would follow just as suddenly, since it is the sugar that the oxygen burns, maintaining the conscious state. Fortunately, there is no way of suddenly dropping the sugar content to zero. As the blood sugar goes down, the amount in the brain is reduced slowly producing symptoms that can be recognized by those acquainted with hypoglycemia. Our problem is to acquaint the public with the symptoms that may occur and to remind the physicians that this syndrome is far more prevalent than they have thought.

Three classes of food are available for man and animals in nature. They are somewhat similar to the three sources of energy available for modern industry. The carbohydrates resemble natural gas in being the fuel of choice because it is readily available and easily moved from place to place. The proteins correspond to crude oil that can be modified and used in our ovens and hot water heaters. About 58 percent of the proteins can be converted into glucose by the liver. The fats that contain more energy correspond to coal and, although they are the main source of energy for muscles and other tissues, 10

percent of the fat molecule can be converted into glucose if needed. This source saves the lives of our hibernating animals that do not eat during the long winter months. The glucose-stores would soon be consumed but the fat, used all winter, supplies enough glucose to keep the brain functioning, and the old bear comes out in the spring as smart as he was when he went to sleep. Prolonged hypoglycemia in either animal or man leads to irreversible brain damage.

The liver can furnish a small amount of glucose from other sources. Alcohol can be converted into glucose. During muscular contraction lactic acid is produced which can become glucose. The liver can convert fructose of honey, galactose of milk and the mannose of complex sugars into glucose. But the majority of glucose for fuel comes from the carbohydrates and the proteins in the diet.

Each individual experiences some degree of hypoglycemia after each meal. During fasting the blood sugar is kept at a relatively constant level by the release of glucose from the stored glycogen (glucose complex) in the liver or by the manufacture of glucose from proteins. As soon as a meal begins to digest and to be absorbed from the intestines, the blood sugar rises due to the carbohydrate in the food. This signals two changes: first, insulin is released from the pancreas in order to store the new supply of sugar and to aid in the formation of fat; second, because of the rise in blood sugar, the liver (1) ceases making more glucose from protein and (2) ceases releasing the stored glucose in the liver. For some time the body derives its glucose from the meal. When digestion and absorption are completed, the blood sugar begins to fall toward the fasting level again. The pancreas ceases the release of extra insulin which is no longer needed. The liver must again start furnishing glucose to the blood. However, it is not an electrical signal that immediately turns on the liver similar to the electronic gadget that turns a radar range off at a specified time. The liver must have a signal, and that turns out to be a decline in the blood sugar slightly below the normal range. Then the liver again starts releasing and manufacturing glucose. It becomes obvious that everyone has mild hypoglycemia sometime after each meal, or even after each snack between meals. If the liver is functioning normally, this drop in blood sugar is so small that no symptoms arise, and the individual is not conscious of any fluctuations of the blood sugar.

## CHAPTER 4

# The Multiple Causes of Hypoglycemia

Hypoglycemia was first recognized as the result of overdoses of insulin to diabetics. Obviously too much insulin can lower the blood glucose level to the point that serious symptoms develop. It was only natural for Harris, who first noted hypoglycemia in non-diabetics, to assume that the individuals were producing too much insulin. Before long a patient was found with a tumor of the insulin-producing cells in the pancreas which produced insulin continuously day and night. Relief was only obtained when the tumor was surgically removed. Such cases are rare; less than one thousand have been reported in the medical literature of the world during the past 50 years. Many doctors including myself have never seen such a case during their years of practice.

It was mentioned above that the liver not only stores glucose during digestion but also releases the stored glucose when needed. Furthermore, if glucose is in short supply, the liver can produce huge quantities from proteins and a smaller amount from fats. If the liver is removed from an animal, at once the blood sugar begins to fall rapidly, and death from hypoglycemia will soon occur unless glucose is given intravenously. This leaves no doubt that the liver is the most important organ in the body for the maintenance of a normal blood sugar. It should come as no surprise that diseases of the liver might cause hypoglycemia. Hepatitis, an infection of the liver cells, will markedly interfere with functions of the cells, and hypoglycemia is common during such infections. Likewise, poisoning with heavy metals or chemicals will lead to hypoglycemia. Alcoholic cirrhosis leads to scar tissue in the liver and destruction of liver tissue. Here again, hypoglycemia will raise its ugly head. Cancer of the liver cells is rare, but when it occurs, the blood sugar will soon fall. The liver is a favorite site for metastases of cancers elsewhere in the body, and these metastases soon interfere with the liver's ability to store or manufacture glucose. Excessive exercise may require more glucose than the

liver can furnish. Again the hypoglycemic syndrome may appear, but rest soon abolishes the symptoms in most people. Hence, a multitude of diseases may affect the liver, but fortunately the sum of all of them is not enough to make hypoglycemia very frequent.

Thus it is apparent that an overproduction of insulin may cause a few hypoglycemias, but some malfunction of the liver is a far more common cause. As important as glucose is for the brain, mother nature did not leave the liver alone to combat stress and furnish glucose as needed. Some of the other endocrine glands support the liver in maintaining a normal blood sugar; these will be discussed in the following chapter.

## CHAPTER 5

# The Intricate Mechanism for Controlling Blood Sugar

From the previous chapters it is apparent that two of the major factors in the control of blood sugar are insulin, for the burning of glucose, and the liver for storage and manufacture of glucose when more is needed. Since glucose is the major fuel for the brain, and since the human brain is the factor that elevates man above the other animals, mother nature took special precautions against a lack of glucose in the bloodstream at all times. The liver is the largest organ in the body and it has many, many functions. The storage and the manufacture of glucose may be the most important, but there are checks and balances on the liver as there are on other organs in the body. Relative to the glucose-functions of the liver, the other endocrine glands play a role in the action of the liver. For more details on this subject, the reader is referred to the textbook of Best and Taylor, *The Physiological Basis of Medical Practice*, third edition, The Williams and Wilkins Company, Baltimore, 1943. Dr. Best was one of the co-discoverers of insulin; he died in 1978.

Insulin is produced in the beta cells of the pancreas and is responsible for the burning of glucose by the tissues, for the storage of sugar in the liver, and for the conversion of glucose to fat. Hence its effect on the blood glucose is to lower any elevated values and prevent diabetes. Yet, also in the pancreas is another hormone that elevates blood sugar. Glucagon is produced in the alpha cells of the pancreas, and it will stimulate the liver to release glucose in case the blood sugar falls below the normal range.

The pituitary gland, situated at the base of the brain, is likewise important in regulating the blood sugar. The pituitary produces more than one hormone, but one of the important ones is the growth hormone. If this hormone is produced in excess during growth, a giant may result. This is rare, but on one occasion I had a patient

measuring 8 ft. 6 inches. In order to look into his throat, I had him sit in a chair; his head and mine were on comparable levels. If the pituitary becomes overactive after growth has been completed, the facial bones are distorted, and the fingers become thickened; the condition is called acromegaly. The growth hormone will stimulate the liver to release glucose causing a high blood sugar and the escape of sugar into the urine. On the contrary, if the growth hormone is deficient, not only will the growth be stunted, but the liver is sluggish and hypoglycemia may be present.

The influence of the pituitary on diabetes was mentioned in chapter one. In addition to confirming Houssay's work, my colleagues and I found that animals with only the pituitary removed were so prone to hypoglycemia that they had to be fed at frequent and regular intervals. Obviously the liver was not functioning properly in storing and releasing glucose when needed. Animals or humans without pituitary hormones are highly sensitive to insulin injections and cannot tolerate small doses which would cause no effects in normal controls. Extracts from the pituitary glands of animals from the slaughter house, when injected into normal animals, will cause a rise in the blood sugar and if continued long enough will produce permanent diabetes. It is evident that the pituitary hormone has a profound effect on the liver of normal animals and of man. The pituitary also produces hormones stimulating the thyroid and the adrenals each of which may have an influence on the carbohydrate functions of the liver. Unfortunately the pituitary hormones are proteins, and are destroyed by digestion if administered by mouth. Furthermore, injection of animal proteins into the human leads to immunity to the foreign protein. Hence, unlike insulin, pituitary therapy to the human is not successful. It is fortunate that hypoglycemia from pituitary deficiency is seldom seen.

