HOW TO PREVENT HEART ATTACKS

LEE FOUNDATION FOR NUTRITIONAL RESEARCH
MILWAUKEE 1, WISCONSIN

By

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INTRODUCTION

Pain in the chest, also called precordial pain, angina pectoris, due to embarrassed heart action is a common complaint. It may occur at any age: in youngsters playing vigorously in games, as well as in the very old. Most of the victims are above 40 years of age. People who have this symptom which is a signal that the heart is not functioning normally, usually have recurrent attacks of the chest pain. As time goes on, these attacks may become more severe, last longer, and recur more often. At first they occur with slight or moderate exertion, and then they occur even at rest, sometimes waking the victims from sleep. Then, one day, they suffer the "Heart Attack" which may be fatal or non-fatal. The mechanism which had been causing the recurring attacks of pain over the months or years is the same one which caused the final heart attack. The recurrent attacks of pain were not accompanied by any permanent damage to the heart of a serious nature, although it is possible for some of the attacks to leave small scars. On the day when the "Heart Attack" occurred, the causal mechanism was so severe that it produced permanent damage to the heart muscle. And, if the damage to the muscle is extensive enough, death may ensue. If it is moderate, the victim recovers with a damaged heart.

In this monograph I shall describe the causal mechanism responsible for the recurrent attacks of heart pain and I shall describe the treatment which has proven successful in the prevention of the attacks of chest pain. And, if one can prevent the attacks of chest pain, one can also prevent the final blow — the "Heart Attack."

At the present time most authors and investigators have been focusing attention on the condition of the coronary arteries which supply the heart muscle with the necessary nutrients present in the blood. These investi-

gators have stressed the anatomical state of the arteries caused by arteriosclerosis. Practically all writers on the subject believe that the poor circulation resulting from the narrowed sclerotic coronary arteries causes an "ischemia" (reduced blood flow) of the heart muscle which they believe is the cause of the pain and the final heart attack. This concept leaves so much unexplained, and there is so much contradictory data, that it has created much confusion, disagreement, and difficulty in understanding the disease.

The concept which will be presented in this monograph regarding the cause of this form of heart disease is new, and, if I may be permitted to say so, it simplifies and clarifies the problem, helping to bring order out of chaos. The treatment devised for the prevention of the attacks of pain and the final heart attack is based on the causal mechanism and hence is a logical, and not empirical, form of therapy. It can prevent the attacks of pain in the young as well as in the old. The best proof that the concept is correct is this: the therapy is successful.

The reason for writing this monograph is obvious. Our country is faced with a mortal enemy that is becoming more menacing with time. In October, 1941, I published an article in the Medical Annals of the District of Columbia, titled: "The Control of the Anginal Syndrome with a Low Carbohydrate Diet," in which I presented evidence to show that the consumption of certain foods was the fundamental cause of recurrent heart pain and the eventual heart attack. I recommended a diet aimed to prevent as well as to treat the disease. This article apparently has made little impression. It was commented upon by a few individuals who abstract such medical information for the medical profession and the public. According to the yearly statistics put out by the U. S. Public Health Service, it is apparent that the ideas suggested in that article for the control of an important disease have not reached the average American. All of the clinical and experimental data which I shall present in this monograph has been published by me in several articles and in a tract on polio. The following list of references is for those readers who may wish to refer to the technical papers published by me.

Publications by Dr. Benjamin P. Sandler:

1. The pain mechanism in peptic ulcer and pseudoulcer syndrome. The control of these disorders with a low carbohydrate diet. The Review of Gastroenterology. Nov.-Dec., 1940, Vol. 7, No. 6.
2. Mucous and ulcerative colitis. The Review of Gastroenterology. March-April, 1941. Vol. 8, No. 2.
3. Production of neuronal injury and necrosis with the virus of poliomyelitis in rabbits during insulin hypoglycemia. American Journal of Pathology. January, 1941. Vol. 17, No. 69.
4. Chronic abdominal pain due to hypoglycemia. With a note on the pathogenesis of neurotic symptomatology. Surgery, March, 1941. Vol. 9.
5. The control of the anginal syndrome with a low carbohydrate diet. Medical Annals of the District of Columbia. October, 1941. Vol. X, No. 10.
6. Treatment of tuberculosis with a low carbohydrate diet. American Review of Tuberculosis. September, 1942. Vol. XLVI, No. 3.
7. Treatment of tuberculosis with a low carbohydrate high protein diet. Diseases of the Chest. April, 1950. Vol. XVII, No. 4.
8. Diet Prevents Polio. Published by the LEE FOUNDATION for NUTRITIONAL RESEARCH, 1951.

Chapter I

THE INCIDENCE OF HEART DISEASE AND SUGAR CONSUMPTION

Heart Disease today is the leading cause of death in the United States and in those countries having similar dietary, cultural, and living standards. Heart disease is causing more deaths and more concern than any other disease because its incidence has risen rapidly since 1900 in those countries that are generally regarded as exponents of Western Civilization, i.e., countries which have made the greatest advances in all aspects of material existence. And, what is most disconcerting, heart disease is a minor cause of disability and death in all the so-called primitive countries where, by comparison, the inhabitants have made little progress materially and have experienced little advance in living standards.

Aware of the marked difference in the incidence of heart disease in Western countries and in the backward countries, several studies comparing the diets of the two groups have been made in the hope of discovering a dietary component that could be responsible for the greater incidence in the Western group. Up till now, the greater fat content of the Western diet has been implicated as the causal factor and some researchers have advocated a significant change in the Western diet aiming to reduce the total amount of fat in the diet and to change the type of fat in the diet. There has been much disagreement with this theory and considerable controversy is going on.

I do not subscribe to the theory that the fat content of the diet is responsible for this difference in incidence of heart disease. I have evidence to implicate the carbohydrate content of the diet, especially the ingestion of refined sugar and starch. I have found that the most striking feature of the primitive peoples' diet is the practically complete absence of refined foods, especially sugar. The

consumption of refined sugar by the Western countries, by contrast, is appalling. Refined sugar and starch is a luxury in primitive countries and the general population is just too poor to buy them.

I have pointed out this great difference in sugar consumption in my book, "Diet Prevents Polio." In that book I presented evidence to show that susceptibility to polio was linked to the ingestion of sugar and starch. I also stressed two facts: (1) that the incidence of polio was highest in the Western countries with their advanced sanitation, high living standards, and high per capita sugar consumption; and (2) that the incidence of polio was extremely low or nil in countries that had the lowest per capita sugar consumption. For example, a country like China, with per capita sugar consumption of about 3 pounds per year, has never had a polio epidemic; whereas the United States, with per capita sugar consumption of about 100 pounds per year, has been visited by many epidemics, especially since 1900.

In an article in the June 28, 1947 issue of the Journal of the American Medical Association, Dr. A. B. Sabin, a leading investigator and authority on polio, discusses certain problems which have baffled students of the disease. I shall state some of the problems he mentions and shall attempt to throw some light on their solution.

1. Dr. Sabin states: "No circumstance in the history of poliomyelitis is so baffling as its change during the past 50 years from a sporadic to an epidemic disease."

An increasing consumption of sugar, as shown in the following table, helps explain this change in the incidence of the disease.

TABLE NO. 1

The Consumption of Sugar in the United States

Years	Pounds per Capita, Yearly Average
1880-1890	44
1890-1900	56
1900-1910	65
1910-1920	82
1920-1930	100
1930-1937	95
1939	103.2
1955	95.4

2. Dr. Sabin writes: "Another peculiar circumstance which may contain an important clue is that epidemics have occurred with the greatest frequency and severity in the very countries in which sanitation and hygiene have undoubtedly made the greatest advances."

I offer the following explanation: Advanced countries are wealthier countries and advanced sanitation and hygiene are, as a rule, to be found in wealthier countries. Advanced sanitation and hygiene have helped prevent such diseases as typhoid fever, cholera, malaria, and tuberculosis, because the environment has been controlled by purification of water and milk, by drainage of swamps, by proper sewage disposal, and the organisms responsible for these diseases have been "kept away from our doors." The fact that polio has not been prevented by advanced sanitation and hygiene indicates that its incidence is controlled and influenced by factors quite different from the factors that bring about the spread of typhoid and other diseases. As previously stated, advanced sanitation and hygiene are to be found in the richer countries, and one of the unfortunate evils that accompanies wealth is the consumption of sugar in large quantities in the form of luxury foods, such as ice cream, candies, soft drinks, cakes, pies, pastries, and the like. Poor countries cannot afford these luxury foods, sanitation, and hygiene. That is how I would explain the greater incidence of polio in countries with advanced sanitation and hygiene. The table No. 2 shows the extreme differences in sugar consumption in various parts of the world and it will be readily noted that the countries with the lowest sugar consumption are the most backward in sanitation and hygiene. From the table we readily note that sugar consumption is by far the greatest in the richer countries where one would also expect to find advanced sanitation and hygiene. Epidemics of polio have occurred with greatest frequency and severity in the high sugar consuming countries.

The following is a quotation from an article entitled

TABLE NO. 2
Sugar Consumption by Geographic Divisions
(Crop year Sept. 1, 1938 to Aug. 31, 1939)

Country	Per Capita Consumption (Pounds — Raw Value)
United States	103.2
Canada	102.9
Mexico	37.1
Other North America	41.2
Argentina	71.0
Brazil	51.7
Other South America	28.8
Sweden	119.3
Holland	89.4
Switzerland	85.1
United Kingdom	112.6
Germany	63.3
Italy	21.3
Poland	29.8
Rumania	14.6
Spain	17.2
China	3.2
India	24.3
Japanese Empire	29.1
Java (Dutch East Indies)	11.6
Other Asia	13.0
Algeria, Morocco, and Tunisia	44.4
British South Africa Union	58.6
Egypt	20.4
Other Africa	3.7
Australia	114.5
Other Oceania	81.0

"The Community Problem in Coronary Heart Disease. A Challenge for Epidemiological Research," which appeared in the September, 1956 issue of the American Journal of the Medical Sciences, and was written by D. C. Miller, F. J. Stare, and Paul D. White. Dr. White is well known as the heart consultant to President Eisenhower. "To realize that the first cause of death in the United States is a disease little known 50 years ago comes as something of a surprise to physicians and public alike. . . . No disease has ever come so quickly from obscurity to the place coronary heart disease now occupies, to maintain itself there with a permanence presumably to endure in this country for years to come."

These two statements regarding the greatly increased

incidence of two important diseases are most striking. It is more than a coincidence that the increased incidence of both diseases since 1900 may be directly correlated with an increase in the consumption of sugar since 1900. Added to these important clues as to the epidemiology of the diseases, is the clinical mass of information contained in this book to support the contention that the ingestion of refined sugar and starch is a fundamental causal factor in the prevalence of both polio and heart disease.

Even after reading all the information contained in this book, there will be those who will say, "The evidence offered in this book is not 100% fool-proof. The book does not answer all the questions. The author has not proved his point."

It may take many years to collect all the needed data to prove beyond doubt that the ingestion of refined sugar and starch is the fundamental causal factor in the epidemiology of heart disease. However, there is now in our hands sufficient evidence to state that these dietary components are direct causal factors in the development of heart disease. The evidence is sufficient to plan a form of therapy and prevention. And the proof of the pudding is in the eating. The diet which has been devised to afford relief from attacks of heart pain and to prevent their occurrence has been successful. This is the best evidence of all.

Besides describing the diet that has proven successful in the treatment of what is now called "coronary heart disease" this monograph will attempt to clarify certain aspects of this disease. Because of many gaps in our knowledge there is much confusion and lack of agreement on various aspects of the disease. All leading writers on the subject have had occasion to refer to the existence of confusion. For example, the term coronary heart disease is used by many implying that the disease is due primarily to anatomical changes in the coronary arteries (coronary arteriosclerosis). There is no unanimous agreement on this.

Dr. Paul D. White, a leading authority, has written in his textbook "Heart Disease": "atherosclerosis of the coronary arteries has been placed definitely as an integral part of the disease; of itself, however, it does not account for the complete clinical and pathological event." (1)

A large mass of data has been accumulated by investigators in heart disease and the following facts are enumerated with the purpose of showing that coronary arteriosclerosis is not the fundamental causal factor.

1. The commonest lesion of the coronary arteries is arteriosclerosis and eventually all hearts will be affected. Yet, only a small percentage of individuals with coronary arteriosclerosis get recurrent attacks of chest pain or fall victims to a heart attack.
2. Extensive coronary arteriosclerosis may be found at autopsy in individuals who never suffered from heart disease and with no evidence of a deficient coronary circulation in the heart muscle.
3. Myocardial infarction can occur with coronary thrombosis or occlusion.
4. Myocardial infarction can occur without coronary thrombosis or occlusion.
5. Coronary thrombosis or occlusion can occur without myocardial infarction.
6. Complete obstruction of the mouths of the coronary arteries due to syphilis or arteriosclerosis of the aorta may occur without evidence of embarrassed heart action during life.
7. There are individuals with angina pectoris whose hearts show normal coronary arteries at autopsy.

A perusal of the above facts indicates that there are many gaps in our knowledge and it is small wonder that there is much confusion in the literature. Of course, such a state of affairs is always present when not enough is known about any problem in medicine, law, physics, or other branches of knowledge.

Certainly, the accumulated data makes it difficult to

accept the idea that an obstructed coronary circulation, partial or complete, is the fundamental cause of angina pectoris and the final heart attack. Such a concept leaves too many questions unanswered and many questions are answered unsatisfactorily.

Chapter II

THE HEART ATTACK, ARTERIOSCLEROSIS, AND DIETARY FAT

Heart disease is the leading cause of death among adults in the United States today. Its great incidence suggests that it is caused chiefly by a factor to which the entire population is exposed; a noxious factor that is widespread; a hidden, widely prevalent danger working to harm the individual in a vital area — the heart. Such a causal factor, in order to adversely affect the general population must be able to reach its victims by an easy and subtle method, such as via the general diet. Evidence will be presented to indicate that the causative factor is present in the average daily diet of the American people.

This book deals with a common form of heart disease, namely the "Heart Attack." This affliction is sudden and dramatic in its onset, sometimes fatal, more often non-fatal. It is followed by practically complete recovery in some non-fatal cases and partial invalidism in others. All of us are familiar with the term "Heart Attack." All of us know of a friend, or a relative, who has suffered a heart attack. Each day one reads or hears of a prominent citizen, of either sex, more often of the male sex, dying of a sudden attack. The victim may be a young man of 35, or a middle aged man of 50, or an elderly man of 75. Very often the victim had had no previous signs or symptoms of heart trouble and was struck down without warning in the first and only attack. It is a common occurrence for an individual to have been examined routinely by his private physician, or to have been examined for insurance purposes by a specialist using the electrocardiograph and other apparatus, and to have been declared of sound health, only to suffer a heart attack a few days or few weeks later. This is not due to any error on the part of the examining physician. It just indicates that medical science at present

has no means of predicting whether or not an individual will suffer a heart attack, fatal or non-fatal, in the near or distant future.

The victim of a heart attack suffers a serious illness, totally incapacitating for a varying period of time, usually several weeks, from which he may recover and return to practically his former level of physical and mental activity, or from which he may enter a prolonged period of chronic heart disease and be disabled to a varying degree, depending on the amount of damage to the heart muscle. Or, the victim may go on to die after several days, weeks, or months, from progressive heart failure or from another heart attack.

Heart attacks may occur at any age after 20 years. The fundamental cause is not due to the aging process, obviously, although the incidence of the disease increases with age. The average age at death is 58 in males, and 56 in females. The presence of high blood pressure seems to favor the occurrence of attacks. The disease is more serious, occurs at an earlier age, and is more common in diabetics. This latter fact, namely, that diabetes favors the incidence and severity of the disease, affords a definite clue that the deranged carbohydrate (sugar and starch) metabolism of the diabetic is a fundamental causative factor. And, since the main constituents in the diet that adversely affect the diabetic are sugar and starch, it suggests that these same dietary components could also be the causative agents in the non-diabetic individual. Evidence will be presented to show that the ingestion of sugar and starch is the fundamental cause of the heart attack in the non-diabetic as well as in the diabetic.