In 1934 my colleagues and I studied experimentally the effect of adrenal deficiency on diabetes and the sensitivity to insulin. The adrenals occur in pairs, one above each kidney. If one adrenal was removed first, then the pancreas removed, the resulting diabetes was milder than in the presence of both adrenals. If the pancreas was removed first and the insulin requirement established, the removal of one of the adrenals caused the insulin requirement to fall to about half the previous level. It is obvious that the adrenals are also important to

the liver in its control of glucose metabolism. The injection of active adrenal extracts or administration of synthetic hormones from the adrenal cortex will elevate the blood sugar of normal animals or the human, and glucose may appear in the urine. Animals with a reduced hormone-output of the adrenals are highly sensitive to insulin injections and will go into convulsions with small doses. Adrenal abnormalities are again rather scarce, and hypoglycemias from adrenal deficiency are not often seen.

Even the sex glands may have an influence on carbohydrate metabolism. Too much of either male or female hormone will suppress the pituitary to some extent since the hormones have a feedback on the pituitary to keep a proper balance on all of the hormones. The influence of the sex hormone being indirect through the pituitary is comparatively mild and is seldom important in hypoglycemia.

### THYROID AND HYPOGLYCEMIA

Last but far from least is the influence of the thyroid gland on hypoglycemia. This is the gland that has been most neglected, and unfortunately the one most often at fault. Its action is more subtle, and has been overlooked by most investigators ever since hypoglycemia became fashionable. Over 50 years ago it was reported that thyroid administration aggravated diabetes; more sugar was excreted when the diabetic was given an excess of thyroid. This information led to an attempt to treat diabetes by removing the thyroid. Less insulin was necessary, but the side-effects from lack of the thyroid were far worse than the diabetes; further thyroidectomies were abandoned. However, in a forthcoming book it will be pointed out that 98 percent of the diabetics are lacking in thyroid and that bringing their metabolism up to the normal range not only improves the symptoms but also inhibits hardening of the arteries which is the diabetics' worst problem. The Hypoglycemia Association in their pamphlet "Hypoglycemia and Me" caution against the use of thyroid since, if the patient suffers from adrenal deficiency, "adrenal crisis" may be precipitated by thyroid therapy. This is true since raising the metabolism requires more adrenal hormone as well. However, these cases are rare, and if the physician finds a very low blood pressure before any therapy is given, 5 mgs of prednisone may be required with the thyroid. Many patients who feel worse when thyroid therapy is started also need adrenal, and

the use of 5 mgs of prednisone daily along with the thyroid therapy may turn the failure into a resounding success. Most authors on hypoglycemia do not mention the thyroid, or if they do, little attention is paid to it.

These authors have overlooked Best and Taylor's statement that the profound effects on carbohydrate metabolism by removal of the pituitary can be reversed by thyroid administration. This would indicate that much of the effects on the liver are due to thyroid atrophy after loss of the pituitary. In other words, the thyroid has a profound effect on the liver. We have other evidence that a lack of thyroid is accompanied by a sluggish liver. In the first place, it has been apparent for a century that patients with myxedema (very low thyroid activity) may have a yellowish tint to their skins. This has been found to be due to the presence of too much carotene in the blood. The liver converts carotene into vitamin A which is colorless. Under the administration of thyroid, the liver becomes more active and the carotene soon disappears. In the second place, the cholesterol level of the blood is usually elevated in hypothyroidism. Thyroid administration will lower the cholesterol, and if too much is given, the cholesterol will fall below normal. The liver converts cholesterol to bile salts which are eliminated in the bile; this process is the usual means of eliminating excess cholesterol. The liver is sluggish in this function among thyroid-deficient individuals. A third example of a sluggish liver is in regard to handling excess glucose during the glucose tolerance test. The hypothyroid patients store glucose more slowly in the liver and may excrete glucose in the urine. Most of these are called prediabetic. Yet when they are placed on thyroid, the glucose tolerance test is normal. Many of the alleged prediabetics are really low in thyroid function which may account for the fact that so many of them never become diabetics. It seems certain that in hypothyroidism the liver is sluggish.

Since a sluggish liver is the most common cause of hypoglycemia, it should follow that the hypothyroid patient is highly susceptible to low blood sugar. At any rate, the secretary mentioned in chapter one was obviously a low thyroid in addition to her hypoglycemia. Diet had helped, but had fallen far short of solving all of her problems. Without any change in diet, she was placed on thyroid therapy and told that this should correct her syndrome of hypoglycemia. Naturally she was skeptical after 12 years of suffering. Some of her other symptoms

began to fade, as time passed. About six weeks later, it was decided to check her reaction to carbohydrates. She and her husband were invited to dinner. During the meal she was picking out the high protein foods and carefully avoiding the starches. Even my wife was not aware that an experiment was being performed. For dessert, a large piece of lemon-cream pie was placed in front of her. Her eyes bulged as she pointed to the pie saying, "That will kill me." I assured her that if it did, I could sign the death certificate. After encouragement, she ate the pie with gusto. She was still not convinced, however, and decided to make a real test. Unbeknown to me, she bought a pound box of chocolate candies and ate the entire contents at one sitting. The following day she confessed and admitted that her hypoglycemia had vanished.

Since my interest in hypoglycemia had been rekindled, many patients diagnosed as hypoglycemic elsewhere, have been placed on thyroid therapy. The results have been uniformly good. Individuals so treated can eat with impunity foods which had been avoided for years. Not only can they eat anything they want, but most of the other symptoms of hypoglycemia and hypothyroidism have disappeared also. For the first time in years they have energy and can enjoy life. So far, among those seeking my help, there have been none with liver failure due to infections, poisons, alcohol or malignancies. In any of these cases, thyroid therapy would be useless. However, for the functional hypoglycemias, the odds for improvement are very high.



## CHAPTER 6

### What is the Incidence of Hypoglycemia?

One of the hottest "rhubarbs" in modern medicine is over the question of hypoglycemia. Following Harris' demonstration in 1924, physicians began to encounter similar cases. More frequent feedings, less sweets in the diet and the high protein intake recommended by Conn in 1936 have turned miserable, handicapped patients into healthy, happy, productive members of society. News of relief from a previously unrecognized disability travels faster by the grapevine than by publication in a scientific journal. Comparatively few people have read the report of Dr. Harris; it did not attract the attention that it deserved. However, the neighbors of the patient, his relatives, their neighbors and their relatives were already aware of a new miracle in a comparatively short time. The busy physician has relatively little time to read; a few medical meetings each year and a few medical journals make up his continuing education. A rare, new disease is seldom placed on the program of the medical meetings.

Sometimes a rare, new disease becomes rapidly more prevalent, and if it causes death, it draws considerable attention. This was true of heart attacks. A disease unknown in America at the turn of the century became killer number one in less than 50 years. Millions upon millions of dollars have been spent in trying to curtail this new plague, and countless mistakes have been made. Environmental factors such as diet, a lack of exercise, smoking, and a host of others have been blamed, but all have struck out when careful examination of all known facts have been tabulated. The answer has come from some autopsies at Graz, Austria, where each hospitalized death receives an autopsy in compliance with a law passed over 200 years ago. This data was compiled by Barnes, Ratzenhofer, and Gisi in 1974 (*Journal of the American Geriatric Society*, Volume 22, page 176).

The causes of all deaths, which numbered about 2000 each year, were compared for 1930 and 1970. It was only the infectious deaths

that had decreased during this 40-year interval. Better medical care and the antibiotics caused a remarkable fall of 56 percent of deaths from infection. These deaths occur at an early age, and once deaths from these diseases are curtailed, a huge section of the population lives longer and must face the diseases occurring at a later age. More deaths were saved from tuberculosis than from any other single disease. Although all deaths at advanced age were somewhat increased by this New Population, the leader was heart attacks with a rise of 1000 percent, emphysema with a rise of 372 percent, and lung cancer with 300 percent. Was there something peculiar about those who had survived tuberculosis that might influence their susceptibility to diseases of an older age group? Yes, indeed. Since 1919 patients with tuberculosis have been known to have a weakness towards hardening of the arteries. Among the autopsy protocols the degree of atherosclerosis increased yearly, and by the time individuals approached 50 years of age, each was a prime candidate for a heart attack. The average age of death of the tubercular patient at Graz prior to antibiotics was 40 years. Since very few heart attacks occur before the age of 40, it is easy to understand the reason that heart attacks were scarce prior to antibiotics. The autopsies further showed that a low cholesterol diet, such as was consumed during World War II, did not prevent but actually was accompanied by a marked acceleration of hardening of the arteries. Emphysema was increased, apparently because the patient susceptible to but escaping death from infections lived longer, and repeated infections over the years caused more damage to the lungs. Lung cancer showed a sharp rise. Apparently those patients escaping death from tuberculosis were not susceptible to the other 149 cancers that older people may encounter. For some unknown reason, those dying from malignancies usually succumbed to lung cancer.

These autopsies leave little doubt that a New Population of adults arose as a result of antibiotics prolonging the lives of individuals susceptible to infections. Is it possible that this New Population is also more susceptible to hypoglycemia? The answer is, "yes," beyond a shadow of a doubt. Animals or humans susceptible to infectious diseases are often low in thyroid function. I have demonstrated that rabbits or rats deprived of their thyroid glands usually die from pneumonia at about half the normal life-span. Graz was a goiter area until 1963 at which time iodized salt was introduced. Hence, most of the

population was suffering from a relative hypothyroidism. Their incidence of deaths from infections, prior to the antibiotics, was far greater than that observed in the United States. Undoubtedly the antibiotics have caused a very marked increase in adult hypothyroidism in the U.S. just as they did in Graz. In the previous chapters it was pointed out that the hypothyroid patients are the ones likely to develop the syndrome of hypoglycemia. In a recent book, *Solved: The Riddle of Heart Attacks*, Robinson Press, Fort Collins, Colorado 80524, Barnes and Barnes point out that the tremendous rise in heart attacks in the 20th century has been due to survival of hypothyroid patients who formerly would have died at an early age from one of the infectious diseases. Today, as a result of the antibiotics, millions of the New Population are alive; however, nothing has been done to correct their hypothyroidism which was inherited from their ancestors.

One of the symptoms of hypothyroidism is hypoglycemia; hence there has been an explosion of the latter, similar to that of heart attacks. All of the cases of hypothyroidism will not display the symptoms of hypoglycemia at one time, but most of them can be expected to do so if sufficient stress is present.

Returning now to the "rhubarb," what is behind all the wrangling? The major problem has been unsatisfactory methods for the diagnosing of hypoglycemia. Modern medicine must be scientific; hence, there must be a test to prove each diagnosis. This is a commendable idea if a suitable test exists for the problem at hand. However, attempts have been made to diagnose hypoglycemia with the same test used for diabetes; yet hypoglycemia is the exact opposite of diabetes! The glucose tolerance test was devised to see how well an individual uses glucose. The diet must be changed to one of high carbohydrate for a few days before the test. Then a tremendous amount of glucose is given to the patient in a sickening sweet drink on an empty stomach. The blood sugar is followed for several hours to note how long the elevation in sugar persists. The urine is tested for any spillage over the kidney threshold. This quantity of glucose calls for a considerable amount of insulin to be secreted from the pancreas. As the blood glucose falls, some of the extra insulin is still present in the blood. In some individuals sufficient sugar to combat the extra insulin cannot be mobilized by the liver, and hypoglycemia with severe symptoms may appear some four to six hours after the glucose was ingested.

At that time I was seeing many hypothyroid patients, because my physiological background had been centered around thyroid-deficient animals. I found at once that the patients' symptoms and the response to thyroid therapy did not correlate with the PBI test. Hence, I never adopted it. Now, 40 years later I still see patients who had been taken off thyroid therapy as the result of a "normal" PBI test; their symptoms had promptly returned. No amount of insistence of the patients that thyroid therapy was needed moved the previous physicians who believed that the test could not be wrong. Yet after 40 years, the symptoms again disappear when thyroid is started. These patients have been labelled "anxiety reactions" and sent to the psychiatrist by those doctors who depend upon every test devised. During this 40-year interval the "anxiety reactions" have become so numerous that some physicians have stated that as much as 80 percent of their practice is made up of these patients.

Yet none of these purists of medicine took heed when the PBI test was finally declared worthless in 1967 by no smaller an institution than the Harvard Medical School. Through the years thousands upon thousands of pitiful patients had been denied appropriate therapy because of a faulty lab test. A rash of new thyroid function tests appeared in short order. None of them in my experience select the patient who has clearcut hypothyroid symptoms. Thyroid therapy leaves no doubt about the diagnosis. Have we arrived at a state of Super Scientific Endeavor in which the way a patient feels plays no role in his treatment, and he must modify his body himself to fit the errors of a machine? GOD FORBID!

There are still a few physicians of the old school who listen to the patients' complaints and attempt to relieve the suffering regardless of laboratory tests. Forty years ago the physician often used the therapeutic test of a small dose of thyroid if he was suspicious that the patient suffered from low thyroid function. This perhaps is the safest of our hormones in use today since, if a little too much is administered, the patient's gland compensates by reducing its production. This does not harm the gland, and its production can be increased at any time that more hormone is needed. In most cases the thyroid therapy *was* indicated in the therapeutic test, and the symptoms began to disappear in a couple of weeks.

Hypoglycemia faces an uphill fight in view of the opinion of many a physician who feels that it is only an "anxiety reaction," and the same physician is likely to feel that thyroid-deficiency symptoms are also a figment of the imagination.

One report is worthy of mention. Since the performance of an airline pilot must be flawless and since hypoglycemia interferes with proper mental activity, it should come as no surprise that the airlines are interested in hypoglycemia. A report by Harper and Kidera in *Aerospace Medicine*, Volume 44, page 769, 1973, is pertinent. Using the glucose tolerance test (although poor), they found that 25 percent of the airline pilots over 40 years of age suffered from hypoglycemia. Airline pilots are carefully selected from only physically able men, and if they run as high as 25 percent with hypoglycemia, the incidence in the general population with all their illnesses must run much higher. It would appear that those physicians with a fixed idea about the rareness of hypoglycemia should take a second look.



## CHAPTER 7

# The Diagnosis of Hypoglycemia

There is only one difficulty in the diagnosis of hypoglycemia — the physician must recognize that this syndrome is very common and it must always be considered in each individual patient. Since hypoglycemia usually accompanies hypothyroidism, and the latter is the most common disease entering the doctor's office and its diagnosis is the one most often missed, it is imperative that the symptoms of each disorder be kept in mind for each new patient. There are about 50 symptoms of hypoglycemia and each of these can be found among about 100 symptoms of hypothyroidism. If a layman is aware of the multitude of symptoms accompanying hypoglycemia, a more accurate diagnosis can be made by the patient than by most physicians using erroneous tests. No other syndrome has such diagnostic symptoms as uneasiness, weakness, hunger, sweating, fast pulse, and relief of all the symptoms in a few minutes after the ingestion of food.

In addition to the 50 symptoms seen in hypoglycemia, there are 50 more accompanying hypothyroidism. Only a few will be mentioned to illustrate the diverse locations and seriousness of the symptoms. Undue fatigue in spite of adequate rest applies to both hypothyroidism and hypoglycemia. Frequent headaches also apply to both. In the hypothyroid, growth may be retarded in the young, but accelerated after puberty. Susceptibility to infections in the respiratory system, the urinary system, or on the skin is common. Menstrual periods may begin early or late; they may be irregular or may be accompanied by severe cramps. Miscarriages may occur; babies may be stillborn, and crib-deaths may be due to a lack of vitality as a result of thyroid deficiency. Mental changes are common, either in children, adults, or in advanced age. A history of pneumonia, repeated urinary infections, rheumatic fever, or allergies may make one suspicious of hypothyroidism. Ulcers of the stomach, acne, psoriasis, eczema, and several other skin diseases are frequently associated with low thyroid function. *Since the thyroid affects the metabolism of every cell of the body, most*

*diseases may be aggravated by hypothyroidism.* Any of these symptoms deserve attention concerning hypoglycemia.