We speak of the leading cause of adult deaths as "disease of the heart and blood vessels." However, disease of the blood vessels of the heart and elsewhere in the body is not always present in many victims of the heart attack, especially when the victims are between the ages of 20 and 40 years. Above 40 years, victims usually

show abnormalities of the smaller arteries, especially the coronary arteries which supply the heart muscle. The common abnormality is a change in the character of the wall of the artery, namely, thickening, hardening with loss of elasticity, and deposition in localized areas of a foreign substance within the wall of the artery, called cholesterol. Cholesterol is a normal constituent of the blood, but it has no place in the cells that go to make up the walls of arteries. Hence it is foreign to these cells, an abnormal component which damages and destroys normal cells and eventually causes the cells to be replaced by inelastic fibrous scar tissue — a process known in medicine as arteriosclerosis or atherosclerosis (sclerosis = hardening). In advanced cases of arteriosclerosis the mineral calcium is frequently deposited in these damaged areas and so the wall of the artery becomes hard and brittle, with roughening of the lining membrane of the arterial lumen. Instead of a continuous smooth lining, there is a roughened lining, often puckered, with localized areas called placques, where the lining is hard, fissured, and even ulcerated. Small hemorrhages may occur beneath the lining of such damaged arteries.

It is generally believed that such a damaged artery within the heart muscle becomes obstructed at the time of the heart attack resulting in severe damage to the segment of heart muscle nourished by the obstructed artery. It is also generally believed that the obstruction is caused by the formation of a clot at a point where the artery is narrowed by the sclerotic process and where the lining membrane is damaged sufficiently to cause obstruction to the flow of blood with slowing of the stream and eventual clot formation. This clot formation is termed in medicine, "thrombosis." And, since the nutrient arteries of the heart muscle are branches of the left and right coronary arteries, the term "coronary thrombosis" has been used to describe the sequence of events leading to the heart attack.

This theory, which attributes the heart attack to arte-

riosclerosis and thrombosis is weakened by the fact that many victims of heart attacks, especially those between 20 and 40 years, show no evidence of arteriosclerosis and coronary thrombosis at necropsy. In other words, one may be the victim of a heart attack with entirely normal coronary arteries. There is, therefore, some other factor which is the fundamental cause of the heart attack, a factor which is not accompanied by or dependent on any abnormal change in the wall of the coronary artery. A further argument against the arteriosclerotic theory is the fact that many individuals reach old age with advanced arteriosclerosis, usually generalized, and die from some non-cardiac cause without ever having suffered with symptoms of impaired cardiac function. Evidence will be presented to show that another mechanism is the cause of the heart attack, a mechanism that will apply to all cases of heart attacks regardless of age and regardless of the presence or absence of coronary arteriosclerosis. It is my belief that the occurrence of thrombosis in a coronary artery during a heart attack is a secondary phenomenon, purely incidental, and is not the prime factor initiating the attack.

Much has been written in scientific and lay publications about the role of fat in the diet as a cause of the cholesterol deposits in the arteries with resultant atherosclerosis and heart attacks. Some researchers have implicated fatty foods in the average American diet as the fundamental cause of coronary sclerosis and heart attacks. Fatty foods in the diet are a source of cholesterol which is always found in the blood in concentration ranging from 140 to 200 mgms. in each 100 cc. of blood. The body can also synthesize cholesterol from ingested sugar and starch, a fact that has been apparently overlooked by some researchers. Foods containing fats, such as eggs, meat, cream, poultry, cheese, yield much cholesterol. Some authors who advocate the arteriosclerotic theory as the cause of heart attacks state that the ingestion of such

foods causes an abnormal rise in the cholesterol content of the blood. They state that some of the excess cholesterol is deposited in the arterial wall, thereby leading to arteriosclerosis. This theory is weakened by the fact that many individuals with arteriosclerosis do not show elevation of the blood cholesterol level. Another argument against the fatty theory is the fact that the Eskimo living within the Arctic circle is notoriously free of arteriosclerosis and heart attacks, and yet consumes an extremely high fat diet compared with the average diet of Americans in the United States. The Eskimo living within the Arctic circle (as distinguished from the Eskimo living in contact with the white man in Alaska and eating the white man's diet) lives entirely on protein and fat in the form of meat, fish, and blubber. The advocates of the fatty theory are at a loss to explain this immunity from heart attacks and arteriosclerosis which the Eskimo enjoys.

Some researchers have stated that "unsaturated fats" such as the fats found in vegetable oils do not cause elevation of the blood cholesterol as do the fats found in animal foods such as meats, eggs, and cream. These researchers have advocated a drastic reduction in the ingestion of these latter foods that yield the "saturated fats." This is a most dangerous and undesirable recommendation because these foods are vital and highly important to the body economy since they are the best sources of the proteins needed by the body for all purposes: fetal development, growth during childhood, maturity, tissue repair following infections and trauma, and normal daily bodily functions. These animal proteins yield the essential amino acids (the fundamental building blocks of proteins) which are needed to synthesize essential body proteins such as hormones, immune bodies, digestive enzymes, cellular enzymes needed for vital cellular functions going on in every living cell, and for the synthesis of a wide variety of body proteins needed for the replacement of tissues lost in daily wear and tear. On the face of it, to implicate these animal foods

as the ultimate cause of heart attacks because of their fat content is highly dubious and dangerous and unless absolutely confirmed as the cause of arteriosclerosis they should not be eliminated from the diet nor even slightly reduced. Elimination of animal protein from the diet would leave one with a diet that is largely vegetarian. I have always regarded the human as a carnivorous animal. He is built like one. He has canine teeth. Normally he has the small torso and abdomen that go with the smaller digestive tract of the carnivore. Herbivorous animals must have large digestive tracts and large bellies to accommodate the tract. Compare the lion, tiger, and panther, which are carnivores, with the cow and elephant, which are herbivores. The lion is sleek, fast, with small narrow belly, and relatively more powerful weight for weight than the larger, clumsier cow or elephant. The cow and elephant must eat for most of the 24 hours in a day, day in and day out, and thus need a longer digestive tract. The lion can eat its fill of meat from its prey in a few hours and then do without food for several days, if necessary. The herbivore must eat relatively great quantities of vegetable food (grass) in order to get enough protein for essential needs. It is estimated that a cow must eat 14 pounds of grass to produce one pound of meat. It is thus obvious that meat is a highly concentrated source of protein and is thus a more efficient source of food and energy than any vegetable food.

Chapter III

HEART PAIN IS NOT DUE TO CORONARY ARTERIOSCLEROSIS

This monograph is concerned not only with the attacks of chest pain which afflict older individuals, the disease commonly called "angina pectoris," but also with the attacks of chest pain caused by heart malfunction in youngsters, adolescents and young adults. Practically all medical authorities on this subject distinguish between the heart pain in the younger group and the heart pain in the older group, usually stating that the pain in the younger group is "functional" or "nervous" in origin, whereas the pain in the older group is organic in origin in that it is due to anatomical changes in the coronary arteries, the nutrient arteries which supply the heart muscle with oxygen and sugar essential for proper functioning of the muscle. This distinction was necessary because the pain in the older individuals was attributed to the anatomical arterial changes which occur with aging and hence could not apply to the younger individual with normal coronary arteries. Much confusion resulted when young adults in the second and third decades died from heart attacks only to show normal coronary arteries at the post-mortem examination. Their attacks were exactly similar to those killing older individuals who showed narrowed and thickened arteries at post-mortem. Evidently some other factor was causing the heart attack.

I disagree with this distinction between the heart pain in the young and the heart pain in the old. Evidence will be presented to show that the fundamental causal mechanism responsible for the pain is the same in all ages, readily substantiated by the fact that a single form of therapy aimed to correct this causal mechanism can prevent attacks of pain in all age groups. Any form of therapy that can prevent the attacks of pain can be expected to

prevent the occurrence of the heart attack. The concept that a single causal mechanism is responsible for the heart pain in all age groups will serve to dispel much of the confusion that exists today regarding heart disease.

At the present time, it is generally believed that the heart pain is caused by a reduced delivery of oxygen to the contracting heart muscle and that the reduced delivery is caused by narrowed thickened sclerotic arteries which bring the blood in which the oxygen is carried. The narrowed arteries are unable to deliver sufficient oxygen and so the work of the heart muscle is impaired. This abnormal condition has been termed "ischemia" of the heart muscle. I do not attribute the pain to the narrowing of the coronary arteries and subsequent impairment of the circulation of the blood through the heart muscle. Young people have heart pain and yet have normal coronary arteries. The heart pain in older individuals with narrowed sclerotic arteries is modified by the presence of these abnormal arteries to the extent that: (1) the individual is more susceptible to the attacks of pain in the chest; (2) the attacks come on with greater frequency, at rest as well as on exertion; (3) the attacks are more severe and last longer. The narrowed sclerotic arteries serve only to aggravate the attacks of pain, but are not the fundamental cause of the pain. Millions of individuals reach old age with generalized arteriosclerosis involving the coronary arteries, never experience heart pain, and die from some non-cardiac cause. Actually, only a small percentage of all individuals with narrowed coronary arteries suffer heart pain during life or die from a heart attack. Post-mortem examinations of hearts of individuals dying in advanced age from accidental causes and who never suffered heart pain, have often revealed marked narrowing and thickening of the coronary arteries. And yet, in younger individuals dying suddenly from a heart attack and in whom slight narrowing of the coronary arteries is found at post-mortem, death has been attributed to the

slight coronary changes. Clearly some factor, other than the coronary arterial changes, is causing the attacks.

There have been published reports of post-mortem examinations of elderly individuals dying of a non-cardiac cause, with no history of heart disease during life, and in whom the coronary arteries were completely obstructed at their origin by arteriosclerosis or syphilis of the aorta, the large arterial trunk from which the coronary arteries arise. Certainly, if interference with the blood flow through the coronary circulation is the cause of heart pain and heart attacks, such individuals should have had some symptoms or signs of cardiac embarrassment during life. Some writers on the subject, when confronted with such baffling facts, have been at a loss to explain these exceptions. Other writers have attempted to explain these contradictory instances but have been unconvincing and have only added more confusion to an already baffling problem.

Heart pain in older individuals with coronary arteriosclerosis has been called angina pectoris. The pain has been attributed to a reduced oxygen delivery to the heart muscle resulting from the reduced blood flow through the narrowed sclerotic arteries. This has been called coronary insufficiency by some. However, it is difficult to attribute the attacks of pain to permanent irreversible anatomical changes in the coronary arteries chiefly for the following reason: the attacks of pain may come on suddenly, persist for a variable period of time, and disappear completely, but the arterial lesions are static. Hence the fundamental causal mechanism must be one that can appear suddenly, persist for a while, and then disappear — a completely reversible mechanism. Anatomical arterial changes are not reversible. This fundamental mechanism can vary in severity, and, while it is in itself reversible, it can lead to irreversible changes which occur during the heart attack in the form of permanent heart damage (thrombosis, infarction) from which there may be partial recovery, complete recovery, or death.

The mechanism causing the chest pain and the eventual heart attack would thus have to be an exceedingly labile one, that can come without warning, vary greatly in severity, and disappear spontaneously. Such a mechanism could very readily involve an essential nutrient to the heart muscle which is present in the blood stream, a biochemical dissolved in the blood which is capable of wide fluctuation in short periods of time from normal to abnormal range, and capable of embarrassing the heart muscle during such abnormal fluctuation. There is such a chemical in the blood, an essential nutrient for the heart muscle, essential for normal heart action, which must be available to the heart every moment of life in order to permit the heart to beat around 70 times per minute, in the adult during rest, for every minute of life. This chemical is called the blood glucose or blood sugar.

Chapter IV

THE BLOOD SUGAR AND ITS REGULATION

Sugar is an essential constituent of the blood. It is also called "blood glucose." In the fasting state, as in the morning before breakfast, the blood sugar concentration is between 80 and 90 mg. per 100 cc. of blood. Even after several days of fasting the blood sugar will be maintained around this level in a well-nourished individual. It is essential to normal health that the blood sugar be maintained at this level, and that it should not fall below this level for periods longer than an hour. After a meal containing sugar the blood sugar rises at once, usually reaches a concentration of 120 to 140 mg. after 1 to 2 hours, and then gradually falls during the third and fourth hours to the previous fasting level. (Fig. 1.) In some individuals the blood sugar may reach 180 mg. and higher after a meal containing sugar, and, accompanying this high level,

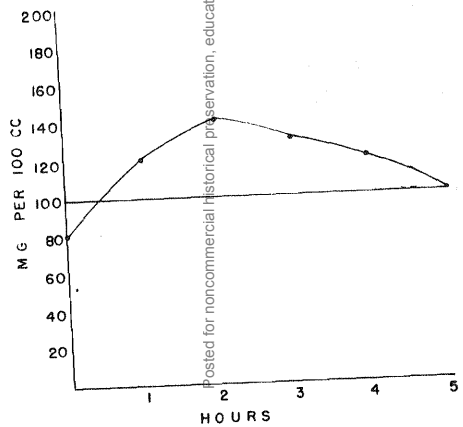


FIG. 1
Normal glucose tolerance curve. Subject drank a solution containing 100 grams of glucose. The blood sugar level rises gradually to a peak during the first two hours and then falls during the third and fourth hours to the previous fasting level. All blood sugar values lie well above the 100 mg. baseline, except the fasting value.

sugar may be found in the urine. This abnormally high concentration of blood sugar is called in medicine hyperglycemia (hyper-excessive; glycemia-blood sugar). The opposite state, an abnormally low blood sugar concentration, is called hypoglycemia (hypo-less; glycemia-blood sugar). Figs. 2, 3, 4, 5 show the hypoglycemic type of curve, and Fig. 6 shows the hyperglycemic type of curve.

There is a strong possibility that the sugar, as it exists in solution in the blood during the fasting state or when an individual is eating an exclusive protein and fat diet like the Eskimo does, is different chemically from the sugar which results from the digestion of the refined sugar which one buys in a package and uses to sweeten coffee. There is no doubt in my mind that the sugar in the blood of a carnivore is different chemically from the commercial product called dextrose which is derived by processing corn starch with chemicals. The manufacturers of dextrose, in their advertisements, make no distinction and would have you believe that the sugar derived from eating

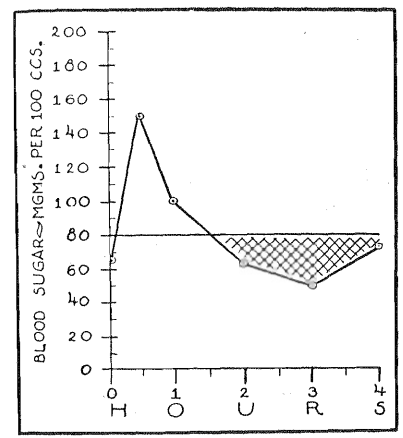


FIG. 2
Hypoglycemia. Blood sugar curve after 100 grams of glucose. Note the sharp rise to 150 mg. at ½ hour followed by a sharp fall to abnormally low levels for about 2½ hours. Hatched area indicates the extent and duration of the low blood sugar period. During this latter period total body oxygen absorption will fall to abnormally low levels.

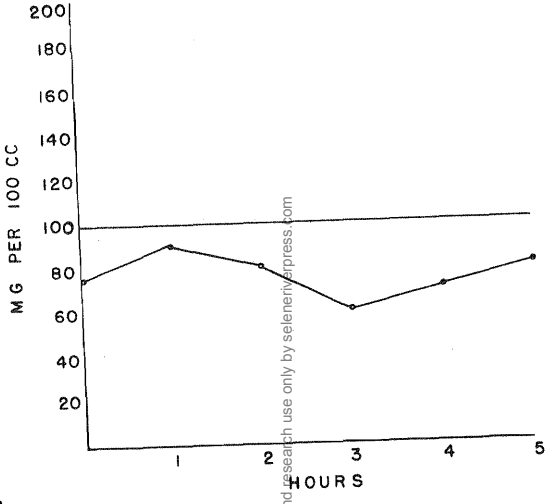


FIG. 3
Hypoglycemia. Blood sugar curve after 100 grams of glucose. This is the "flat" type of curve obtained in some patients with the glucose tolerance test. Note that all blood sugar values lie below the 100 mg. baseline.

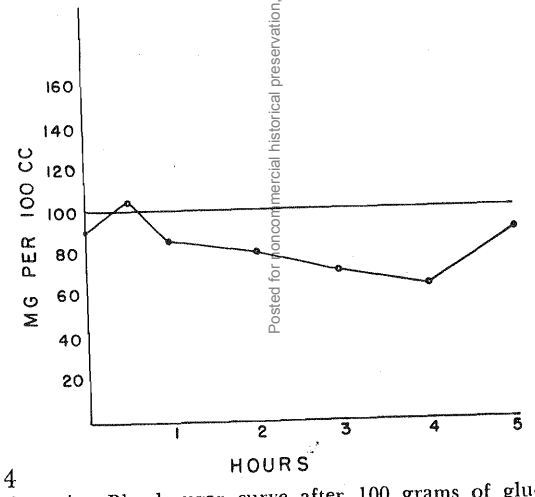


FIG. 4
Hypoglycemia. Blood sugar curve after 100 grams of glucose. There is a slight rise at the ½ hour followed by a progressive fall during the next 3½ hours. At the fourth hour there is a spontaneous rise which is due to adrenal-sympathetic stimulation, the defense mechanism that prevents a further fall in blood sugar.