For years the 6-hour glucose tolerance test has been the last word in the diagnosis of this disorder. Yet the investigators on both sides of the Atlantic have realized that the test is a poor one. Modifications have been made leading to much more expense as a result of other blood tests run at the same time. It appears that attempts are being made to prove that hypoglycemia is not present rather than trying to demonstrate the syndrome. Professor Marks in England admits that the same patient at one time will show hypoglycemia with the glucose tolerance test while at another time the test may be normal. Hence he suggests that, in addition to the blood glucose determinations during the test, the blood also be checked for adrenal cortex secretion which is sometimes increased during low blood sugar levels. He furthermore requires brain tracings during the test since sometimes the brain waves change during hypoglycemia. After all of these time-consuming and expensive tests are run, he states that one should not make a diagnosis of hypoglycemia unless the patient has experienced the symptoms of hypoglycemia sometime in the past when the patient was on a regular diet! If the diagnosis is to be made from a history of a past episode of low blood sugar, why add the expense and inconvenience of laboratory tests if they are not going to be considered in the diagnosis?

The present American diagnostic methods are even more colossal than are the methods of the British. The University of California suggests that a case of suspected hypoglycemia go on a diet high in carbohydrate for three days. This gets all of the little enzymes activated that are concerned with glucose metabolism. Then the patient enters the hospital for three days. On the first day a detailed history and physical examination are carried out and base line studies for thyroid function, adrenal hormone, pituitary hormone and the daily urinary output of adrenal hormones are made. On the second day a 6-hour glucose tolerance test is carried out following the blood glucose levels, as well as the pituitary and adrenal levels, at periodic intervals. On the third day an intravenous glucose tolerance test is run, repeating the blood studies as before. From all the sophisticated and expensive tests, one is supposed to diagnose hypoglycemia if it is present. One diagnosis is sure to be made—the patient is financially embarrassed regardless of the results. This expenditure of time and money seems unjustified in

view of the fact that although the patient may have had a hypoglycemia at one time, if there was less stress prior to the hospitalization, the tests might prove negative anyway with a resultant waste of time and money.

After all, an intelligent patient can make the diagnosis of hypoglycemia with little degree of error from observation on himself. A physician, if he understands the syndrome, can be relatively sure of the diagnosis from the history. The present treatise indicates that most hypoglycemias occur in hypothyroid patients. The patients can bring to the doctor the Basal Temperature readings for two or three days. Basal Temperature is determined before arising in the morning after a good night's sleep by placing a thermometer (well shaken down the night before) in the armpit for 10 minutes by the clock. Men, young children, and women beyond the menopause can take their temperatures for two or three days at a time. Women subject to menstruation should check their Basal Temperatures only on the second and third days of the menstrual periods.

If the Basal Temperature is below 97.8 degrees Fahrenheit, one must be suspicious of a thyroid deficiency. A few rare conditions may also give a low reading. However, if the symptoms of hypothyroidism or hypoglycemia are present, thyroid therapy should be tried.

Until the present, the treatment for hypoglycemia has consisted of eliminating sweets and other carbohydrates from the diet, and increasing the amount of protein. One does not have to be a mental giant to carry out these procedures. Application of a little reading on the part of the patient will accomplish as much as can be obtained by seeing a series of doctors. It is amazing how much patients have taught physicians about hypoglycemia! If diet were the only treatment for the low blood sugar, the physicians' services might well be avoided. However, it now appears that a simple test for hypoglycemia may make a diagnosis consistently; the test and treatment require the services of a physician.

#### A SPECIFIC TEST FOR HYPOGLYCEMIA

Hypoglycemia results from the inability of the liver to furnish enough glucose to meet a given emergency. In 1934 my colleagues and I utilized a test to see how well animals with a pituitary or an adrenal

deficiency could meet the demand for glucose after intravenous insulin. Most investigators at that time felt that hypoglycemia was due to an excess of insulin in the blood. We found that small doses of insulin which would cause no effect in normal animals would produce severe hypoglycemia in the presence of either pituitary or adrenal deficiency. It is obvious that this procedure would identify animals with a tendency toward hypoglycemia.

The Insulin Tolerance Test was included in *Laboratory Aids in Endocrine Diagnosis* by Roberto F. Escamilla, M.D., Charles C. Thomas, Publisher, Springfield, Illinois, 1954. The test consists of drawing a fasting blood sugar, then injecting 0.1 units per kg. (2.2 pounds) body weight of regular insulin intravenously. Blood samples are then drawn for glucose at 20, 30, 45, 60, 90, and 120 minutes. Analysis of the blood in normal individuals reveals that the fasting level falls to about 50 percent of the initial reading in 20 to 30 minutes, but rebounds to the fasting level by 120 minutes. If the patient has hypoglycemia, symptoms are going to appear and glucose solution should be available for oral administration. In extreme cases intravenous glucose should be available and administered if necessary.

This test measures what one wants to know — can the individual meet emergencies of hypoglycemia? For all practical purposes the blood sugar determinations can be omitted. There will be no doubt that symptoms of hypoglycemia will appear if the subject is vulnerable, and one should not let the symptoms persist very long until glucose is administered. In a matter of one hour the test is over with no major expense. This simple, inexpensive test should satisfy the purists who must have a test for every diagnosis. It will probably move many alleged anxiety reactions to the category of hypoglycemia.

Personally, I feel that even the intravenous insulin tolerance test is superfluous. As pointed out in previous chapters, 95 percent of the hypoglycemias are of the reactive type, and those I have seen are suffering from hypothyroidism; the low blood sugar is only one of the many symptoms. To date, when the symptoms of hypothyroidism are relieved, hypoglycemia, like the others, disappears. The diagnosis of hypothyroidism can be made from a careful history and physical examination better than it can from laboratory procedures. I routinely use the Basal Temperature test to confirm suspicions of hypothyroidism. The thyroid therapy for these individuals makes many

friends and keeps a huge number of patients away from the over-crowded roles of "anxiety reactions." If the armpit temperature before arising in the morning is below 97.8 degree Fahrenheit and the patient has symptoms and physical findings of hypothyroidism including hypoglycemia, he will receive thyroid therapy in my office. Many of these patients have been refused thyroid therapy elsewhere because the blood tests were normal. The patients did not go to the doctor for an erroneous laboratory test, but they came with symptoms and they appreciate relief from their many complaints.

In summary, a diagnosis of hypoglycemia can be made without major expense in both time and money. Since hypoglycemia is only one of the many symptoms of hypothyroidism, attention should be focused on the cause of the low blood sugar. Relief of the cause is soon followed by cure.



## CHAPTER 8

# The Treatment of Hypoglycemia

The discoverer of hypoglycemia, Dr. Seale Harris, had a very effective, simple relief for the condition — small feedings at three-hour intervals. Indeed this would relieve the low blood sugar at the time, but it offered nothing in the way of a cure. It had some drawbacks. If the blood sugar fell during the night, one must get up to eat, or if crackers were taken to bed for middle of the night consumption, the crumbs would be uncomfortable before morning. Furthermore, unless the feedings were balanced closely, obesity would result from frequent feedings. One wonders if the "democratic coffee break" were not started by one with hypoglycemia; he would need his donut and coffee in mid-morning. At any rate, better therapy was needed, and that came from Professor Jerome Conn at the University of Michigan.

Conn suggested reducing the carbohydrate in the diet and emphasizing protein. This reduces the amount of free glucose in the blood which triggers more insulin secretion. Since 58 percent of protein can be converted slowly into glucose, the blood sugar is not elevated above the normal level. There is one rather serious objection to this approach. It takes energy to convert the protein to glucose, and it has been clearly established that a high protein diet lowers the metabolic rate; symptoms of hypothyroidism will be aggravated. Furthermore, weight is likely to increase unless the total calories are reduced.

Some personal observations on myself will illustrate the point. I was interested in seeing how many calories it would take to maintain a steady body weight, first on a relatively high content of protein as opposed to a high-fat intake. By using over 100 grams of protein daily with a small amount of carbohydrate and fat, my weight was steady for weeks on 2,000 calories daily. On the other hand using over 100 grams of fat daily and cutting the protein to 60 grams, my weight remained constant on 3,000 calories daily. It surely was fun eating the additional 1000 calories each day. This again emphasizes the advantage of a high-fat diet for reducing weight. More calories could be

consumed since more were burned up. This probably explains the experience on the high protein diet of Weight Watchers; loss is satisfactory at first, but after a few months a plateau is reached due to the lowered metabolism from so much protein. Hypoglycemia may be controlled on the high protein diet, but the other symptoms of thyroid deficiency which usually accompany hypoglycemia are aggravated.

Thyroid therapy for hypoglycemia does not require special diets; however, one must not overlook the tremendous contribution of Carlton Fredericks. Some readers may be unable to find a physician willing to use thyroid in this condition. The hierarchy of medicine uses only the blood tests for the diagnosis of thyroid deficiency. The shortcomings of blood tests are covered in the recent book, *Solved: The Riddle of Heart Attacks* by Barnes and Barnes, mentioned previously. Hence, at the present time those patients who cannot find a doctor willing to use thyroid will have to depend upon a diet. They are referred to the paperback, *Low Blood Sugar and You*, by Fredericks and Goodman, Grosset and Dunlap, New York, 1969. Fredericks is a nutritionist and one of the best; Goodman is a physician. Each of them treats hypoglycemia a little differently. Goodman advises 5 meals daily of lean meat, fish or poultry, low-carbohydrate vegetables, any of the fruits except bananas or candied fruits, skim milk but no coffee or carbonated drinks nor chocolate, and none of the starchy foods except bread.

Fredericks makes no restrictions on the fruits and suggests seven feedings daily. He goes into great detail about various concoctions that would delight the gourmet's palate. He allows more of the complex starches. Both authors markedly restrict fats by avoiding butter, by using skim milk and lean meat. For those interested in treating hypoglycemia by diet alone, the readers are referred to the above publication.

Before we leave diet, let an old physiologist throw in his two-bits worth. One of the adjuncts in treating hypoglycemia is to delay the arrival of an excess of sugar in the blood, thereby avoiding so much secretion of insulin. Years ago it was demonstrated that a fat meal empties out of the stomach more slowly than a carbohydrate meal. The farmer makes money from this principle by fattening his hogs on corn — pure starch. The pig, being smarter than the human, lies down and takes a nap when his belly is full. We attempt to work. In

one and one-half hours the pig's stomach is empty, the hunger contractions awaken him from a deep sleep, he promptly goes to the trough and eats again.

Forty years ago I became interested in obesity and had the opportunity to hospitalize and study obese patients over long periods of time. For three months they were instructed to eat just as they had done at home. A WPA dietician's only job was to prepare and weigh the foods selected. Without exception, the pachyderms were selecting protein and starch but avoiding fat like the plague. They were governed by the old adage that fat is fattening. None realized that the farmer had disproved this theory hundreds of years ago although physicians may never have heard about it.

When it was obvious that fat people were eating high carbohydrate foods and avoiding fats, their diets were changed. Using 50 grams of carbohydrate, 70 grams of protein and over 100 grams of fat each day, they could lose weight readily and comfortably. Only one case will be mentioned, an eighteen-year-old girl who weighed 295 lbs. She was placed on 1300 calories daily of the above diet and she found, to her chagrin, that on most days she could not eat all that she was allowed. Her weight loss was steady at 12 pounds each month for the ensuing 9 months. She went home on her diet, 110 pounds below her entrance weight, and fortunately she continued her diet. It took two years for her to reach 137 pounds, her optimum weight, but she had learned how to control her appetite.

Now for a few words to those who are screaming "You are killing people with a high animal fat diet." This subject is discussed in detail in the book by Barnes and Barnes mentioned earlier. If high animal-fat diets were dangerous, all the farmers would have died centuries ago and the rest of the population would have starved. Butter, cream, whole milk, fat meat, bacon, sausage, etc., maintained my ancestors for years, and there is yet to occur the first heart attack. All the evidence against fat is circumstantial. There is a much better explanation for heart attacks.

The high fat, low starch diet has been used for 40 years in the treatment of obesity, and no one in the group has suffered a heart attack. It was this type of diet that the diabetic had to depend upon before insulin was discovered. The slow emptying time of the stomach is a very good friend of the hypoglycemics, and I would recommend it

to those who cannot get thyroid therapy. One can avoid so many meals each day, and a slow source of energy arriving in the blood promotes a feeling of well-being. Eggs are encouraged, and it should be added that there is no evidence that cholesterol is implicated in heart attacks. In general, each meal should contain a liberal supply of protein and fat but a minimum of carbohydrate. Breakfast for me is composed of two eggs fried in bacon grease, 3 thin strips of bacon, a small orange and a glass of milk. One should drink whole milk although, if skim tastes better, a generous supply of cream on berries or on a small baked apple will make up for skimmed milk. The other two meals consist of a serving of fat meat, a low-starch vegetable with a thick pat of butter on it, and a salad with plenty of the old-fashioned salad dressings such as blue cheese, mayonnaise, thousand island or French dressings. Dessert can be any of the berries with cream on them, a small orange, a small apple, half a grapefruit with no sugar, or a small baked apple with heavy cream. If one is overweight, starchy foods are avoided. If the weight is normal, bread, potatoes, and other carbohydrates can be used. For a snack, cheese is excellent since it contains no carbohydrate and plenty of protein and fat. That does not apply to cottage cheese since it has very little fat.

The accompanying tables list the carbohydrate content of common foods.

#### PERCENTAGES OF CARBOHYDRATES IN VEGETABLES

##### 5% Vegetables

Asparagus	Kohlrabi
Bean sprouts	Lettuce
Broccoli	Okra
Cabbage	Olives — ripe
Cauliflower	Peppers
Celery	Pumpkin
Chard	Radishes
Chinese cabbage	Spinach
Cucumber	String beans
Eggplant	Summer squash
Endive	Tomatoes
Greens — beet	Turnips
Greens — mustard	Watercress

10% Vegetables

Beets	Olives — green
Brussel sprouts	Onions
Carrots	Rutabagas
Dandelion Greens	Winter squash
Leeks	

15% Vegetables (not much)

Artichokes	Parsnips
Oyster plant	Peas

20% Vegetables (avoid)

Beans:	Potatoes
Kidney, Lima, Navy	Hominy
Corn	

## PERCENTAGE OF CARBOHYDRATES IN FRUITS

5% Fruit

Avocado	Strawberries
Honey dew melon	Watermelon
Muskmelon	

10% Fruit

Blackberries	Peaches
Grapefruit	Tangerines
Oranges	

15% Fruit (sparingly)

Apples	Loganberries
Apricots	Mulberries
Blueberries	Pears
Cherries — sour	Pineapple
Grapes	Plums
Huckleberries	Raspberries

20% Fruit (avoid)

Bananas	Grape juice
Cherries — sweet	Prunes — fresh
Figs — fresh	

The reducing diet is excellent for hypoglycemia, and if one is underweight, the quantity of each food eaten is increased slightly. Diabetics will do better on such a diet and will require less insulin than with a liberal quantity of starch. (Incidentally, in a forthcoming book it will be pointed out that the hardening of the arteries in the diabetics is due to a lack of thyroid hormone, and 98 percent of them need thyroid therapy).

#### A NEW APPROACH TO TREATING HYPOGLYCEMIA

In the previous chapters evidence has been presented that most patients with hypoglycemia suffer from a thyroid deficiency which causes a sluggish liver incapable of meeting stress with sufficient glucose production. It seems reasonable that this hypothesis will stand the test of time since over the past 40 years treatment of hypothyroid symptoms alone in thousands of patients has not produced one case of hypoglycemia. This work began before hypoglycemia became fashionable, but since the syndrome has become more prevalent, cases presenting the syndrome of low blood sugar have responded uniformly to thyroid therapy without any dietary restrictions.

If the Basal Temperature, as described in the previous chapter, is low, and if the patient has symptoms of thyroid deficiency including hypoglycemia, thyroid therapy can safely be administered.

A baby needing thyroid will tolerate one-quarter grain of desiccated thyroid daily until the age of 3 or 4 at which time the child may need as much as one-half grain. Between ages 6 and 12 one grain may be necessary, and above that age, the dosage may be adjusted as for an adult. An adult may begin with one grain of thyroid daily and continue this dosage for a month. Then the Basal Temperature is repeated; if it is still low *and symptoms persist*, the dosage may be raised to 2 grains daily for the next month. More patients will require two grains than any other dosage, but some will need only one grain and a few require 3 or 4 grains. If symptoms are not relieved by 4 grains, one

should suspect that adrenal deficiency accompanies the hypothyroidism. Some patients will require 5 mgs of prednisone daily with the thyroid. Arthritis will usually need the combination.