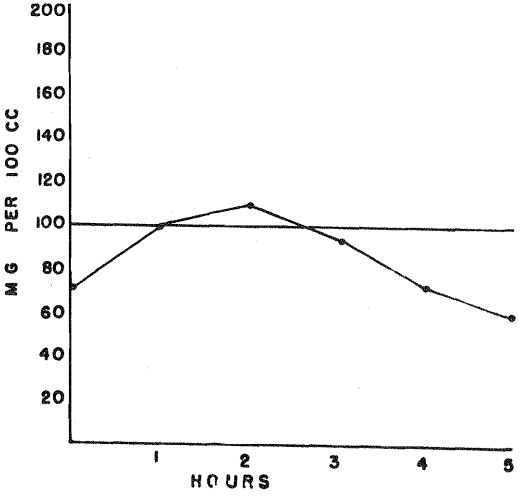


FIG. 5
Hypoglycemia. Blood sugar curve after 100 grams of glucose. The contour of this curve resembles that in the normal tolerance test (Fig. 1) but it is moderately depressed so that most of the levels lie below the 100 mg. baseline.

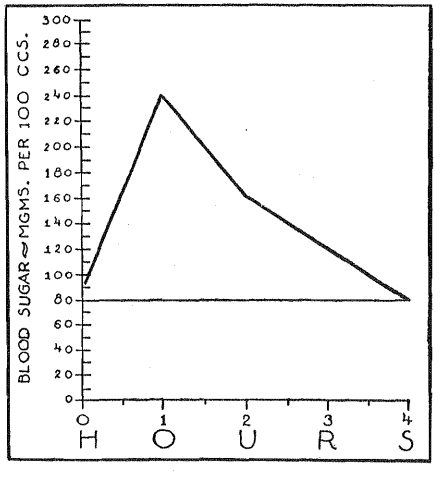


FIG. 6
Hyperglycemia. High blood sugar type of curve after 100 grams of glucose. Note sharp rise to around 250 mg. after one hour with rapid fall during the second and third hours to fasting level. Individuals with this type of curve have symptoms similar to those encountered in hyperglycemia. Individuals who give this type of curve may be regarded as potential diabetics.

meat is the same as their artificially prepared dextrose. From clinical observations made during the past 20 years and from close study of my own reactions to various foods I am absolutely convinced that the blood sugar derived from meat and other protein foods is different chemically from the sugar derived from eating a candy bar. In the chapter on "Gamma Glucose" I shall discuss this matter in detail.

The maintenance of the blood sugar at normal levels is brought about by an efficient regulatory mechanism. The main organs in this mechanism are the liver, the autonomic nervous system, and certain glands of internal secretion called endocrine glands. The liver is at the center of this mechanism and serves as the storehouse of the blood sugar supply. The foods we eat are digested and broken down into simpler chemicals which are absorbed from the gastro-intestinal tract, carried to the liver, and there built up (synthesized) by the liver cells into a complex compound called glycogen. Glycogen has also been called "animal starch." However, glycogen has a different chemical construction from starch and it would be wrong to regard starch as the equivalent of glycogen. To the cells of the body, glycogen is a natural source of energy, whereas starch is to be regarded as an unnatural, even harmful, source of energy.

In the fasting state glycogen is constantly being broken down in the liver cells into a simpler chemical called glucose or sugar which is then liberated into the blood stream at such a rate as to maintain the blood sugar level at around 80 mg. per 100 cc. This breakdown of liver glycogen is controlled chiefly by nerves of the autonomic system which consists of two divisions, the sympathetic and the parasympathetic. The autonomic nervous system is so called because it is not under direct voluntary control; it regulates involuntary functions such as digestion, intestinal movements, the heart rate, the blood pressure, respiration, and body temperature. In general, the action

of the sympathetic system (division) is to facilitate and hasten the breakdown of liver glycogen with subsequent elevation of the blood sugar level, and the action of the parasympathetic division is to favor the synthesis and storage of liver glycogen. The two divisions are thus essentially antagonistic, and, at any one moment, the blood sugar level will be the resultant of these two opposing forces.

As for the endocrine glands, the important ones in blood sugar regulation are the pancreas and the adrenals, both of which are under the control of the pituitary gland. The pancreas produces the hormone insulin which brings about the storage of glycogen and thus prevents the development of hyperglycemia. (Diabetes, a disease characterized by an inability to metabolize carbohydrates in normal fashion, due to an insufficiency of insulin, is always accompanied by a chronic hyperglycemia.) The adrenal glands produce a hormone called adrenalin whose action on the liver is to facilitate the breakdown of liver glycogen with subsequent elevation of the blood sugar. Adrenalin works in conjunction with the sympathetic nerves, and, since both produce the same effect, the two together are known as the adrenal-sympathetic system.

Since the heart of the blood sugar regulatory mechanism is the liver with its glycogen storage, and since glycogen is being built up from the products of digestion, it becomes apparent that the amount of glycogen will depend on the nutrition of the individual. This is most important because it is the one factor that the individual can control.

In the fasting state glycogen is constantly being broken down into glucose because glucose is continually being removed from the blood stream since practically every organ in the body utilizes glucose as a source of energy. Glucose combines with oxygen within the cell, a process called oxidation, in order to provide the energy needed by the cells of the body to perform their particular function. The body's cells are working every moment of life

and a steady supply of glucose and oxygen must be made available by the blood. The heart, the brain and spinal cord (which comprise the central nervous system), and the muscles, utilize glucose practically exclusively for energy purposes in performing their specialized functions. A constant delivery of glucose is needed at every moment of life in order that such vital organs be adequately provided with their source of energy. These organs are never in a resting state.

The constant demand of the tissues of the body for glucose necessitates a constant source of supply. Failure of this supply mechanism may result in death. If the supply of glucose fails the body will draw on its own tissues (muscle and fat) to get glucose. Under such circumstances these tissues break down yielding split-products which are synthesized to glycogen in the liver and then paid out as glucose into the blood. If this state is prolonged, weight loss results with eventual emaciation. This mechanism explains the weight loss and emaciation that often accompany worry, anxiety states, and chronic diseases such as cancer and far advanced tuberculosis. In these states the individual loses his appetite, becomes malnourished, and draws on his own tissues to maintain blood sugar levels.

At this point I wish to stress the importance of a steady supply of glucose to the heart muscle, a muscle which never stops working until the day of death. It is, indeed, a remarkable organ. It beats continually, automatically, silently, and rhythmically. At rest it beats in such a fashion that one is never aware of its beat. Abuse of the heart muscle, such as during abnormal stimulation by nicotine, can so increase the force of the heart beat, with or without an increase in rate, as to make one aware of the beat, a phenomenon called palpitation. Palpitation is normal during periods of increased forceful heart action when such action is demanded by exertion and emotional states. To stimulate the heart action needlessly by nicotine is to

increase the work of the heart unnecessarily and thus actually shorten the life of the individual by needless wear and tear of the heart muscle. And, what is of more importance, the nicotine, by continuous insult to the heart muscle by years of tobacco smoking, may impair the normal function of the organ, at first temporarily with reversible damage, and later permanently, with irreversible damage. The manner in which nicotine adversely affects the function of the heart will be discussed in another chapter.

I also wish to emphasize the fact that the blood sugar supply to the central nervous system is particularly important, because abnormal fluctuations in the blood sugar level affect not only the function of the heart but also the function of the central nervous system. Many of the symptoms experienced by heart patients are due directly to the effects of the fall in blood sugar on the cells of the brain and spinal cord. The blood sugar must not only be supplied continuously, but must also be maintained at optimum level, around 80 mg. per 100 cc. When the blood sugar falls below 80 mg. certain organs, especially the central nervous system, will be embarrassed and signs and symptoms of disturbance in function make their appearance. The severity of the signs and symptoms will depend on how low and at what rate the blood sugar falls.

When the blood sugar falls to 60 to 70 mg., symptoms are usually mild and may consist of slight headache, faintness, muscular weakness, hunger, irritability, and perhaps a feeling of nervousness or tension.

When the blood sugar falls to 50 to 60 mg., the symptoms are more marked and will consist of headache, dizziness, unsteady gait, faintness, weakness, marked irritability, pallor, sweating, tremors, palpitation, and general nervousness.

If the blood sugar falls to 40 mg. or lower, unconsciousness usually occurs. The unconsciousness is exactly like that seen in the ordinary fainting spell and is usually accompanied by pallor, sweating, and feeble rapid pulse.

Recovery from such faints is usually spontaneous and is brought about by a spontaneous rise in the blood sugar which occurs as a protection against a further fall in the blood sugar level. The abnormally low blood sugar level stimulates certain centers in the central nervous system, and from these centers impulses go out over the sympathetic nerves to the liver with a resultant increase in breakdown of liver glycogen and subsequent rise in blood sugar.

At the same time the adrenal glands respond to the fall in blood sugar by an increased output of adrenalin into the blood. The adrenalin acts upon the liver to step-up the breakdown of glycogen so that more glucose will be liberated into the blood. The effects of this adrenal-sympathetic stimulation with rise in the blood sugar level brings about spontaneous recovery from the unconscious state. The cells of the brain now receive their normal amount of sugar and resume their normal functions, one of which is responsible for the condition of awareness or consciousness.

The hypodermic injection of adrenalin will also hasten recovery because the injected adrenalin has the same effect upon the liver as the body's adrenalin. The common practice of applying cold water to the head, the inhalation of vapors of spirits of ammonia and vigorous rubbing of the skin, hasten recovery from simple faints because these measures can likewise cause stimulation of the adrenal-sympathetic system.

If the adrenal-sympathetic response is weak, recovery may be slow; if it is absent, recovery will not occur and death from hypoglycemia ensues. If the adrenal-sympathetic response is adequate, but the liver glycogen storage deficient, the blood sugar may not be restored to normal levels because there is not enough glycogen available. Such deficient storage will result from poor nutrition.

Actually, some of the symptoms experienced by the patient who suffers a fall in blood sugar are side effects due

to the strong adrenal-sympathetic stimulation which mobilizes sugar from the liver in order to effect a rise in blood sugar — obviously a protective mechanism. These side effects of the adrenal-sympathetic stimulation are responsible for the following signs and symptoms of the patient with angina pectoris: palpitation, pallor, cold sweat, tremors, rapid pulse, and an increase in the rate of respiration.

Summarizing, it is evident that the blood sugar must be maintained at 80 mg. for normal bodily function, and that symptoms appear if the blood sugar falls below this level. These symptoms will be mild, moderate, or severe, depending on how low the blood sugar falls. Spontaneous recovery from low blood sugar results from adrenal-sympathetic stimulation of the liver with subsequent rise in blood sugar. Recovery from low blood sugar will thus depend chiefly on (1) an adequate protective response by the adrenal-sympathetic system and (2) the amount of glycogen stored in the liver, which, in turn, will depend on the nutritive habits of the individual.

How the low carbohydrate diet elevates and stabilizes the blood sugar levels.

Since I have stated that the cardiac symptoms are caused by abnormal blood sugar fluctuations and since I have stated that these symptoms may be controlled and prevented by a low carbohydrate diet, I shall now show graphically how the diet actually elevates the blood sugar to more normal levels, and stabilizes them at these levels. The accompanying charts show clearly how the blood sugar is controlled by what we eat. These graphs were obtained from patients whom I was studying and treating for low blood sugar. (Figs. 7, 8.)

You will note that after these patients drank a solution of pure glucose they had a period of low blood sugar which began one to two hours after the glucose was taken and which lasted for one to two hours, and longer. This study

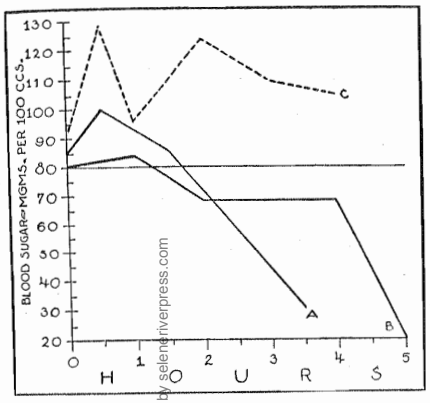


Fig. 7
The effect of various meals on an individual with hypoglycemia. Curve A shows the course of the blood sugar after 100 grams of glucose. Curve B shows the course after a high carbohydrate breakfast consisting of one orange, a bowl of oatmeal with cream and sugar, a piece of cake, and a cup of sweetened coffee. Curve C shows the course after a low carbohydrate breakfast consisting of one orange, two eggs, one slice of bread and butter, and a glass of milk-cream mixture (4 oz. milk and 3 oz. cream).

A low carbohydrate meal elevates and stabilizes the blood sugar levels. This stabilizing effect is important because some of the symptoms of low blood sugar are due to a rapid fall in blood sugar which accompanies wide fluctuations in blood sugar levels following the ingestion of sugar and starch.

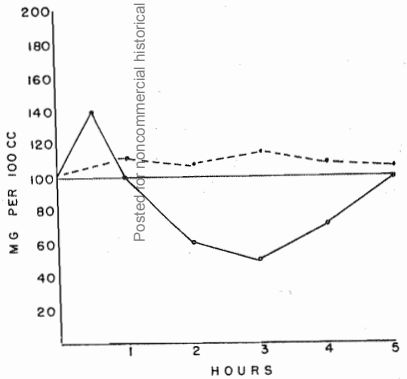


Fig. 8
The effect of a low carbohydrate breakfast on another type of hypoglycemic curve. The solid line shows the course of the blood sugar after 100 grams of glucose. The dotted line shows the course after a low carbohydrate breakfast consisting of one orange, two eggs, one slice of bread and butter, a glass of milk-cream mixture.

of the blood sugar is called the "glucose tolerance test" and is employed for the detection of hypoglycemia or hyperglycemia. You will also note that when the patients ate a meal containing sugar and starch they also had periods of low blood sugar which came on an hour or so later and which lasted from one to two hours. The low blood sugar was more marked and lasted for a longer time after the glucose solution than after a meal containing starch.

It is an established fact that this paradoxical depressant effect on the blood sugar level is more readily exerted by sugar than it is by starches. I have observed these results in hundreds of cases and similar results have been obtained by other investigators. It is a surprising paradox: the more sugar (and starch) you eat, the more likely you will develop low blood sugar. Drs. E. P. McCullagh and C. R. K. Johnston have shown how the glucose tolerance test is readily influenced by diet. (2)

It had been found by other investigators that a meal consisting of protein, fat, and non-sugar and non-starch carbohydrates, never caused low blood sugar. The addition of sugar and starch to such meals could readily produce low blood sugar. Figs. 7 and 8 show clearly how the blood sugar is maintained at 80 mg. and higher after a meal containing no sugar and little or no starch, whereas, in the same individual, a meal containing sugar and starch will cause low blood sugar. I have thus found that a diet completely free of sugar and starch and consisting of proteins, fats, and non-starchy vegetables, may be adhered to for years with beneficial effect and absolutely without harmful effect. There is no supporting evidence to indicate that sugar and starch are necessary for health or for energy purposes. The human is a carnivore and can thrive on protein and fat alone, if necessary.

The Eskimos thrive well on meat and fish which yield only protein and fat and heart disease is a rarity among them. American and European explorers in the Arctic

regions have lived on meat and fish for as long as 18 months and have maintained perfect health all the time on such a diet. Vilhjalmur Stefansson, the Arctic explorer, has described his experience on such a diet in great detail. He states that he was in perfect health on a diet which consisted of protein and fat alone. Eskimos who live on meat and fish are not susceptible to many of the white man's diseases. They do become susceptible when they live among white men and eat the white man's diet with its sugar and starch. It is true that the Eskimo's fresh contact with the white man exposes him to infectious diseases to which he (the Eskimo) has not had the opportunity to become immune. The presence of sugar and starch in the Eskimo's new diet is of greater significance. A United States public health officer stationed in Alaska has blamed this dietary factor for the great susceptibility of the Eskimo to tuberculosis, for example.

The significance of the hyperglycemic curve.