If a patient has had a previous heart attack, the initial dosage should be one-half grain and the maximum dosage should not exceed 2 grains.

Results from thyroid therapy in hypoglycemia may be expected in about six weeks after the optimum thyroid dosage has been found. The patient would then be able to add foods that previously produced symptoms. In a short time, three regular meals daily containing sufficient fat should be tolerated without the syndrome of hypoglycemia appearing.



## CHAPTER 9

### Hypoglycemia — Alcohol — Auto Accidents — Crime

It has been demonstrated in previous chapters that millions of cases of hypoglycemia have arisen from natural consequences. The reduction in carnage of babies and young individuals by better medical care and antibiotics has given rise to a NEW POPULATION of adults suffering from hypothyroidism. In hypothyroidism the liver function is sluggish and not enough glucose is produced from protein to sustain the blood glucose during even mild degrees of stress. Recently it has become apparent that one of man's habits makes a substantial contribution to the pool of hypoglycemias.

For centuries man has attempted to temporarily escape the realities of life by drinking concoctions most of which are made by fermenting some form of carbohydrate. The result is ethyl alcohol. Alcohol is a mild anesthetic agent which removes the stress of the day's activities and temporarily puts the problems of the world on the back burner. In many households it is routine to have one or more drinks before dinner. No doubt the recipients become more relaxed and may better enjoy the meal. However, the individual's weight problem is also neglected; more food than necessary may be consumed. The alcohol is as detrimental to the human's weight as corn is to the hog's — leading to obesity. If one must drink, there is no better time than during a meal. Food, being absorbed, alleviates the drop in blood sugar; the alcohol curtails the production of glucose by the liver.

Only in recent years has it been realized that alcohol reduces the conversion of protein into glucose by the liver. This is clearly illustrated in young children. About the age of six seems to be the appropriate time for children to sample the contents of a whiskey bottle when no one else is around. The quantity consumed is unknown, but is probably small due to the undesirable taste. Somewhere between five hours and two days, the child may be found in convulsions or coma.

The youngster is rushed to the hospital; the heroic measures are started. It is now known that glucose should be given intravenously at once, followed later by an examination. Blood studies have revealed that the blood glucose is below the level for consciousness. If this low level has been present too long, the child may never regain consciousness and death may occur as long as 17 hours after having entered the hospital. In this instance, irreparable brain damage had occurred before the glucose was administered. Growth is a very potent stress in children, and it is not surprising that they are highly susceptible to hypoglycemia.

The clearcut experiment of A. R. Arky and his colleagues leaves no doubt that alcohol stops the liver from producing sugar from protein and from releasing it into the blood stream. (*Journal of the American Medical Association*, volume 206, 1968, page 575). Six healthy male volunteers served as subjects; each had two insulin tolerance tests. At the first test, three controls were started on an intravenous infusion of physiological salt solution, the other three were started on an infusion of alcohol that would simulate moderate drinking. After one hour each subject was given intravenously 0.1 units of insulin per kg (2.2 lbs) body weight. Blood samples for sugar were drawn 20, 40, 60, 80, 100, and 120 minutes later. In each subject at 20 minutes, the insulin had reduced the glucose level to about 50 percent of the starting value. In those having received salt solution the blood glucose then began to climb and reached the initial level after a period of two hours. Those subjects receiving alcohol had a marked lag in the return of the blood glucose to the normal level and were still below the starting point two hours after the insulin had been given. The experiment was repeated with those who had previously received alcohol now getting salt solution and those having had salt solution now receiving alcohol. The results of the two tests were identical. Those receiving alcohol had a marked delay in the recovery of their hypoglycemias. It is apparent that in a normal individual, alcohol will create a hypoglycemia.

The old adage that children and weak women should not drink has a firm physiological basis. Now we must add that both men and strong women with thyroid deficiencies should also curtail their use of alcohol. Their sluggish livers will not meet the demand for sugar, and serious hypoglycemia may result. Many of those with low thyroid function have already learned the hard way. In taking routine histories, it

is commonly found that many patients with low thyroid function are unable to drink alcoholic beverages because they feel ill at ease and have an unpleasant hangover. They would rather avoid the alcohol than suffer the consequences. When I correct their thyroid deficiency, I purposely neglect to tell them that they now can drink without the ill effects. Why start a new habit?

Diabetics are likewise highly susceptible to alcohol. About 98 percent of them suffer from hypothyroidism which would increase their susceptibility to alcohol. Their disturbance in sugar metabolism is accompanied by very little sugar stored in the liver. After a few drinks hypoglycemia develops and must be quickly relieved with intravenous glucose. If unconsciousness persists very long, damage to the brain is irreparable; such patients are likely to die a few days later without regaining consciousness. Again autopsies show no cause of death, yet the liver is free of any stored sugar.

### HYPOGLYCEMIA, ALCOHOL AND AUTO ACCIDENTS

The symptoms of hypoglycemia and alcohol intoxication are identical. It is more than coincidence that intravenous glucose will restore, in a matter of minutes, normal mental functions to either a child or an adult in alcoholic coma. Glucose is not an antidote for alcohol; it merely restores to normal the blood glucose as well as the glucose around the nerves in the brain. These facts raise some very important questions both physiological and legal. As illustrated above, alcohol may produce hypoglycemia in normal individuals. If the individual is subject to hypoglycemia in the first place, the alcohol will act like a double-edged sword, and the drop in blood sugar will be more extreme. More than 50 percent of our fatal auto accidents are associated with drinking drivers. It is necessary to know what the blood glucose is at the time of the accident. If the blood sugar is below the level compatible with consciousness, the driver is temporarily insane, and hence, cannot be held responsible for his actions.

The effect of alcohol in the blood on the mental and muscular reactions of the individual have been carefully studied. (*The Causes, Ecology and Prevention of Traffic Accidents*, H. J. Roberts, M.D., Charles C. Thomas, Publisher, 1971). Alcohol is a mild anesthetic and bolsters the individual's confidence. A person weighing 150 lbs. will begin to show brain impairment at a blood alcohol level of 0.035

percent which could be obtained with two 12-oz cans of beer or two ounces of whiskey. Critical judgment, visual perception and tolerance to glaring lights will be diminished. At blood levels of 0.05 percent alcohol, the reaction-time to changing lights is prolonged and muscular performance is impaired. This state of intoxication can be reached with three cans of beer or three ounces of whiskey. If the blood level reaches 0.06 percent of alcohol, the chances of such a driver having an auto accident are doubled. If the blood alcohol is found to be 0.1 percent the driver is classed as legally drunk and his chance of having a driving accident is six times greater than that of a normal individual. A concentration of 0.15 percent raises the danger of an auto accident to 25 times that of the normal. Any concentration above that is a signal for everyone else to get off the highway and leave the drunk to his own demise! It must be realized that some people may tolerate more alcohol than others. If the individual suffers from hypoglycemia, he may become intoxicated by merely smelling the cork on the bottle!

The new evidence indicates that the mental and physical deterioration of alcoholism may be due to a lack of glucose in the blood. If this is true, the highway patrolman should require a blood glucose as well as an alcohol determination on the blood. Hypoglycemia could cause a fatal accident at a time when the blood alcohol was within "safe limits." Such a driver is just as dangerous on the highway as the "dead drunk."

Some care must be used in the interpretation of the blood glucose level. If the driver was suffering from hypoglycemia and the alcohol was having very little effect, the excitement of the accident might trigger the release of adrenaline which in turn could elevate the blood sugar above the normal range. It is my personal opinion that each individual involved in a traffic accident should have the insulin tolerance test, described in chapter seven. Only by this procedure can cases of hypoglycemia be detected. One suffering from the hypoglycemic syndrome is a time-bomb driving down the highway. Such an individual should have a reduction in his carbohydrate intake and be treated with thyroid therapy. Relief from the tendency for hypoglycemia and the other symptoms of hypothyroidism will be appreciated by the individual. He is likely to continue his therapy, and it is unlikely that he will be involved in an auto accident as a result of hypoglycemia.