The individuals who show high blood sugar levels (hyperglycemia) in the glucose tolerance test have symptoms that are similar to those encountered in hypoglycemia. It is impossible to predict from the history and clinical examination which type of curve will be obtained in the glucose tolerance test. Individuals with hyperglycemia are mild diabetic cases and sugar may be found in the urine after a meal containing sugar and starch, but not consistently, and the urine may be free of sugar after an all-night fast. Fully developed cases of diabetes usually show urinary sugar consistently. Mild hyperglycemic patients are readily controlled by a low carbohydrate diet and usually have normal blood sugar levels on such a diet without the use of insulin, as shown in Fig. 9. It is indeed a remarkable fact that the same low carbohydrate diet will elevate to normal range the low blood sugar of the hypoglycemic patient and lower to normal range the high blood sugar of the hyperglycemic patient. I have also

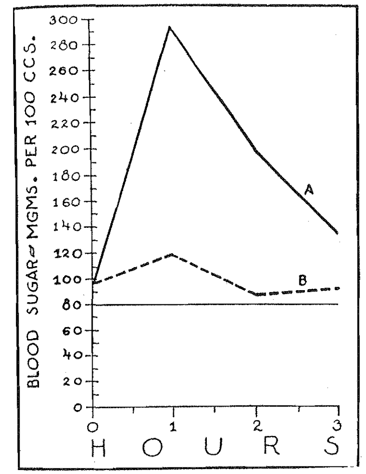


FIG. 9
The effect of a low carbohydrate meal on the blood sugar in a patient with hyperglycemia. Curve A was obtained after 100 grams of glucose; note sharp rise to abnormally high levels followed by a rapid fall to previous fasting level. Symptoms exactly like those seen in hypoglycemia may occur during the period of rapid fall. Curve B was obtained after a low carbohydrate meal; note that all blood sugar levels are within normal range and do not show wide fluctuations.

found that if a patient is underweight he will gain weight on the low carbohydrate diet; and if he is overweight he will lose weight on the low carbohydrate diet. The gain or loss in weight will occur regardless of whether the patient is hypoglycemic or hyperglycemic.

Physical exertion and its relation to heart attacks and low blood sugar.

Cardiac pain and fatal heart attacks frequently follow severe physical exertion; sometimes during the exertion, sometimes after an hour or so, and sometimes after 12 to 36 hours. What is the mechanism responsible for the immediate or delayed effects? How does physical exertion cause a fatal heart attack? In line with the ideas presented in this monograph, it will be necessary to show that such exertion will cause an ultimate fall in blood sugar to abnormal levels.

Physical exertion, when immoderate, is accompanied by strong and prolonged muscular contractions which are performed at the expense of the blood sugar and result in a severe drain on liver glycogen stores. The depletion of liver glycogen results in a reduced output of glucose into the blood and the blood sugar concentration falls. Drs. Levine, Gordon, and Derick of Boston, in an article published in the Journal of the American Medical Association (Vol. 82, page 1778, 1924) reported low blood sugar in six of nine marathon runners at the end of a long race. The blood sugar values of the six were 45, 47, 49, 50, and 65 mg. per 100 cc. All six finished the race in poor general condition and one of them was on the verge of collapse. Three who finished in good condition had normal blood sugar values, viz., 82, 82, and 89 mg. per 100 cc.

The marked fatigue and exhaustion after severe physical exertion is due largely to a depletion of liver glycogen and resultant lowering of the blood sugar level. If the blood sugar falls sharply during the exertion, an attack may occur during the actual performance. If the blood sugar fall is delayed, the heart attack will be delayed. To maintain normal blood sugar levels after unusual exertion and thereby prevent a heart attack, I would recommend a short rest period followed by frequent small feedings of high protein foods, such as meat, eggs, poultry, fish, concentrated meat or chicken broth, with non-starchy vegetables. Tomato juice should be drunk rather than sweetened soft drinks or fruit juices. Sugar, foods containing much sugar, and sweet soft drinks are not recommended because there is a strong possibility that they may cause a sharp fall in blood sugar similar to the fall caused by glucose in the tolerance test.

In the case of the individual who suffers a heart attack 12 to 36 hours after the physical exertion, I would say that there was a delay in restoring to normal the liver glycogen storage due to inadequate nutrition and so there

was a continuous depression of the blood sugar level because of the reduced output of glucose from the liver. Then, following the ingestion of sugar or foods containing sugar, there was an aggravation of the hypoglycemia with resultant heart attack.

In the average normal adult, under normal nutritional conditions, when the liver glycogen storage is maximal, the amount of glycogen stored in the liver is only about 90 grams (3 ounces). This is a relatively small quantity and severe physical exertion may readily exhaust the glycogen stores after a short time with resultant lowering of the blood sugar levels.

Chapter V

THE PREVALENCE OF LOW BLOOD SUGAR

Personal observations since 1937 have convinced me of the frequency of low blood sugar in children, adolescents, and adults. I have performed hundreds of glucose tolerance tests routinely on patients attending a medical clinic for disorders and diseases common in everyday practice. I found evidence of low blood sugar in more than half the cases. I found that a diet aimed to correct low blood sugar did away with or greatly alleviated many symptoms not only in those who showed low blood sugar with the tolerance test but also in those who did not reveal low blood sugar at the time. I also found that an individual may show normal blood sugar on one occasion and low blood sugar on another occasion. I have concluded that any human can experience low blood sugar as long as he or she consumes sugar and starch. McCullagh and Johnston have shown how the continued ingestion of a high carbohydrate diet may cause chronic hypoglycemia and the continued ingestion of a low carbohydrate diet may elevate the general level of the blood sugar. (2)

Many healthy people have symptoms of low blood sugar without realizing that the symptoms are due to low blood sugar. For example, many individuals experience a physical and often mental let-down in their daily activities around 11 a.m. and 4 p.m. At those hours they get a little tired, may have a slight headache or a sensation of lightheadedness, become a little moody or depressed or irritable, and usually are hungry, especially for something sweet to serve as a "pick-up." And so they will usually partake of a cup of coffee or tea or chocolate, pie, cake, pastry, cookies, candy bars, ice-cream, soft drinks, or the like. These sweet foods and beverages afford a rapid relief from their symptoms because they cause a rapid rise in blood sugar level.

I regard as artificial the rapid rise in blood sugar level produced by eating foods containing sugar. The sugar is an artificial stimulant; and in some people the desire for sweets amounts to a craving, and the demand for something sweet during this craving amounts to an addiction. I regard this craving for sweets as abnormal. In the first place the low blood sugar is abnormal and could have been prevented. However slight, it is abnormal and is caused by a dietary error, namely, the ingestion of sugar and starch. The low blood sugar that comes on around 11 a.m. is due to eating sugar and starch at breakfast, and the low blood sugar at 4 p.m. is due to eating sugar and starch at the noon meal. On a high protein low carbohydrate diet the fall in blood sugar at 11 a.m. and 4 p.m. does not occur and so there is no physical let-down and no need for a pick-up. Cigarette smoking can also serve as a pick-up because nicotine can cause an immediate rise in blood sugar level by stimulating the adrenal-sympathetic system, the rise occurring at the expense of liver glycogen.

The physical and mental pick-up which follows eating something sweet is accompanied by a rise in blood sugar which lasts for about 30 to 60 minutes and which is soon followed by another fall in blood sugar. A vicious cycle is thus set up. This rise and fall in blood sugar may occur several times during the waking hours and, as a result, many individuals acquire the habit of drinking 4 to 10 cups of coffee or tea or cocoa daily, or they eat candy at frequent intervals, or drink 4 to 10 bottles of soda pop, or find it necessary to smoke at frequent intervals. The desire for a cigarette actually coincides with a fall in blood sugar and the feeling of satisfaction that comes with a smoke is due to a rise in blood sugar. Denicotinized cigarettes do not satisfy because they do not cause a rise in blood sugar.

Coffee, tea, cocoa, not only cause a rise in blood sugar by reason of the added sugar, but also because they contain caffeine or related chemical compounds that stimulate

the adrenal-sympathetic system and thus cause a rise in blood sugar at the expense of liver glycogen. People who smoke a great deal and drink much coffee and who have poor appetites are in a chronic state of malnutrition because they are steadily depleting the liver glycogen stores and are failing to replenish the stores with proper food.

Many people are irritable in the morning before breakfast because of low blood sugar. Some people are so irritable, moody, morose, or depressed before breakfast that they dare not say anything or do anything until they have had something to eat, if only a cup of coffee. They have learned that they are more pleasant after having eaten something. The blood sugar usually reaches its lowest point in the morning before breakfast because of the all night fast. I have found that eating less sugar and starch the night before prevents low blood sugar in the morning. (See Fig. 10.)

The effects of low blood sugar on morning behaviour

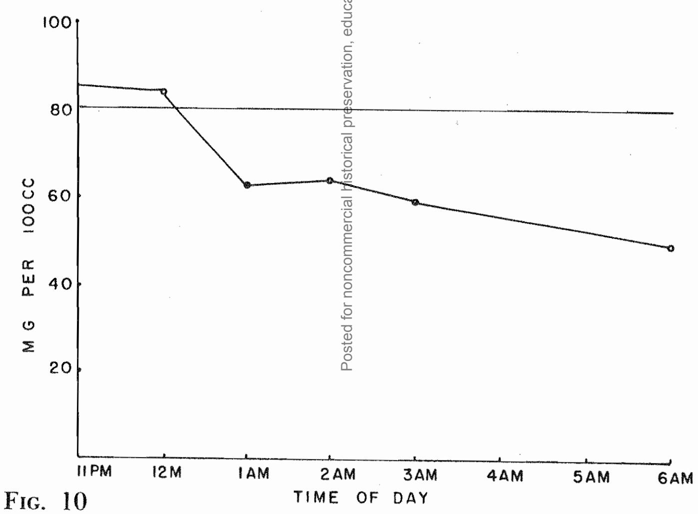


Fig. 10

Course of the blood sugar between the hours of 11 p.m. and 6 a.m. in a patient who had hypoglycemic symptoms around 5 to 6 a.m. Note gradual fall during sleep to the low level of 50 mg. at 6 a.m. Symptoms were readily controlled by a low carbohydrate diet.

and mood is profound. It is a matter of common knowledge that individuals who are irritable and cranky and prone to start arguments before breakfast experience a remarkable change after they have had something to eat. Mothers know that an irritable, cranky, and crying infant is usually a hungry infant. And most wives know that a grumpy husband will feel better after he has had something to eat. It is of great advantage to the wife and mother to know that the blood sugar level is fundamentally related to the behaviour of the husband or child. Having this knowledge, she will readily excuse misbehaviour in hungry members of the family. Thus much domestic friction can be avoided.

Statesmen are learning that properly fed citizens are happier, more contented, and easier to handle; and that poor nutrition goes hand in hand with unrest, violence, and rebellion. The stability, behaviour, and morale of a nation is the sum total of the stability, behaviour, and morale of its individual citizens. A properly fed people is a healthy people.

Errors in judgment in all life's activities are often due to the fact that the individual made a decision during a period of low blood sugar at which time he may have been moody, irritable, depressed, or unstable. I have schooled myself never to make an important decision in the morning before breakfast because my frame of mind may be such as to lead to a decision that I would later regret having made. I therefore always delay making a decision until after I have had a good breakfast. It is surprising how one's attitude toward a problem or a person can change after a good meal, and this only because a rise in blood sugar brings about better brain function by increasing glucose-oxygen consumption. Thus decisions are more likely to be correct when made after a good meal. Experienced negotiators in business and government have learned that the dinner table is a good place to accomplish things.

I have digressed somewhat merely to point out that low blood sugar can happen to everybody, at any age, and often, because we all eat sugar and starch.

Good health is not something one is born with and which persists from year to year as a permanent possession or as a permanent physical characteristic. Good health is largely the result of bodily chemical conditions which fluctuate normally at every moment within a narrow range. The range of fluctuation is controlled by several factors, especially diet and physical activity. And one of the most important chemicals is the blood sugar. To a great extent, then, health will depend on the fluctuation range of the blood sugar. Good health thus becomes a condition which is at any moment dependent on labile (unstable) chemical states in the blood and tissue fluids, and which may be lost or impaired temporarily when the chemical equilibrium is upset. If an individual is able to eat regularly a diet adequate in quantity and quality, indulge in normal physical and mental activity, and get adequate rest and sleep, he may be reasonably assured of continuing good health. Some individuals seem constitutionally healthier and sturdier than others, but they are only relatively so. Healthy and sturdy individuals can succumb to disease very readily when they are under stress, when they become careless in dietary habits, and when they over-exert physically or mentally.

Chapter VI

THE FUNDAMENTAL MECHANISM CAUSING HEART PAIN AND THE HEART ATTACK

The causal mechanism responsible for heart pain in all age groups involves the blood sugar concentration and the immediate precipitating cause of the recurring chest pain, and the final heart attack, is a rapid sharp fall in blood sugar level. The therapy which has been used to prevent the attacks of pain is a diet which is capable of preventing the abnormal fluctuation in blood sugar level. The diet was successfully employed in all age groups, thereby indicating that the causal mechanism was the same in all age groups. The average daily diet of the citizens living in the United States is responsible for the abnormal fluctuation in blood sugar levels because of its high sugar and starch content. The diet used to prevent the attacks of chest pain contains a greatly reduced sugar and starch content and so the abnormal fluctuation is prevented. In severe cases of chest pain, the sugar and starch were practically eliminated.

Physiological Considerations

The heart, whose function is to pump the blood throughout the circulatory system performs a great amount of work during its lifetime. The heart, as an organ, is a powerful muscle, with inherent rhythmicity, free of voluntary control, beating around 72 times a minute, at rest, in the average adult. Its rate varies with age and activity. It is obvious that it performs a great deal of work during each 24 hours and it does its work exceedingly well. It is remarkable that it performs its work so well considering the fact that it is actually being embarrassed by poor diet, overstimulation with nicotine and other drugs in so many individuals.

The heart muscle is made up of innumerable muscle

cells. Like all muscle tissue it needs certain nutrients to perform its work of contraction and relaxation. The chief nutrients are sugar and oxygen, both of which are brought to the muscle by the blood. The sugar is carried in solution and the oxygen is carried by the red corpuscles. In the corpuscle, the oxygen is loosely combined with the hemoglobin to form the chemical called oxyhemoglobin. The smallest blood vessels, namely, the capillaries, lie in close contact with the muscle cells of the heart and the sugar and oxygen are readily given up by the blood to the muscle cells. In the muscle cells, the sugar and oxygen react chemically to yield the necessary energy needed for the work of contraction. The sugar is said to be oxidized (burned) with the formation of energy and the waste products, carbon dioxide and water. The latter waste products readily pass from the cells to the blood where they are removed in the lungs during respiration. The energy which is yielded by the oxidation of the sugar exists in three forms: (1) muscular energy manifest by the contraction of the cell; (2) heat; and (3) chemical energy. Among the many varied and complicated chemical reactions that occur during muscle contraction, is the production of lactic acid, which is not strictly a waste product because it can be synthesized back to a source of energy in the liver in the form of glycogen which in turn can break down to yield glucose (sugar) which can be fed back to the blood for the maintenance of the blood sugar level. Under certain conditions, lactic acid may accumulate in abnormal quantities in a muscle, including the heart, and it is believed that this accumulation of lactic acid along with a few other acid metabolites produced during muscle contraction is responsible for the heart pain. One of the conditions that may cause this abnormal accumulation of metabolites is the condition with which this book is chiefly concerned, namely, abnormally low sugar utilization by the heart muscle.

Since the heart never stops beating, and since it needs

a supply of sugar and oxygen every moment of life, it needs a steady source of sugar and oxygen at every moment of life. This source is the blood. It is thus obvious that it is extremely important that the blood contain normal concentrations of sugar and oxygen at all times in order that the heart perform its function in normal fashion. Any significant interference with the supply of sugar or oxygen will embarrass heart action and the degree of interference will determine the degree of embarrassment — from a mild fleeting chest pain to the severe crushing pain of the fatal heart attack. In normal individuals with normal red cell count, the oxygen content of the blood is normal, and the blood is said to be fully saturated with oxygen. In individuals with anemia, i.e., with a reduced number of red blood corpuscles and therefore reduced hemoglobin, the oxygen content of the blood will be reduced and so there may be interference with heart action. But this condition is not often a cause of heart pain, even in extreme anemia, because the oxygen content of the blood, while extremely low in severe anemia, does not fluctuate. The heart muscle cells can accommodate to the reduced oxygen content of the blood, get used to it, and modify their activity accordingly. Thus no pain is experienced by the anemic individual because there are no violent fluctuations in the oxygen concentration. In the case of the anemic individual with heart pain, the pain can be prevented by a change in diet, just as in the non-anemic individual with heart pain, indicating that the pain mechanism in the anemic individual is the same as in the non-anemic.

It is otherwise with the blood sugar. As will be shown, the blood sugar in patients with heart pain fluctuates widely, reaching abnormally low levels in some patients, i.e., absolute hypoglycemia, and in other patients falling rapidly from high to lower levels, which, although not hypoglycemic, are said to be relatively hypoglycemic, because of the rapid change. Thus the rate of the fall in

blood sugar is extremely important. This explains the occurrence of chest pain in a diabetic individual where the blood sugar may fall from a level of 300 mg. to 250 mg. in a very short time, although both levels are well above the normal range. In individuals who experience these sharp falls in blood sugar, the effect on the heart will be aggravated if, in addition to the rapid fall in blood sugar, there is a fall to absolute hypoglycemia. In such cases there will be two harmful factors at work: (1) the sharp fall in a short period of time and (2) the fall to absolute hypoglycemia.

Numerous observations made by me have indicated that abnormally low blood sugar concentrations by themselves do not cause heart pain if there are no violent fluctuations in the blood sugar. The rapid rate of change in the downward direction results in a severe environmental change for the heart muscle to which it fails to accommodate readily and so the muscle is embarrassed and the symptoms of pain are felt by the patient.

It has been shown that a cell utilizes oxygen in proportion as it utilizes sugar. Oxygen is useful to the cell only if there is some fuel to burn (oxidize) for the production of energy. The blood may be normally saturated with oxygen, but if the blood sugar level is half of what it should be, the body will consume less than normal oxygen. This has been proven to be true for every tissue that utilizes sugar for energy purposes and the two organs that utilize sugar exclusively for energy purposes are the heart and the brain (central nervous system). These two organs will be very sensitive to any sharp fall in blood sugar. The muscle cell of the heart uses sugar exclusively for energy purposes, and so if insufficient sugar is being delivered to it by the blood, it will utilize less oxygen. If the blood sugar falls to 50 mg., or about half of what it should be, a muscle cell will utilize half as much oxygen as it should. A cell that is burning less sugar than it should because of a reduced delivery of sugar occasioned by a fall

in blood sugar, is also utilizing less oxygen than it should and it is suffering from the same effects as if its blood supply were reduced to the point where a comparable anoxemia prevailed.

The Role of Glucose in Cellular Oxidations

It has been shown both in man (3) and dogs (4) that the oxygen uptake of the brain falls during insulin hypoglycemia. Cruickshank and Startup (5) using a heart-lung preparation found that the oxygen consumption of the myocardium (heart muscle) falls as the blood sugar concentration falls. When faced with a slight hypoglycemia the heart is forced to utilize its glycogen since the amount of available glucose was insufficient for its energy needs. Evans (6) also demonstrated that the oxygen uptake of the dog's heart increased on addition of glucose to the circulating blood. Holmes, (7) Dickens and Greville, (8) and Wortis, (9) using the Barcroft-Warburg technic, found that the oxygen consumption of excised pieces of brain and heart fell as the amount of glucose in the nutrient medium was reduced.

It has been accepted by many that the immediate cause of heart pain is an acute oxygen lack on the part of the heart muscle. Rothschild and Kissin, (10) and Katz, Hamburger, and Schutz, (11) induced typical anginal seizures in patients by exposing them to reduced oxygen tensions. Conceivably, any method of reducing the oxygen consumption of the myocardium will cause pain. A reduction in glucose utilization by the heart muscle will therefore cause pain because of the concomitant fall in oxygen consumption. Such a reduced glucose-oxygen utilization will occur during hypoglycemia, a period when there is an insufficiency of available glucose, and in hyperglycemia, where the available glucose is not being oxidized in sufficient quantity.

As has been stated, it is my opinion that a rapid spontaneous lowering of the blood sugar is the cause

of the precordial pain. The pain does not depend so much on the absolute value of the blood sugar as on the rate of change in the concentration. A single specimen of the blood sugar taken during an attack may show a low, normal, or high value. Confusion may arise because symptoms usually associated with hypoglycemia may be observed to occur at hyperglycemic, normal, and hypoglycemic levels. John (12) reported 24 instances of insulin reactions in diabetics. In 11 the blood sugar was 80 mg. or above, and in 5 of these 11 the blood sugar was 200 mg. or more. Titus and Dodds, (13) in discussing John's paper wrote: "These patients undoubtedly conform to the condition which we have designated as 'relative hypoglycemia' since it is obvious that their blood sugar had been higher a few moments before, and had been suddenly lowered to these other levels, still high but relatively hypoglycemic." Macleod, (14) aware of the fact that individuals may be comfortable with blood sugar values as low as 40 mg., and that severe symptoms have been observed at 70 mg., during experimentally induced hypoglycemia, suggested that symptoms depend not so much on the absolute value of the blood sugar as on the rapidity with which that level is reached. Soskin and co-workers, (15) who induced precordial pain and electrocardiographic changes in elderly diabetics with insulin, made the following pertinent statement: "The magnitude of the electrocardiographic deviations induced by insulin did not depend on the absolute level at which the blood sugar was maintained. Analysis indicated that the length of time over which the insulin was administered and the extent of the change from the accustomed level to the new level were the important factors." Mann, (16) has also stated that symptoms ordinarily associated with hypoglycemia will depend on the rate at which the sugar leaves the blood stream. My own experience supports the concept of relative hypoglycemia. I have observed two patients to be virtually aglycemic for over an hour complaining of only marked weakness and

dizziness. Apparently in such instances the blood sugar fell gradually enough to permit the organism to adjust itself.

The symptoms occurring during an anginal seizure are of two types, cardiac and neurologic. Neurologic symptoms will be headache, dizziness, sweating, pallor, flushing, faintness, and syncope. The cardiac symptoms will be pain, heaviness, distress, or other form of precordial discomfort. There are patients who have attacks of faintness or syncope without precordial symptoms, and who finally emerge from such syncopal episodes with anginal symptoms, or with actual cardiac infarction. Further, precordial pain may occur without any neurologic symptoms. The type and number of symptoms occurring during a seizure will depend on whether the fall in blood sugar affects predominantly the nervous system or the heart.

It has been shown that a sudden reduction in the inhaled oxygen content of the air will embarrass heart action and even cause heart pain. The pain has been attributed to a temporary anoxemia resulting from the reduced oxygen saturation of the blood. This evidence indicates that a sudden partial oxygen lack can cause pain. This is true. But the experimental reduction of inhaled oxygen content represents a sudden change in environment which is never experienced by the patient with heart pain in ordinary life, except for the rare instances when he is suddenly transported to very high altitudes with reduced atmospheric oxygen content.

Therefore the patient with heart pain experiences all the ill effects of reduced oxygen inhalation whenever the blood sugar level is abnormally reduced. Hypoglycemia, absolute or relative, means reduced oxygen consumption by all cells that require sugar for energy purposes. The heart muscle suffers an oxygen lack of varying degree every time the blood sugar falls at an abnormally rapid rate.

What Actually Happens in the Heart Muscle to Cause Pain?

At the present time, it is generally accepted that the reduced oxygen consumption in the heart muscle results in an accumulation of lactic acid and other metabolites in abnormal quantity sufficient to cause an irritation of nerve endings in the heart muscle. Impulses travel up the nerves to the central nervous system where, by an obscure process, the sensation of pain is felt, not in the heart itself, but is referred to the chest and upper extremities. Most often the pain is felt in the left side of the chest from which area it may radiate to the left shoulder and down the left arm, usually along the inner aspect, to the ring and small finger. Often the pain is felt beneath the sternum (breastbone) in the midline and is accompanied by a "burning" sensation. The pain may also radiate around to the left side to the back and be localized beneath the shoulder blade. Sometimes the pain is felt in both right and left sides of the chest, and occasionally it radiates to the right shoulder and down the right arm. At times it may radiate around to the right shoulder blade. Some patients complain of a tightness at the root of the neck, or a tight-feeling across the upper chest. In severe attacks and in fatal attacks the pain may be described as vise-like because of the squeezing and crushing sensation. Some authors state that heart pain may be felt in the upper abdomen and not in the chest at all. Such localizations of pain may simulate the pain of gall stone colic or ulcer of the stomach.

The degree of the pain is no doubt related to the degree in the fall in blood sugar level. A mild fall in blood sugar, i.e., a fall that is of moderate extent and at a slow rate, will cause a mild attack of pain which lasts for a short time and disappears completely without any residual soreness. Moderately severe pain will result from a moderately severe fall in blood sugar. Since there are innumerable potential variations in the rate of fall in blood

sugar as well as in the amount of fall, it is no wonder that the pain may vary in severity, duration, and frequency in the same patient from time to time. Since the area of heart muscle that is irritated by the reduced sugar-oxygen consumption may vary with different attacks, so the part of the chest and extremities to which the pain is referred may vary with different attacks. And, since the blood sugar level may recover spontaneously by a protective mechanism (to be explained later on) which is called into play by the fall in blood sugar, the pain will pass off by itself after a varying period of time depending on the adequacy of the protective mechanism.

Therefore, if the heart muscle suffers a reduced oxygen consumption whether because the individual is exposed to a sudden reduction in the atmospheric oxygen, or because of a reduced sugar-oxygen consumption following a sharp fall in the blood sugar, pain will be felt because there will be a partial tissue asphyxia in both circumstances. Pain and soreness will disappear when the tissue oxidations are restored to more normal level and the products responsible for the pain are oxidized and removed.

In the previous paragraphs I have discussed the sequence of events leading to and causing an attack of heart pain which has come and gone without causing any permanent damage to the heart muscle. I shall now discuss the sequence of events in the severe heart attack which causes an "insult" to the heart muscle accompanied by permanent damage in the form of (1) an infarct alone; (2) an infarct with coronary thrombosis; and (3) coronary thrombosis without infarct.

In addition to these three types of heart attack, there are cases reported in the literature where death "seemed" to be due to heart attacks and in which there was no evidence of any of the above three mentioned cardiac insults. This type of death usually occurs in young adult males, 25 to 40 years, and post-mortem examination has

shown either normal coronary arteries or coronary arteries with minimal or moderate arteriosclerosis. Since no other anatomical cause of death was found, death was attributed to the mild arteriosclerosis. These young adults may have attacks of chest pain at some time previous to death, or even shortly before death. My own studies have suggested that death, in such individuals, is due to the rapid sharp fall in blood sugar to extremely low level — a pure hypoglycemic death. Death is due, in such instances, to the sudden deprivation of sugar in the vital centers of the medulla, an important part of the brain. As has been stated, the central nervous system is very vulnerable during sharp falls in blood sugar. There have been many such deaths reported in the medical literature. Some cases have shown brain hemorrhage. In such individuals the central nervous system has felt the effect of the fall in blood sugar before the heart muscle had time to suffer any permanent damage.

What Actually Happens When the Patient Has A Severe Heart Attack?

Before going into the explanation, it will be necessary to define certain terms.

Coronary Thrombosis

This signifies a thrombus (blood clot) formation in a branch of the left or right coronary artery. Most often the left coronary or one of its branches is involved since these supply the left chamber of the heart which carries most of the work load. The left ventricle (chamber) expels the blood which is to go to all parts of the body except the lungs and thus performs more work than the right ventricle which pumps only the blood going to both lungs for purification and oxygenation. The blood clot usually forms in an artery that is the site of advanced arteriosclerosis and that has a roughened ulcerated lining in the lumen. A normal coronary artery is less likely to be the site of a thrombosis.

Cardiac Infarction

This designates the wedge-shaped area of heart muscle that dies as a result of the "insult" to the heart muscle during the severe fall in blood sugar, i.e., the heart attack. It is not due to an obstruction, occlusion, or thrombosis in the coronary artery in that area because infarction may occur with patent coronary arteries. In most cases infarction occurs with thrombosis. The size of the infarct and adequacy of the healing process will determine the future course of the disease in the patient. In successful recovery, the infarct is replaced by strong scar tissue after several weeks and for all practical purposes the patient recovers completely.

What Happens During A Non-Fatal Heart Attack?

To begin with there is a sharp fall in blood sugar with resultant sharp chest pain which may last several hours indicating that a cardiac "insult" with permanent damage has occurred. There will now be signs and symptoms which were never experienced by the patient in previous uncomplicated attacks of chest pain. These signs and symptoms will be: more severe and prolonged chest pain with residual soreness, marked weakness, prostration at times, a fall in blood pressure usually, pallor, sweating, increased respirations, dizziness, headache. Sometimes a victim may faint for a few minutes, and, on regaining consciousness, complains of chest pain or soreness. After a day or two there may be a rise in body temperature along with an increase in the white blood cell count, signs of a generalized reaction to the localized death of heart muscle.

The sharp fall in blood sugar compromises the heart muscle by reducing the amount of sugar available for its work, thereby causing a partial tissue asphyxia because of the reduced oxygen consumption. Although the entire

mass of heart muscle is embarrassed, only a small area appears to suffer intense damage. What actually happens in this area is based purely on speculation. I believe that a localized area of heart muscle, wedge-shaped, with base lying toward the base of the heart and with sides pointing towards the apex of the heart, apparently carrying more of a load than other parts of the muscle, is so irritated by the effects of the reduced sugar-oxygen consumption and the accumulation of lactic acid and other metabolites, that it goes into a sustained cramp. This cramp would be comparable to the sustained cramp that one may experience in the calf muscle during sleep or during swimming. The cramp in the heart muscle is tonic, i.e., there is no intermittent contraction and relaxation. The cramp may last for several minutes, and even longer, producing the severe, crushing, viselike pain so often described by victims of a heart attack. The sustained cramp will include the branch of the coronary artery coursing through the muscle and the artery will be distorted, perhaps kinked, or doubled on itself, with resultant temporary complete obstruction so that no blood will be able to flow through the artery for the duration of the sustained cramp.

As a result of the cramp and temporary obstruction to the coronary blood flow any one of the three types of insult to the heart muscle may ensue: (1) an infarct alone; (2) an infarct with thrombosis; (3) coronary thrombosis without infarct.

I shall discuss the three eventualities in detail:

(1) *Infarct alone* will result from temporary complete obstruction to the flow of blood. The area of heart muscle that is deprived of blood for the duration of the cramp suffers irreversible changes and subsequent death. When the cramp disappears, the distorted coronary branch straightens out with resumption of blood flow. The only permanent anatomical change resulting from the insult is the infarct.

(2) *Infarct with thrombosis.* The infarct will result for the same reasons given above under infarct alone. However, thrombosis occurs in the coronary branch that is caught and distorted in the cramped area of muscle, probably because the cramp is more prolonged and/or the coronary branch is so severely narrowed, hardened and ulcerated that thrombosis readily occurs. Thus, when the cramp disappears, infarct is present along with thrombosis.

(3) *Thrombosis without infarct.* Here the cramp causes distortion of the coronary branch with temporary complete obstruction. Thrombosis occurs because the artery is severely sclerotic and perhaps ulcerated. Infarct does not occur because the cramp was not sustained long enough to cause localized death of heart muscle, even though thrombosis occurred.

In the case of the patient who survives the heart attack, what will one find in such a heart several months after the attack, if one were to examine the heart? Well, in the case of the patient with infarct alone, one will see the wedge-shaped scar replacing the infarct and no other damage. In the case of the patient with both infarct and coronary thrombosis, one will see the healed scar and the thrombotic occlusion in the coronary branch. In the case of thrombosis alone, one will see the thrombotic occlusion but no scar since there was no infarct.

An Explanation for the Variability of Symptoms in Angina Pectoris

The time of the day or night, the duration, and the circumstances attending the occurrence of pain in angina pectoris may be so variable from patient to patient and even in the same patient, as to make it difficult to explain the pain on a single mechanism, i.e., the coronary arteriosclerosis. For example, a patient may have pain only on exertion early in the course of the disease. After a few months or longer, in addition to the diurnal attacks, pain may occur at 2 a.m. waking him from sleep. If the pain were due to exertion early in the course of the disease

how is one to explain its occurrence at rest and sleep? Of course, it has always been possible to find an explanation for the pain in these two diverse circumstances. The pain on exertion was said to be due to an insufficient blood flow to the heart muscle with temporary anoxemia. If the pain occurred at night during sleep, it was said that the slowing of the heart rate and physiologic fall in blood pressure plus the narrowed sclerotic arteries so reduced the blood flow as to cause a temporary anoxemia with resultant heart pain.