It has been known for years that some individuals are accident-prone, not only on the highway but in everyday living. They apparently make foolish judgments, such as stepping into a hole or failing to duck their heads for a low ceiling. To my knowledge no one has connected hypoglycemia to such individuals, but if the blood sugar should prove to be low, such a person would be a sitting duck for an accident. Since the majority of hypoglycemias are low in thyroid function, a Basal Temperature could be run by such a person without expense. If the Basal Temperature was found to be low, then he would be eligible for an intravenous "insulin tolerance test." Should this demonstrate a hypoglycemic syndrome, a change in diet and some thyroid therapy might alter his accident-proneness, and in case he did have a few drinks, he would be less likely to have an auto accident on the highway.

Heavy fines, suspension of driving licenses or even jail sentences have done little to curtail accidents from alcohol. Penalties do nothing to relieve an underlying illness, if it be present, while correction of a thyroid deficiency may alter a physical state of tiredness and produce a more pleasant and cooperative individual. I have never seen an alcoholic who was not low in thyroid function. Some of them spontaneously give up drinking when they feel better. Any regime that will improve their physical health is certain to aid with their mental problems. This seems to be a more physiological approach to their errors in traffic than penalties by the law. Glucose determinations on the blood of all participants of accidents, insulin tolerance tests on those who are low in blood sugar, and the use of thyroid should be a small price to pay for the carnage on the highway.

### HYPOGLYCEMIA AND CRIME

For many years one of the glands of internal secretion has been blamed occasionally for criminal behavior of some individuals. There has not been a close link between the two, and chance could have explained the occurrences. Now that hypoglycemia is better understood and is apparently due to a thyroid deficiency, there is a good reason to suspect this endocrine disorder may often be responsible for an otherwise stable individual going berserk. It has been known for a century that hypothyroid patients might become insane and have hallucinations. They see things or hear voices that aren't there. A few years ago,

one of my patients had such an experience. She was a 13-year-old girl who was cleaning motel rooms after school to help the strained finances of the family. The walls in the rooms began to talk to her with resultant consternation. In my office the examination clearly indicated that her thyroid function was low. She was placed on thyroid therapy, and in a short time she no longer heard the talking-walls. The voices returned, however, when she ran out of medication. Resumption of therapy again abolished the hallucinations.

Unidentified flying objects have attracted much attention in recent years. They are usually seen by only one individual at the time and they usually occur at night. Are some of them due to hypoglycemia and hallucinations? It seems peculiar that flying objects should avoid crowds. The law of chance should bring some of them into inhabited areas. Why are they more often seen after dark? Surely if they are for observation of our country, more could be seen in daylight. Why have our radar screens failed to pick up a strange object in the sky? It is well established that hypoglycemia progressively increases as the interval from the last food lengthens. Hence, the blood sugar is lower in the night than during the day. You may rest assured that if one of my patients reports an unidentified flying object, I shall run an intravenous insulin test and see if the individual has a hypoglycemic syndrome. This appears to be a more rational approach to the solution than an attempt to catch the flying object.

Likewise, some people hear voices in the middle of the night. A "Supreme Being" may order them to do something. If the "order" is to kill, a murder may be committed. It is in the middle of the night that hypoglycemia may be more severe. There is no doubt that a parent who suddenly murders a child is insane at the time. There is no known insanity that can appear so suddenly and disappear so quickly as hypoglycemia. The horrendous murders without cause or reason must be due to insanity. When they are caused by individuals who have been normal up to that time, there is reason to suspect hypoglycemia as being one factor. You will recall in chapter one that a very normal college student became belligerent 30 minutes after a dose of insulin, had to be restrained and injected with glucose for relief. He was sure that his colleagues were trying to poison him with the orange juice.

The best way to cure crime is to prevent it. The time to start is in childhood. The psychiatrist examining a criminal immediately goes to

the childhood history. Usually there is early evidence of abnormal behavior. I have seen my share of behavioral problems during the past 40 years. Almost without exception, the unruly preschool child has been low in thyroid function. Insulin tolerance tests have not been run for hypoglycemia, but the Basal Temperatures have been low. Thyroid therapy has converted the maverick into a cooperative member of the family. If he is of school age the teacher soon notices the difference and brags to the parents how the youngster has turned around. These children are tired, and fatigue irritates the nerves in any age group. In school their attention span is short and their retention poor. Untreated, they soon become depressed, unwanted and seek the wrong company. I have no doubt that most of them would show hypoglycemia if tested, but why waste the time and effort. If the temperature is low and thyroid therapy will correct it, mission accomplished.

It is my opinion that juvenile or adult offenders of all categories should be carefully screened for hypothyroidism and hypoglycemia. If their physical abnormalities were corrected, the time of incarceration might be materially shortened and recurrences should be markedly reduced. There is a reasonable chance that first offenders might adjust to society and hardened criminals prevented.

#### HYPOGLYCEMIA AND ALCOHOLISM

Some writers have felt that hypoglycemia may be the cause of chronic alcoholism. This seems unlikely since, as was seen above, alcohol depresses the manufacture of glucose from protein; hence, at once the hypoglycemia would be worse and coma would soon end the drinking bout. Herein we find a logical explanation for the craving for alcohol. The individual is tired, weary, demoralized and at the end of his rope. The anesthetic properties of alcohol dull his senses and give him some relief. Hypoglycemia may be present and, if not, it will be when sufficient alcohol has been consumed. However, it seems more plausible that the hypoglycemia was one of the symptoms of hypothyroidism which led to the drinking habit. Correction of the hypothyroidism has been quite beneficial in kicking the habit. Thyroid therapy has caused some alcoholics to quit drinking without any coercion on the part of others. Those who really want to quit usually can when the thyroid is straightened out. Surely thyroid therapy has a place as an adjunct in rehabilitation programs.

## HYPOGLYCEMIA AND OTHER DISEASES

Some writers have accused hypoglycemia of causing many other diseases. Marks in England has stated that some authors believe that any disease without a known cause may be due to hypoglycemia. Now it appears that hypoglycemia is only one of the many symptoms of hypothyroidism; it is obvious where the mistakes have been made. Rheumatic fever has always occurred in hypothyroid patients in my experience. The resistance to infectious diseases is low in the thyroid-deficient patient. Repeated throat infections are followed by allergic reactions on the valves of the heart, and rheumatic fever may develop. Hypoglycemia may be present, but it is due to the hypothyroidism as is the rheumatic fever. Both the rheumatic fever and the hypoglycemia are fellow travelers in hypothyroidism; one does not cause the other.

Likewise, asthma is alleged to be due to hypoglycemia. Evidence is rapidly accumulating that the asthmatic person suffers from thyroid deficiency as well as a mild adrenal deficiency. A deficiency of either of these hormones will interfere with the manufacture of glucose in the liver. One should expect that hypoglycemia may be present, but this does not mean that the hypoglycemia causes the asthma; it is only an innocent bystander. Each of the diseases alleged to have been caused by hypoglycemia is really a part of the hypothyroidism. The latter is the dog that wags the tail, hypoglycemia; we have no evidence that the tail wags the dog.

In summary, present evidence indicates that hypoglycemia is only one of the many symptoms of hypothyroidism. The latter is the most common disease entering the doctor's office. Hence, since any case of hypothyroidism may exhibit hypoglycemia under sufficient stress, there are millions of both disorders around. Many physicians deny that either syndrome is prevalent because they are depending upon inaccurate blood tests for thyroid function and the unreliable glucose tolerance test for hypoglycemia. The Protein Bound Iodine was the first blood test recommended for thyroid function. Selenkow (*Journal American Medical Association*, volume 202, page 153, 1967) points out that so many other factors affect the test that it alone is worthless for estimating thyroid function. Yet for 35 years this was the only test used by specialists as well as physicians at large. As a result, patients with a previous diagnosis of hypothyroidism were taken off thyroid therapy because the test indicated they did not need medication. The

original symptoms returned, and physicians refused to restore therapy. There began a deluge of diagnoses of mental disturbances not infrequently called "anxiety reactions." Yet the patients, who were relieved of their symptoms by thyroid therapy and whose symptoms returned when the treatment was stopped, know that their primary diagnosis remains thyroid deficiency. Similar patients with the same basic symptoms know from their reading that they, too, are lacking in thyroid hormone. Many of them have been to two or more well known clinics where their tests were all negative. It has been suggested that they see a psychiatrist. Some have done so, again with disappointment. Some began at once to seek a physician with more experience in the use of thyroid. Such patients have come to my office from each of the 50 states, from Mexico, and from Canada. Invariably the patients were right, and thyroid therapy relieved their symptoms of hypoglycemia, hypothyroidism, or both. They go home relieved with the knowledge that their complaints were not mental in origin. For years their relatives and friends have traveled the long distance because of the success on some of their acquaintances. Clearly the results are permanent, and not due to temporary enthusiasm or the relief would be short-lived. Many physicians are insulted if patients make suggestions about the cause of their illnesses. The sooner the clinicians realize that the patients may know more about their symptoms of thyroid deficiency than most physicians do, the sooner the tremendous number of mental cases will decrease and some of the loss of public esteem for physicians will be restored.