When pain was felt on exertion, it would always pass off on rest, for obvious reasons. When pain occurred during sleep, waking the patient, the patient would usually sit up or walk about the room until the pain wore off, although the nocturnal attack would last longer and was more severe than the attack that occurred on exertion during the day. In the night attacks, mild exertion thus seemed to help the patient get over the attack. Since the occurrence of pain on rest and during sleep evidently couldn't be explained by the same mechanism responsible for the pain on exertion, some clinicians invented the term "angina decubitus" which did not explain anything and only added to the already existing confusion. Thus the spontaneous recovery from the attack at night could not be attributed to rest because the patient usually got up and stirred around. Also puzzling and difficult to explain is the fact that the attack at night was often longer and more severe than the attack during the day. A nocturnal attack sometimes lasted for two hours whereas a diurnal attack would last for only several minutes.

Then again, at the onset of the disease, the attacks of pain would occur only on exertion during the day, once or twice a week, and gradually increase in severity and incidence and finally occur during sleep as well. This progression of symptoms may occur in a few months time. Does this mean that the arteriosclerotic process has been getting worse in such a short time? Hardly.

I believe it is easier and more logical to attribute the occurrence of pain under such diverse circumstances to the abnormal fluctuations in blood sugar, and it is easier to accept this explanation especially since the low carbohydrate high protein diet readily prevents the attacks occurring on exertion, at rest, and during sleep. Fig. No. 11 shows the course of the blood sugar over a 20-hour period in a patient with angina pectoris and who had suffered one attack of cardiac infarction. This patient had a moderate hyperglycemia. He was having almost daily attacks of precordial pain which occurred fairly regularly between the hours of 4 and 5 p.m. and between the hours of 2 and 6 a.m. Note that the time of occurrence of the seizures coincides with sharp falls to the lowest levels of the day and night. The heart attack which had occurred seven weeks before making these blood sugar studies and which resulted in an infarction, also occurred at 2 a.m. The fall in blood sugar between 2 and 5 p.m. is sharper and of

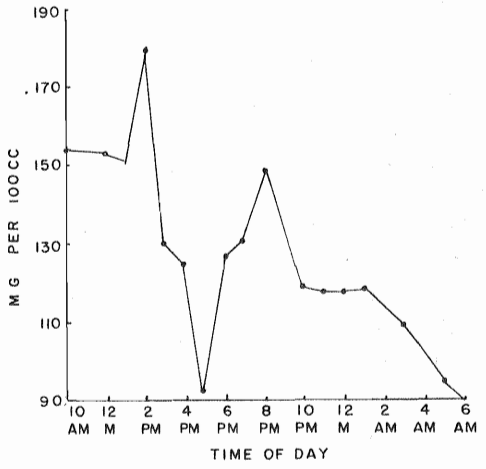


Fig. 11
Course of the blood sugar over 20-hour period in a patient seven weeks after a heart attack. There is moderate hyperglycemia. He had almost daily attacks of precordial pain occurring only between 4 and 5 p.m. and 2 and 6 a.m. Note that the time of occurrence of seizures coincides with sharp falls to the lowest level of the day and night. The original heart attack occurred during sleep at 2 a.m.

shorter duration than it is during the night. This would explain the difference in the length and severity of the attacks between the day and night seizures. The occurrence of the attacks coincides with the fall in blood sugar to the lowest level of the day and night and so the occurrence of pain at those times can be readily explained. Of course, if the patient is engaged in some form of work or activity, such as walking, at the time of the fall in blood sugar during the day, the exertion may serve to favor the occurrence of a seizure at that time. I firmly believe that if the blood sugar were stabilized no seizure would occur even on exertion. This view is supported by the fact that the patient no longer suffers attacks of pain on even greater exertion while on the low carbohydrate diet.

Another puzzling sequence of symptoms in angina pectoris patients is the following: a patient may have breakfast, leave home, walk to the station or to his job, suffer a seizure while walking, stop and rest until the attack passes completely off, proceed to work and engage in strenuous activity the rest of the day with no recurrence of pain. He may have a recurrence the next day or several days later. This sequence of events can not be explained on the basis of temporary anoxemia or ischemia due to rigid narrowed sclerotic arteries, even with the greatest stretch of the imagination. I believe that one can attribute it to a sharp fall in blood sugar occurring soon after breakfast which probably contained more sugar and starch on that particular morning. The patient may have taken more sugar with his coffee or spread more sugar on his grapefruit or cereal. Or, he may have had a lot of syrup with his pancakes and more jelly or marmalade on his toast. He may have had a sweet bun or a piece of pie for breakfast. Figs. No. 12, 13 show a sharp fall in blood sugar soon after the ingestion of glucose during a tolerance test. There was no evidence of a rise in blood sugar during the first 30 minutes. The blood sugar then rose to higher

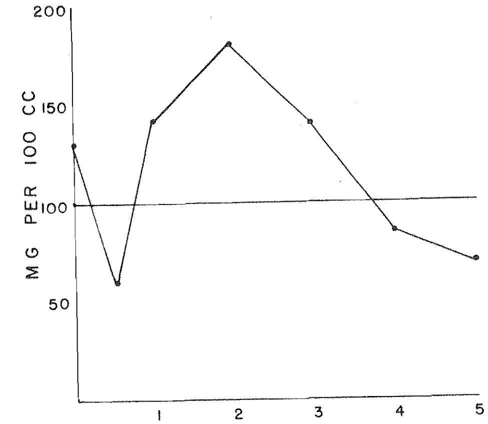


Fig. 12
Course of the blood sugar after 100 grams of glucose in a patient with angina pectoris. Note early sharp fall in blood sugar during the first half hour followed by a sharp rise to around 180 mg. at the end of two hours with gradual secondary fall during the third, fourth, and fifth hours. This type of curve will explain the occurrence of a heart attack or a seizure of precordial pain during or shortly after a meal.

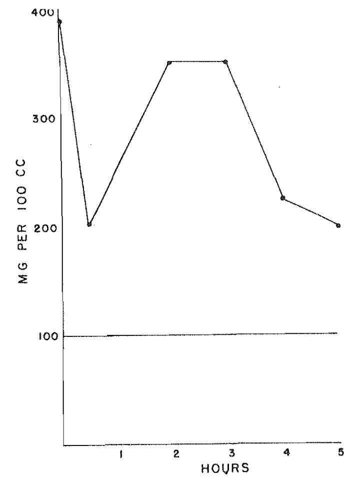


Fig. 13
Course of the blood sugar in a patient with angina pectoris and hyperglycemia (mild diabetes). The curve resembles that shown in Fig. 12, with regard to contour. The chief difference is that this patient had moderate hyperglycemia. Note sharp fall in blood sugar during the first half hour. Such a sharp fall readily explains the occurrence of chest pain or a heart attack during or shortly after a meal.

levels and remained so until the gradual fall during the third and fourth hours.

In the case of the patient cited above and who had his attack while walking shortly after breakfast, the attack passed off spontaneously with rest; this may be attributed to a rise in blood sugar level stimulated by the adrenal-sympathetic defense mechanism. No further pain was experienced during the rest of the day even though he engaged in arduous work. I would explain this by saying that during the rest of the day the blood sugar concentration became stabilized and was maintained at normal levels and so no further attacks occurred.

A few angina pectoris patients have attacks of unconsciousness accompanying the attacks of pain. It is difficult to explain the loss of consciousness on the basis of the coronary arterial changes. The term "syncope anginosa" was coined, but it explained nothing. A sharp fall in blood sugar may readily cause syncope as well as chest pain. Thus both the loss of consciousness and the chest pain may be readily explained by a single mechanism.

If not on the basis of progression of the coronary arteriosclerosis, how is one to explain the progressive worsening of angina pectoris as shown by the increasing frequency, longer duration, greater severity, and the occurrence of the attacks during rest and sleep? I believe that this aggravation of the disease can best be explained on the basis of an aggravation of the metabolic disorder involving the liver glycogen stores and the blood sugar level. The genesis of angina pectoris may be attributed to a depletion of liver glycogen with subsequent effect on the blood sugar level. The amount of glycogen stored in the liver will depend on the nutrition habits of the patient. If a patient discovers that his seizures come on soon after, or even during, a meal, he would surely be afraid to eat a hearty meal and so his nutrition would suffer with further depletion of liver glycogen. And when individuals suffer any kind of symptom which comes on during or soon after

a meal, instinctively they avoid what they consider to be "heavy foods" and to the layman "heavy foods" usually turn out to be the valuable protein foods such as meat and other animal foods. They then decide to eat "light foods" such as toast, cereals, baked potatoes, all high starch foods.

Chapter VII

CASE REPORTS

In this chapter I shall present representative case histories of patients seen by me in a charity clinic and in private practice. All of the patients complained of chest pain attributed to malfunction of the heart caused by abnormal blood sugar fluctuations and in all instances the pain was relieved by the change in diet. I shall present patients in the adolescent group first.

Case No. 1. E.C., a white boy 13 years old, was brought in by his mother because of two recent attacks of unconsciousness without convulsions. The first seizure occurred in school about an hour after lunch, lasted about 20 minutes and was followed by spontaneous recovery. The second seizure occurred one morning at 11 a.m. while playing ball. This attack lasted 15 minutes. He also had frequent headaches which came on while riding the school bus. He was nervous, restless, and "fidgety." He had frequent attacks of smothering sensations and tightness across the chest on exertion such as running. The physical examination showed no evidence of organic disease. A glucose tolerance test revealed: fasting blood sugar 80 mg., ½ hr. 95, 1 hr. 85, 2 hrs. 65, 3 hrs. 65, 4 hrs. 70, 5 hrs. 80. He responded readily to the low carbohydrate high protein diet with between meal feedings. He became less nervous, had better color, and gained weight. The attacks of chest pain gradually became infrequent and stopped altogether after several weeks.

Comment. This boy had attacks of chest pain which came on with exertion and which were readily prevented by a change in diet. In addition he had had two attacks of unconsciousness. Since there was no recurrence of the unconsciousness while on the low carbohydrate diet, and since there was no organic basis for them, it is very likely

that they were caused by the hypoglycemia. One encounters adult angina pectoris patients who are also subject to attacks of unconsciousness. Sometimes both the angina and the coma occur during the same episode, the pain preceding the coma. Or, the patient may fall unconscious and on regaining consciousness he may complain of chest soreness, or pain, and evidence of a severe heart attack may eventually be present. Most authors have been unable to satisfactorily explain the coma during such episodes. However, one may readily attribute both the coma and the chest pain to the fall in blood sugar.

Case No. 2. B.P., a white boy 14 years old, came into the clinic with his mother because of attacks of pain in the upper abdomen and lower chest which came on during exertion. He also complained of nervousness, dizziness and frequent headaches. The mother volunteered that he was "cranky," easily angered, and that he always wanted to eat, preferably something sweet. Physical examination revealed a thin, pale, poorly nourished, alert, somewhat restless boy. There were no signs of organic disease. A tolerance test revealed: fasting blood sugar 75 mg., ½ hr. 100, 1 hr. 90, 2 hrs. 60, 3 hrs. 60, 4 hrs. 75. He responded readily to a low carbohydrate diet and was soon free of the attacks of chest and abdominal pain. There was marked improvement in general health with loss of his nervousness.

Comment. An important complaint in this boy was his irritability. He was described as being cranky and easily angered. This is a group of symptoms often seen in hypoglycemic patients. It is a matter of common knowledge that people are irritable, easily provoked, and hard to deal with, when they are overhungry — all because of an abnormally low blood sugar. This is especially true in the morning before breakfast when the blood sugar usually reaches its lowest level. Many people are irritable and "out of sorts" on waking in the morning. They are not aware that this mental state is due to low blood sugar. As soon as they have eaten something they feel better and

the irritability passes off. Understanding mothers quickly learn to know that a crying baby is usually a hungry one. The low blood sugar in the infant is thus a mechanism to cause it to cry and thereby tell the mother that it is hungry. I have read about a well-known political figure who, because he is very grouchy in the morning on arising, never has breakfast with his wife because he fears he might say something unpleasant and perhaps start an argument. He does not meet with his wife until the noon meal when he is in a better mood. Marked hunger and irritability thus go together. As stated above, this served some useful function in the infant. I believe that this association is also useful to the predatory animals that live by killing other animals. I refer to the carnivorous lion and tiger which must be in a savage mood to kill when they are hungry. No doubt the blood sugar falls very low when these animals are in this state.

Case No. 3. T.F., a white 15 year old boy, was seen in the medical clinic complaining of pain in the chest on exertion, nervousness, weakness, and tremors. His mother said that he was doing poorly at school. He had been in an auto accident at the age of 6 and suffered a fracture of the skull. He had a nervous breakdown at the age of 12 which lasted 3 months. The pain in the chest occurred only on exertion such as running, jumping, and during gymnastics. The pain was felt in the left lower anterior chest region, sometimes in the right. Occasionally the pain was localized to the left costal margin and in the left upper abdomen and was called a "stitch in the side." The pain was sharp, fleeting, and passed off readily with rest. Physical examination revealed a thin, undernourished boy with no evidence of organic disease of the nervous system. A glucose tolerance test showed: fasting blood sugar 70 mg., 1 hr. 100, 2 hrs. 110, 3 hrs. 75, 4 hrs. 60. There was marked and rapid improvement on the low carbohydrate diet especially with regard to the nervousness and chest pain.

Comment. This boy complained of nervousness and tremors. The tremors are actually due to stimulation of the adrenal-sympathetic system which is responsible for the muscular contractions that appear objectively as tremors and subjectively as nervousness. The adrenal-sympathetic system is stimulated to activity by the fall in blood sugar since it is the protective mechanism that comes into play during an abnormal fall in blood sugar. The more rapid and greater the fall the stronger the stimulation, otherwise the individual is apt to die of extreme hypoglycemia. When strongly stimulated, the adrenal-sympathetic system causes a breakdown of glycogen in the liver with liberation of glucose into the blood and subsequent elevation of blood sugar levels. The tremor and nervousness are thus unpleasant side-effects caused by the essential adrenal-sympathetic activity.

It is interesting to speculate further on the origin of the tremors. During sudden exposure to cold, warm-blooded animals respond by shivering which is a series of rapid involuntary contractions of skeletal muscles. The rapid contractions produce body heat and so normal body temperature is maintained. When the body is warmed the shivering stops. Now, during a sustained fall in blood sugar to significant low levels, the body as a whole burns less sugar and so less heat is produced. As a result, the body temperature will fall below 98.6. Instead of the coarse shivering that occurs with a cold environment, there will be fine muscle contractions which appear as tremors.

Case No. 4. B.P., a white 15 year old boy, was brought in by his mother because of fainting spells, headaches, abdominal pain, and pain in the left shoulder on exertion. His mother volunteered that he was nervous, irritable, and over-active. He had had 3 attacks of unconsciousness during the preceding 18 months, each lasting from 3 to 5 minutes, with spontaneous recovery. The pain in the region of the left shoulder sometimes came on during rest or activity, would last 10 to 15 minutes and pass off

spontaneously. Physical examination revealed a thin, alert, high strung boy with no evidence of organic disease. There was no limitation of function or deformity in the region of the left shoulder. A glucose tolerance test showed: fasting blood sugar 65 mg., ½ hr. 75, 1 hr. 70, 2 hrs. 60, 3 hrs. 50, 4 hrs. 50. He responded favorably to the low carbohydrate diet. The attacks of chest pain and pain in the left shoulder disappeared after a few weeks. He gained weight and was no longer nervous.

Comment. The glucose tolerance test revealed moderately severe hypoglycemia during the 3rd and 4th hours. His attacks of unconsciousness may be readily attributed to sharp falls in blood sugar, as were the chest and shoulder pain. It is important to remember this association because it is not uncommon for an adult who is having recurrent attacks of angina pectoris to fall unconscious and, on regaining consciousness, to complain of residual soreness in the chest which is subsequently shown to be due to a heart attack and organic changes in the heart muscle. In such cases the fall in blood sugar embarrassed the heart muscle and the central nervous system simultaneously. Present day authorities on heart disease are unable to satisfactorily explain the unconsciousness in angina pectoris patients on the basis of coronary arteriosclerosis. However, if one accepts the concept offered in this monograph, both symptoms may be readily explained by a single mechanism.

Case No. 5. T.G., a 15 year old white boy, was seen in the clinic because of precordial pain, palpitation, and shortness of breath. These attacks came on suddenly, usually after a heavy meal and at rest. Sometimes the pain was felt in the right lower chest. He also had attacks of upper abdominal pain and nausea. The chest pain, palpitation, and shortness of breath, were aggravated by slight exertion and it was necessary to keep him in bed during these episodes. Physical examination revealed a thin, pale, anxious youth. The only abnormal physical finding was

an elevated blood pressure which varied from 140/80 to 150/100. A glucose tolerance test revealed: fasting blood sugar 75 mg., 1 hr. 90, 2 hrs. 80, 3 hrs. 60, 4 hrs. 70, 5 hrs. 80. He made dramatic improvement on a low carbohydrate diet with between meal feedings. The attacks of chest pain, palpitation, and shortness of breath ceased after a few weeks. He also gained weight and had a good color.

Comment. The rapid response to the change in diet in all these boys was dramatic and readily confirms the origin of the symptoms, especially the chest pain. Two of the boys complained of tightness across the chest just as adults with angina pectoris often do. This certainly points to a common mechanism producing the tightness in the younger group as well as in the older group. The chest pain in these boys was usually of short duration and disappeared readily on rest. Certainly there was every indication that the chest pain was due to some disorder of the heart, a disorder which could come on suddenly, persist for a short time, and then disappear completely. A disturbance in heart action that would fit this pattern very readily is one that could be due to a transient fall in blood sugar. The tendency to hypoglycemia was shown to be present in all the boys by the glucose tolerance test and the rapid relief from symptoms with the change in diet is further proof of their origin. In addition to the chest pain, these boys had other classic symptoms of hypoglycemia such as transient attacks of unconsciousness, nervousness, tremors, headache, dizziness, weakness, cold sweats, irritability, etc. These symptoms also responded to the change in diet.

These patients were seen in the charity clinic of a large city hospital during the depression years, 1935 to 1938. All of the families were on the relief rolls and all had to consume the cheaper starchy foods such as potatoes, cereals, rice, bread, spaghetti, etc. The more expensive protein foods like eggs, meat, dairy products, were eaten only occasionally. This high starch diet was no doubt responsible

for the chronic hypoglycemia. It has been shown by McCullagh and Johnston (2) that the continued ingestion of a high carbohydrate (sugar, starch) diet will produce a chronic hypoglycemia in otherwise normal individuals as demonstrated by the glucose tolerance test. If such individuals are then put on a low carbohydrate high protein diet there is a change in the tolerance test with disappearance of the hypoglycemic type of curve in the tolerance test. (Fig. 14.)

Thus chronic hypoglycemia is not to be regarded as a permanent disorder but rather as a disorder which follows the continued ingestion of a poorly balanced diet in that the carbohydrate portion (sugar, starch) is too large. Some of the boys were reported as doing poorly at school and were reported as being restless, nervous, and presumably inattentive. These symptoms could also be attributed to the effect of the hypoglycemia on the central nervous

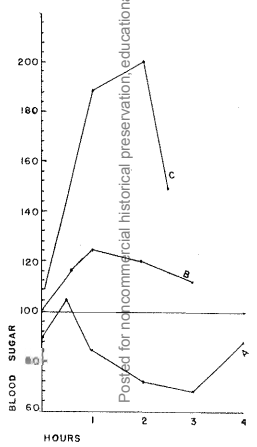


Fig. 14

Case No. 11. D.K. Curves to demonstrate the change in tolerance curve after 9 months on a low carbohydrate diet. Curve A shows course of the blood sugar after 100 grams of glucose at the start of treatment with a low carbohydrate diet, January 5, 1938. Curve C shows the change in tolerance to 100 grams of glucose after 9 months on the low carbohydrate diet, October 4, 1938. Curve B shows the course of the blood sugar after a low carbohydrate breakfast consisting of one orange, 2 eggs, 1 slice bread and butter, 4 oz. milk and 3 oz. cream (C 25, P 20, F 22). Values in curve B are all within normal range.

system, as was stressed in an earlier chapter. The nerve cells of the brain were literally being deprived of their proper fuel (sugar) necessary for their proper function. The nervousness, tremors, and restlessness, were actually due to the adrenal-sympathetic stimulation, as stated previously. Other symptoms and signs such as pallor, and any rise in blood pressure, and cold sweats, may be attributed to the vaso-constrictive action of the adrenal-sympathetic system.

I shall now present several case histories of young adults who complained of precordial pain, often accompanied by shortness of breath and palpitation, and coming on usually with exertion and sometimes at rest. All of these patients responded readily to the change in diet. I shall merely describe the symptoms and give the results of the glucose tolerance test.

Case No. 6. T.M., a white male, 18 years old, complained of nervousness, palpitation, precordial pain, tightness across the upper chest, and dizziness. The chest pain and palpitation would come on suddenly, last for 1 to 3 minutes, and disappear spontaneously. They would occur every 2 or 3 days. The tightness was described as a "sense of constriction" across the upper chest. During the preceding 8 months he had had 3 attacks of palpitation without chest pain. Each attack lasted for about 30 minutes. The physical examination showed no evidence of organic disease. He was a nervous "high-strung" individual. The glucose tolerance test showed: fasting blood sugar 65 mg., ½ hr. 75, 1 hr. 80, 2 hrs. 65, 3 hrs. 60, 4 hrs. 55.

Case No. 7. E.K., a white male 28 years old, complained of nervousness, difficulty in taking a deep breath at times, tightness of the chest and shortness of breath on moderate exertion such as climbing a slight incline. The physical examination was essentially negative. The glucose tolerance test revealed: fasting blood sugar 80, ½ hr. 100, 1 hr. 95, 2 hrs. 55, 3 hrs. 65, 4 hrs. 60.

Case No. 8. J.G., a white male 20 years old, complained

of chest pain in the form of substernal distress and shortness of breath which came on before and after meals and sometimes while in the reclining position. Moderately severe exertion such as playing football would cause attacks of weakness and palpitation. He was nervous and had a fairly marked tremor of the hands. The physical examination was essentially negative. The basal metabolism test was normal. The glucose tolerance test showed: fasting blood sugar 60 mg., ½ hr. 100, 1 hr. 70, 2 hrs. 75, 3 hrs. 35, 4 hrs. 60.

Case No. 9. S.F., a white male 32 years old, complained of a precordial ache and dull pressure accompanied by difficulty in breathing only when he climbed stairs or ran very fast. These attacks would last several minutes and pass off on rest. The physical examination was essentially negative. The glucose tolerance test revealed: fasting blood sugar 75 mg., ½ hr. 95, 1 hr. 80, 2 hrs. 70, 3 hrs. 60, 4 hrs. 60.

The following cases are representative of the large number of older patients with the classic symptoms of angina pectoris. These are reported in detail. Probably all of the patients in this group had some degree of coronary arteriosclerosis.

Case No. 10. J.L., a white man aged 46, a garment worker, was first seen by me in the medical clinic in July 1937 shortly after having been discharged from the hospital with a diagnosis of angina pectoris and high blood pressure. While his health had never been robust, he had had no major illnesses. He was in apparent good health until May 1937, when he began to have severe headaches. He then learned that he had high blood pressure. He soon began to have attacks of shortness of breath on climbing stairs and during sleep.

When I saw him for the first time in July 1937, he complained of headaches, dizziness, shortness of breath, and recurrent attacks of sharp sticking pain in the left chest which came on during exertion such as climbing

stairs or walking up a hill. The pain would disappear after 5 minutes rest. The pain radiated to the left armpit, down the left arm to the elbow, and to the right chest. Physical examination revealed a moderate elevation of the blood pressure which was 160/100. The heart sounds were of good quality and the rhythm was regular. On December 15, 1937, a glucose tolerance test showed: fasting blood sugar 85 mg., ½ hr. 110, 1 hr. 145, 2 hrs. 110, 3 hrs. 70, 4 hrs. 65. He was put on a low carbohydrate diet with between meal feedings. He returned on January 7, 1938, stating that he felt stronger and had less headache and dizziness. The attacks of chest pain were fewer and milder and lasted for about a minute. His sleep was sounder. His face appeared fuller, relaxed, and had a better color. On February 8, 1938, he reported a gain of 8 pounds. This was gratifying because he was underweight and poorly nourished. The blood pressure was 146/108. On March 18 he reported freedom from chest pain. The blood pressure was 140/100. On April 19 he said that he had had no pain during the preceding 4 weeks. He had gained 15 pounds after 14 weeks on the diet. Improvement was steady and sustained throughout the rest of 1938. Once, on September 20, he reported fleeting pains under the left costal margin occurring several times during one week. He continued on the diet all during 1939 and made 13 visits to the clinic during the year. He reported some slight precordial pain on only 2 of these visits. Headaches and dizziness occurred only occasionally and were not remarkable. He was able to work without fatigue and engage in his usual activities. After April, 1939, the blood pressure, which until then had shown moderate elevation, began to be more nearly normal. During 1940 his visits were of a routine nature. He did not complain of any pain, shortness of breath, or palpitation on any of these visits. Headaches and dizziness occurred occasionally and were mild. The blood pressure record of this patient is shown in table No. 3.

TABLE NO. 3

Blood Pressure Readings, Case No. 10

1937		1938		1939		1940		1941	
6/26	160/114	2/8	146/108	1/24	130/90	1/23	124/88	1/23	110/70
7/9	156/110	3/19	140/100	2/28	146/100	2/20	126/90	4/10	130/96
9/8	156/100	4/19	130/100	5/2	120/80	4/10	136/100	5/20	120/80
11/5	145/98	9/20	156/110	8/15	115/80	7/2	126/70		
12/15	put on diet			9/5	120/80	10/15	110/80		
						11/7	120/80		
						12/5	112/80		

Comment. This patient's headaches and dizziness became milder after a few weeks and practically disappeared after a few months, in spite of the fact that there had been no appreciable fall in blood pressure. During the last 6 months of 1938 he had virtually no headaches and dizziness, yet the blood pressure was still elevated. This is emphasized because it suggests that headache and dizziness, even when present in a hypertensive patient, may not be due entirely to the hypertension. His blood pressure after January 1, 1939, except on one visit, was always found to be within normal limits.

Case No. 11. D.K., a white man, aged 47, was first seen on June 10, 1937, complaining of precordial and epigastric pain. The precordial pain was sharp, radiating to the left shoulder and down the left arm to the fingers. The pain would come on during sleep, before and after meals, and on walking. Excitement also brought on attacks. The epigastric pain was described as "cutting" and "gripping," was localized, and was often accompanied by nausea and salivation. At times the precordial pain and epigastric pain would come on together and disappear together. At other times he would have one type of pain without the other.

His first attack of precordial pain occurred in January, 1936. He was awakened from sleep at 3 o'clock one morning by a severe pain across the entire chest, radiating through the back between the shoulder blades, and down the left arm. He was unable to take a deep breath. Severe

pain persisted for 24 hours. He was hospitalized at another institution. The clinical diagnosis was coronary thrombosis although the electrocardiogram showed no abnormality. He left the hospital after 10 days against the advice of physicians. About six weeks later he tried to go to work as a truck driver. He had such precordial pain and weakness on even slight exertion that he soon quit work. Walking short distances, such as 5 blocks, caused pain all during the summer of 1936. In October, 1936, he had another severe attack, waking him from sleep at 3 a.m. This attack was not as severe as the previous one, and he was in bed for only 3 days. He went back to work soon after this illness but had to quit after a few days because of pain on moderate exertion. The attacks now became more frequent. He would have them almost daily. Occasionally he was free of them for 1 to 3 days at a stretch. The pain also occurred during rest, 1 to 2 hours after meals, lasted 5 to 10 minutes, radiated to the back, the left shoulder, and down the left arm to the elbow. Taking a deep breath during an attack aggravated the pain. He also had mild bouts during sleep.

Physical examination on June 10, 1937 revealed a well developed, well nourished individual with sthenic habitus. The heart was not enlarged and the sounds were normal. The blood pressure was 138/80. The electrocardiogram was normal. He was treated with various drugs with indifferent results. The attacks occurred almost daily and usually on exertion. A glucose tolerance test on January 5, 1938 showed: fasting blood sugar 90 mg., ½ hr. 128, 1 hr. 85, 2 hrs. 70, 3 hrs. 65, 4 hrs. 90. He was put on a low carbohydrate diet with extra feedings. All medication was stopped. He returned on January 18 stating that the attacks of pain were milder, of shorter duration, and occurred about 3 to 4 times a week. This improvement occurred in spite of inability to follow the diet strictly because he could not afford the more expensive proteins. By cutting down the intake of carbohydrate-rich foods and

increasing proteins somewhat, together with feedings between meals, he was able to reduce the number and severity of attacks. On February 17 and on March 18 he reported further improvement with respect to the precordial pain and said that the epigastric pain had disappeared. On April 21 he reported complete freedom from precordial pain and epigastric pain during the preceding three weeks. He was now able to follow the diet more closely. On May 10 he reported having 2 mild attacks of precordial pain since the visit on April 21. He improved steadily thereafter. On September 8 he said he had had no precordial pain since May. Since he felt so well, he went off his diet somewhat and was eating 2 slices of bread with meals and was taking sugar with his coffee. During 1939 he was virtually symptom-free.

On May 3, 1940 he reported having short bouts of pain. Questioning revealed that he was eating bread, sugar and other carbohydrate foods in liberal quantities. The recurrence of symptoms could have been attributed to carelessness with his diet. He was told to omit bread and sugar completely. He did so and became symptom-free after 2 weeks. He was free of pain when seen during July, August, September and October, 1940. On November 26 he reported mild precordial pain of a few days' duration. He admitted taking sugar with his coffee. He was again advised to adhere strictly to the diet. He did so and became symptom-free once more. When last seen in January, 1941 he said he was not having any pain.

Another symptom which greatly improved in this patient was headache. He had been having migraine headaches ever since his discharge from the army in 1918. The headaches were severe, painful, and disabling. They lasted 10 to 12 hours and forced him to go to bed. They were always accompanied by nausea, vomiting and weakness. Headaches came at monthly intervals, sometimes occurring 2 to 3 times a month. After going on the diet they occurred only once a month, were less severe and

less disabling. During 1939 they still occurred at monthly intervals but were definitely milder and shorter in duration. During the first 6 months of 1940 they occurred only once a month. During the last 6 months of 1940 he was entirely free of them for the first time in 22 years.

The patient was always overweight. In 1938, before going on the diet, he weighed 160 pounds, about 22 pounds overweight. After 3 years on the diet, he lost 22 pounds and reduced his waistline by several inches.

Comment. This patient illustrates the need for more or less strict adherence to the low carbohydrate diet. Whenever he felt well and was free of precordial pain, he thought he was cured and would then eat bread, sugar, and other carbohydrate foods. The attacks of pain promptly recurred. When he went back to his diet the attacks disappeared. Patients thus find out for themselves that the ingestion of foods containing sugar and starch cause the attacks of pain.

Case No. 12. A.S., a white man aged 69, was first seen on August 17, 1937, complaining of precordial and epigastric pain, severe headaches and dizziness. His general health had always been good until the summer of 1936, when he began to get bouts of pain on exertion and during excitement. Often the precordial pain was accompanied by epigastric pain. The attacks gradually increased in severity and frequency so that by the summer of 1937 they occurred almost daily after walking 1 to 2 blocks. The pain was described as severe and "sticking," was usually localized to the precordium, but sometimes radiated to both shoulders. Rest would afford relief after 2 or 3 minutes. The headaches were severe and sometimes lasted for days. Sharp frontal pains were often present. The attacks of dizziness came on almost any time and sometimes made him walk as though drunk.

Physical examination revealed a well developed, well nourished, nervous, florid individual. The speech was thick and stuttering. The heart was slightly enlarged. The

blood pressure was 250/110. He was given sedatives and dilator drugs without relief. His symptoms continued much the same during 1937. His blood pressure remained elevated and the systolic pressure always was above 200 mm. of mercury (table No. 4). A glucose tolerance test on January 5, 1938 showed fasting blood sugar 50 mg., ½ hr. 100, 1 hr. 130, 2 hrs. 85, 3 hrs. 65, and 4 hrs. 40. He was put at once on a low carbohydrate diet with extra feedings. He returned on January 13, 1938, stating that he was unable to follow the diet because he could not afford the more expensive proteins. Arrangements were made with the relief bureau to give him extra money to buy these foods. With this help he was able to follow the diet, though not completely.