## CHAPTER 10

# Recapitulation

Since the present thesis departs from many of the current ideas about hypoglycemia, it appears appropriate to collect some of the pertinent facts at this time. Chapter one signifies that in normal individuals appropriate administration of insulin can produce hypoglycemia, resulting in behavior which may become hostile. As early as 1930, animal experiments clearly indicated that a pituitary or an adrenal deficiency might also produce hypoglycemia and even convulsions under fasting or as a result of minute doses of insulin.

Chapter two details the first report of hypoglycemia occurring spontaneously without any insulin administration in an otherwise normal person. Food of any kind soon restored the individual to normal, and it was assumed that too much insulin was being secreted by the pancreas. If blood sugars were drawn before breakfast, from apparently normal individuals, about 6 percent were found with values below the normal level, and many of them had some of the symptoms of hypoglycemia. Abrahamson found that several serious diseases such as allergies, rheumatic fever, ulcers, asthma, etc. might display hypoglycemia, and he felt that too much insulin in the blood might have been the cause of these illnesses. Later work has clearly shown that hypoglycemia was not the cause, but a fellow traveler in these diseases.

Chapter three refers to the fact that glucose is the only food utilized by the brain and that it is just as important as oxygen for normal mental processes and behavior. After each meal some of the carbohydrate is stored in the liver to tide the individual over until the next meal. However, since not more than an eight-hour supply can be stored, another source of glucose is necessary and that proves to be the manufacture of glucose from protein by the liver. It is apparent that the liver is the most important organ for the maintenance of a normal blood sugar.

Chapter four points out that any disease of the liver such as hepatitis (liver infection), poisons for the liver cells, tumors both benign or malignant of the liver, metastatic cancers from other tissues lodging in the liver, or even too much alcohol destroying the liver cells may cause

severe hypoglycemia. These causes are rare and do not begin to account for all of the cases of hypoglycemia that occur. Finally, a tumor of the beta cells in the pancreas that produce insulin, may develop occasionally, and this disease of hyperinsulinism is cured only by surgical removal of the tumor.

Chapter five points out that hormones from the pituitary, from the adrenals, and from the thyroid markedly affect the manufacture of glucose by the liver. If any of these three hormones are in short supply, hypoglycemia is likely to appear. Not many cases of adrenal or pituitary deficiencies are encountered, but hypothyroidism is one of the most prevalent diseases and has markedly increased since the advent of antibiotics. Susceptibility to infectious diseases is a trademark of thyroid deficiencies. Prolonging the lives of such individuals by antibiotics and better medical care has boosted the incidence of thyroid deficiencies like mushrooms after a rain. Thyroid administration to such patients soon relieves most of the symptoms of hypothyroidism including hypoglycemia.

Chapter six discusses the big argument over the incidence of hypoglycemia. The specialists who must confirm each diagnosis with a laboratory test have been handicapped since, until recently, we have not had a satisfactory test for hypoglycemia. They have used the old glucose tolerance test for diabetes which, for hypoglycemia, is wrong about as often as it is right. Diabetes is the exact opposite of hypoglycemia. Those patients with the symptoms of hypoglycemia but whose glucose tolerance tests are negative, are labeled "anxiety reactions," and are promptly shipped out to the psychiatrists. On the other hand, equally capable physicians who listen to the patient's symptoms and their reactions to the customary low-starch, high-protein diets have become heroes to a multitude of happy patients. Personal experience indicates that the latter group is correct and that there are indeed many, many cases of hypoglycemia being missed by some of our best physicians because of a faulty lab test.

Chapter seven deals with the diagnosis of hypoglycemia. Symptoms must play a major role in the diagnosis of any disease. The patients go to their doctors because they have a series of complaints. With the high price for medical care, they are not likely to manufacture illnesses. Of the 50 odd symptoms of hypoglycemia, it is found that each and every one of them is one of the approximately 100 symptoms

of hypothyroidism. This would give a high probability that hypoglycemia is only one of the symptoms of low thyroid function. The unreliability of the glucose tolerance test is clearly emphasized both in Europe and America by the many additional tests that have been added to the glucose tolerance test in an attempt to make a diagnosis. The Insulin Tolerance Test seems to be more specific for hypoglycemia. This was used by the author on animals over 40 years ago and has been applied to the human by other clinicians. It is shorter, simpler and cheaper than the unreliable glucose tolerance test. However, this test is also redundant since a firm diagnosis can be made from the history, the physical examination and the Basal Temperature test for thyroid deficiency.

Chapter eight details treatment. The multiple feeding and high-protein low-carbohydrate diet used in the past can still be used as an adjunct, but even these are unnecessary if the individual is placed on thyroid therapy. In about six weeks after the proper thyroid dosage is established, most patients can eat only three meals each day and include many of the foods which had been avoided in the past. Additional help can be obtained by incorporating a generous amount of animal fat in the diet since this delays the emptying time of the stomach and prevents such a dramatic rise in blood sugar. Furthermore, the obese can reduce more readily since they are comfortable on fewer calories. The phobia against animal fats for fear of heart attacks is pure "hog-wash."

Chapter nine discusses the influence of hypoglycemia on auto accidents, behavior, and crime. Alcohol will aggravate hypoglycemia even in a normal individual because it stops the liver from forming and discharging glucose. Most of our accidents associated with drinking may be due as much to hypoglycemia as to the alcohol content of the blood. Traffic officers should run a blood glucose along with the blood level of alcohol. There may be little hope of persuading an individual to stop drinking, but if his thyroid function is normal, his performance is better if he does drink. Hallucinations are common in hypothyroidism; they may be due to an associated hypoglycemia. Some of our violent crimes might possibly result from low blood sugars. Cases have been recorded in which a Superior Being had ordered them to commit their deeds. Certainly hallucinations could make a better explanation than a voice suddenly appearing "out of the blue." Again

hallucinations might explain the sighting of some unidentified flying objects.

From the many experiments at hand, there seems little doubt that hypoglycemic cases have been rapidly mounting during the 20th century. The laymen are thoroughly convinced, and many capable physicians are likewise aware of the facts. Now that we have a specific accurate test for hypoglycemia, we can expect that the purists of medicine can not escape the truth and "anxiety reactions" may dwindle in numbers.

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*"The Nobel Prize Committee missed the boat by not recognizing the genius and medical contributions of Broda O. Barnes, M.D. - my teacher."*

**Stephen E. Langer, M.D.**  
**Author of Solved: The Riddle of Illness**  
**Berkeley, California**

*"I have been acquainted with the work of Dr. Broda O. Barnes for a number of years, and have been actively affiliated with the Broda O. Barnes, M.D. Research Foundation, Inc.*

*I am impressed with his foresight in reporting on the importance of thyroid dysfunction which the internists, family physicians and gynecologists often overlook.*

*I have found it particularly helpful in my own practice in Neuro-psychiatry, where I often encounter hypoglycemia, anemia, multiple addictions, eating disorders, malnutrition and numerous deficits in conjunction with thyroid dysfunction.*

*The use of desiccated thyroid as part of the overall treatment regime has been most helpful.*

*Following his recent death, I am glad that the work of Dr. Barnes will be continued by health professionals and the general public who believe in it.*

*I am one of them!"*

**Harry K. Panjwani, M.D., Ph.D.**  
**Neuro-psychiatrist/Nutrition/**  
**Behavioral Medicine**

*"Such a common problem that is overlooked so frequently and corrected so easily and effectively in such a great variety of human ailments.*

*I can't imagine anyone practicing medicine without knowledge of Dr. Barnes' work."*

**S. Yurkovsky, M.D.**  
**Cardiologist/Member Barnes Foundation**  
**Westbury, NY**

ISBN 0-913730-26-2