He showed improvement within 2 weeks. The precordial pain became milder and occurred less often. He was able to walk greater distances. The epigastric pain disappeared after 3 weeks. Headaches and dizziness gradually disappeared. On April 12 he reported practically complete freedom from these symptoms. The blood pressure was 160/90. On April 19 he reported "rare pain," occasional headache, and no dizziness. The blood pressure was 156/90. On May 3, 1938, he reported occasional headache and said he had "no more pain at all." The blood pressure was 170/90. On all visits to the clinic after May 3, 1938 he reported freedom from precordial and epigastric pain. He had only occasional headache and dizziness. All blood pressure readings after January 27, 1938 showed systolic readings under 200 mm. with proportionate diastolic readings. He felt so well during 1939 that it was necessary for him to come to the clinic only twice. During 1940 he came in only once. When last seen on January 21, 1941 he reported occasional mild pain which came on while walking. He said he could walk up to 8 blocks and return before feeling any pain. He also got mild pain during excitement. Questioning revealed that he was eating some bread, sugar, and now and

then potatoes. He said that since he was feeling so well he thought he could be more liberal with these foods.

TABLE NO. 4

Blood Pressure Readings Case No. 12

1937		1938		1939		1940		1941	
8/17	250/110	1/6	248/120	2/21	180/80	2/7	170/80	1/23	160/100
8/31	200/95	1/13	put on diet			5/23	190/90		
9/16	225/110								
10/10	210/110	1/27	208/100						
10/26	210/100	2/21	160/90						
11/30	220/110	3/17	190/90						
12/30	230/110	4/7	200/100						
		4/12	160/90						
		4/19	156/90						
		5/3	170/90						
		5/26	166/90						
		6/30	180/90						
		7/5	170/80						

Comment. This case shows that virtually complete relief from angina pectoris may be obtained even in an elderly arteriosclerotic with high blood pressure. Improvement was apparent after 2 weeks. He was virtually symptom-free after 3 months in spite of inability to follow the diet strictly. An indication of improvement was the drop in the number of visits to the clinic during 1939 and 1940. Within a few weeks after going on the diet the blood pressure fell and remained below the original high levels.

Case No. 13. W.C., a white man aged 54, unemployed, was first seen on October 25, 1938 complaining of daily attacks of "warm pressure sensations" over the entire chest coming on at rest and while walking and doing housework. He had had seizures several times daily with every attempt at walking since February, 1938. His previous health was good.

The onset of chest symptoms had been sudden one day in February, 1938, while walking in the snow. Soon after this episode, seizures came on regularly after walking 1 to 2 blocks. A typical attack would begin with an uncomfortable distressing pressure sensation in the pit of the stomach, a "melancholic sensation" in the left upper ab-

domen, soon followed by a squeezing vise-like tension over the entire chest, reaching to the root of the neck. There was never any sharp radiating pain. He would stand still during an attack, nervous and apprehensive, with both arms held rigid by his sides. He was unable to talk during an attack. He always tried to belch because it relieved him somewhat of the epigastric distress. Seizures would last from 5 to 10 minutes and leave him exhausted and discouraged. He would always return home, fearing to continue further. Because of these seizures he was afraid to venture forth alone and had his wife accompany him. Similar seizures, although shorter, often came on when he assumed the recumbent position, day or night. He had no headaches or dizziness. He said his head felt "foggy" all the time. Almost every morning on arising he would have nausea without vomiting. He would retch for several minutes until he felt better. There would be no desire for food for some time. His sleep was poor and restless. He would lie in bed fearful and apprehensive of nothing in particular, and would get up and walk around the room.

Physical examination on October 25, 1938 showed a florid, well nourished, well developed, apprehensive individual. The heart was enlarged and the sounds were of good quality. The blood pressure was 182/90. He had been treated with phenobarbital, theobromine, and other drugs for about a year without improvement.

I did not see him again until September 21, 1939. Physical examination showed nothing new and the history revealed that the symptoms were getting worse. The blood pressure was 152/90. A glucose tolerance test showed fasting blood sugar 60 mg., $\frac{1}{2}$ hr. 100, 1 hr. 110, 2 hrs. 90, 3 hrs. 70, 4 hrs. 60. He was put at once on the diet with extra feedings. He returned on October 5, 1939, stating that he had had only 1 attack on walking since September 21 and that he was beginning to feel less nervous and irritable. The blood pressure was 140/80. On January 25, 1940 he reported sustained and steady improvement. He could walk up to 5 blocks without discomfort. His morning attacks of nausea were becoming less frequent and his sleep was sounder. The blood pressure was 142/70. On April 25 he said he could walk up to 6 blocks. The seizures had gradually become milder and lasted only 2 to 3 minutes. He was no longer nervous, apprehensive, and exhausted with these attacks. He was able to speak during a seizure and no longer held his arms rigid by his sides. Whereas he formerly would get discouraged and exhausted and would return home afraid to venture further, he would now recover quickly, and once over a seizure he could walk indefinitely. He was now able to do household duties without discomfort. He was no longer afraid to go out by himself. On July 6, 1940 he said he could walk as much as 10 blocks and return. He felt stronger and had a good appetite. He was less nervous and irritable. He had no more seizures on assuming the recumbent position. His sleep was sounder. The blood pressure was 130/60. After 10 months on the diet he had lost 7 pounds. On August 22 the blood pressure was 142/60. He could walk up to 10 blocks, and the seizures after such a distance were much milder and of shorter duration. Once the seizure was over, he could walk indefinitely. He had complete confidence in himself and was not afraid to venture out by himself. On December 12 he reported sustained progress. The blood pressure was 140/70. When last seen on January 23, 1941, in response to a request by mail, he said he could walk up to 10 blocks, get a short mild seizure, and then be able to continue indefinitely. Occasionally he would get a mild seizure after 2 blocks. He admitted eating potatoes about twice weekly and taking two slices of bread with meals.

Case No. 14. G.S., a white man aged 53, was first seen by me in March, 1938, complaining of precordial pain, shortness of breath, weakness and drowsiness after meals, and frequent night sweats. He had been attending various dispensaries for several years, receiving various drugs for

his symptoms but without results. Only nitroglycerin afforded some relief during attacks of pain.

His attacks of precordial pain began in 1932. From the onset they occurred several times daily and persisted with this frequency until 1938. They occurred on arising in the morning between 5 and 6 a.m., with every meal, and on exertion. The pain was severe, vise-like, and usually localized to the precordium. At times the pain was felt at the root of the neck and sometimes a seizure would begin with a sensation of "soreness at the root of the tongue." Sometimes the precordial pain radiated down the inner border of the left upper extremity to the wrist. The early morning attacks were severe and lasted from ½ to 1 hour. Attacks came on almost regularly with meals. Some occurred in the midst of a meal and were accompanied by nausea and vomiting. Vomiting always brought relief from the pain. Weakness and a drenching sweat would usually follow such episodes. Having vomited, his appetite would return and he would finish his meal. Walking a quarter or half of a city block, and moderate exertion at work, would invariably cause pain, which would disappear after a few minutes' rest. Sudden changes in environmental temperature, such as going from the warm boiler room to a cooler room, or turning on the cold water after a warm shower, would cause pain. Physical examination in March, 1938 revealed a well nourished, well developed, florid individual. The heart was enlarged. The blood pressure was 220/50. A loud diastolic murmur was heard over the aortic area and at the apex. A glucose tolerance test on March 6, 1938 showed fasting blood sugar 65 mg., ½ hr. 130, 1 hr. 130, 2 hrs. 75, 3 hrs. 60. He was put on the low carbohydrate diet on March 8 with between meal feedings. On March 22 he reported that he felt stronger, was less drowsy after meals, had more "pep," and felt "more lively." The attacks of pain were no longer present on arising, and no longer occurred with breakfast and lunch. He still had pain on exertion and about 15

minutes after supper. On March 29 he reported that the pain on exertion was milder and of shorter duration. The night sweats were becoming less frequent. On April 19 he said he still had some pain after the evening meal; however, the pain which formerly came on about 15 minutes after he finished eating and which would last for almost an hour now came on about 3 hours after the meal, lasted for a few minutes and was much milder. The blood pressure on April 19 was 140/50. On May 3 he reported having about 2 bouts of pain weekly, usually after supper while at rest. He had no pain on exertion. He could now walk 5 to 6 blocks without pain or other discomfort. He improved steadily. He felt so well that he did not find it necessary to come back until November 1, 1938. He said that he had been practically symptom-free since the last visit, May 3. He said that on the day before, October 31, he had some pain about 2 hours after supper. Questioning revealed that he was taking 2 or more slices of bread with his meals. He was told to limit himself to a single slice. The blood pressure on November 1 was 148/60. He returned on November 15, December 6, 1938 and January 29, 1939, reporting complete freedom from pain. On January 29 the blood pressure was 170/60. On March 14 he came in because of a cold. He had no pain. On September 26, 1939, he said he had had about 6 bouts of pain during the preceding 16 months, and that he had been able to walk as much as 3 miles without pain or other discomfort. On January 21, 1941 he reported that during 1940 he had had no attacks of pain with meals or on exertion, and that he was able to walk indefinitely. He felt strong. He had slight pain on arising between 5 and 6 a.m. These attacks occurred infrequently. Questioning revealed that he was taking sugar with his coffee. He was told to omit bread and sugar completely. The patient was about 20 pounds overweight when first put on the diet. During the 3 years on the diet he lost 15 pounds. His abdomen, which was always

very prominent, flattened down considerably. The blood pressure on January 21, 1941 was 160/46. There was no change in the diastolic murmur.

Comment. This patient had bouts of pain at rest, during or shortly after meals, on exertion, and with sudden change to a cold environment. The fact that the diet afforded relief from the attacks which occurred under such different circumstances suggests that a fundamental etiologic factor was being overcome. Further, it is customary to attribute the pain in patients with aortic insufficiency to the valvular defect. The response in this patient certainly suggests that the valvular defect was not an important factor in the causation of pain.

Discussion. All of the angina pectoris patients who have been treated with the low carbohydrate diet have reported improvement not only with regard to pain, both precordial and abdominal, but also with regard to general health and well-being. In no case was a rise in blood pressure observed on the high protein intake. On the contrary, any changes observed were always in a downward direction.

Evidence that the syndrome of angina pectoris may be produced by absolute and relative hypoglycemia has accumulated from various sources. Soskin and co-workers (17) induced hypoglycemia in elderly diabetics with cardiovascular disease by means of insulin. By this method they produced both objective and subjective signs of the anginal syndrome. All symptoms were relieved by glucose administration. The electrocardiographic changes were similar to those noted by Feil and Siegel (18) during spontaneous attacks of angina and to those noted by Middleton and Oatway (19) during insulin hypoglycemia, although the blood sugar was not lower than 70 mg. Soskin, in discussing the work of Middleton and Oatway, stated, "It seems likely that the pathological heart requires a greater amount of carbohydrate than the normal heart. This may account for the results of Middleton and Oatway on insulin without hypoglycemia since the blood

sugars of their patients, although not abnormally low for the normal heart, may represent a state of relative hypoglycemia for the diseased myocardium." Parsonnet and Hyman (20) observed the development of the stenocardial syndrome in diabetic patients with old coronary thrombosis following doses of insulin that did not produce hypoglycemia.

That sudden lowering of the blood sugar by insulin in diabetics may cause anginal attacks and cardiac infarction has been reported several times. Joslin (21) reports cardiac infarction following insulin with a blood sugar of 30 mg. Blotner (22) reported 2 cases in which infarction occurred so soon after insulin as to suggest that the two were causally related. Hetenyi (23) cites 2 cases of diabetes associated with angina pectoris in which anginal attacks appeared to be induced by insulin. Ernstene and Altschule (24) cite reports by Gigon, Reinwein, and Van Noorden and Isaacs, all of whom observed insulin to cause death or aggravation of cardiac symptoms in the presence of heart failure. Modern (25) and Turner (26) reported cases of heart pain due to insulin, and Sippe and Bostock (27) and Rosenblatt (28) reported cases of chronic hypoglycemia with the anginal syndrome which responded to treatment with dietary measures aimed to overcome the hypoglycemia.

A low carbohydrate diet with between-meal feedings is beneficial in the management of both chronic hypoglycemia and chronic hyperglycemia because it raises the general level of the blood sugar in the former and does away with the wide fluctuations in the latter. As a result, the myocardium is assured of adequate deliveries of glucose at all times and is not exposed to sudden spontaneous lowering of the blood sugar.

In some patients the anginal seizure tends to occur at approximately the same time of the day and night. Figure 11 shows the course of the blood sugar over a 20-hour period in a patient who had his seizures almost regularly between 4 and 5 p.m. and 2 and 6 a.m. At no time during

the 20-hour period was the blood sugar observed to be below 90 mg. While 90 mg. is not absolute hypoglycemia, for a mild diabetic with a damaged myocardium one may say it represents a relative hypoglycemia. This patient had a severe heart attack with infarction 7 weeks before these blood sugar studies were made. The occurrence of pain coincides with a relatively sharp fall in blood sugar concentration to the lowest levels of the day and night. This patient responded at once to a low carbohydrate diet. He has been able to carry on full activities on such a diet.

Case No. 15. The following observations on a patient with typical angina pectoris are reported in some detail since they give a good clue as to what happens when a patient suffers an attack of angina. A.Z., a white man aged 55, a baker, complained of frequent attacks of precordial pain radiating to the interscapular region, the left shoulder and elbow, of 5 months' duration. The attacks came on during meals, while at work, and at rest during the day and during sleep. The seizures were accompanied by anxiety and marked weakness. He finally had to stop working. Physical examination revealed a normal heart. The blood pressure was 178/110. During the glucose tolerance test (Fig. 15) the following observations were made:

8:00 a.m.	Fasting blood sugar 90 mg. and 100 gms. glucose given by mouth.
9:00 a.m.	Blood sugar 120 mg. patient comfortable.
10:00 a.m.	Blood sugar 105 mg. patient comfortable.
11:00 a.m.	Blood sugar 96 mg. patient comfortable.
11:35 a.m.	Patient suddenly complained of precordial pain, tremors, sweating, palpitation, weakness. He was pale and apprehensive. The blood sugar was 63 mg. He was given some orange juice. The symptoms passed off in about 20 minutes.
12:00 noon	Blood sugar 125 mg. patient comfortable.

These observations are most revealing. This patient was comfortable during the first three hours of the test. Then there was not only a swift fall but a fall to hypoglycemia when the seizure occurred, which was in no wise different from his usual attacks of angina pectoris.

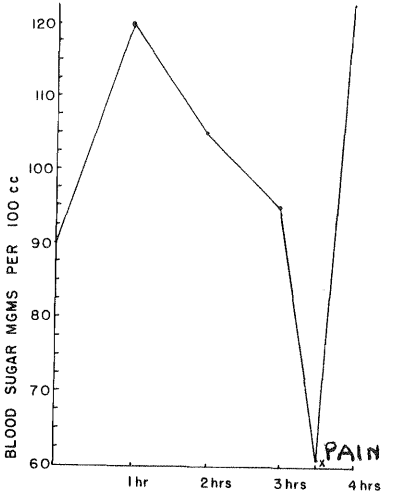


Fig. 15
Course of the blood sugar during glucose tolerance test on patient A.Z. who had a sudden attack of precordial pain 3½ hours after the ingestion of glucose. The attack of pain coincided with the sharp fall in blood sugar which occurred at 3½ hours. Following the ingestion of orange juice the blood sugar rose and the attack passed off.

During the course of these studies I treated a group of patients who were hospitalized for acute cardiac infarction with a dietary regime designed after the concept that the cause of the angina is due to unstable blood sugar levels with a relatively sudden lowering of the blood sugar to absolute or relative hypoglycemic levels. The diet was the same as described above in the other case histories. The hospitalized patients were put on the diet within 6 hours after the heart attack. The patients tolerated the diet extremely well and ample amounts of protein were permitted. If a patient was unable to eat much food at a time, he was given frequent small feedings of high protein foods. The patients on such a regime were far more comfortable than patients with heart attacks whom I have observed on other dietary regimes, all of which allowed carbohydrate-rich foods.

Chapter VIII

THE DIET

The various foods we eat supply the body with three kinds of foodstuffs: proteins, fats, and carbohydrates. In general, animal foods (beef, pork, fish, poultry, dairy products, eggs) supply proteins and fats, with small amounts of carbohydrate in the form of glycogen. Grains, vegetables, and fruits supply mainly carbohydrates with varying amounts of protein and fat.

Protein constitutes by far the greater part of animal tissues and it is essential that the human diet contain adequate amounts of good protein. Proteins are essential for growth, repair, replacement, the production of immune bodies needed to combat infection, the production of hormones, enzymes, and digestive juices. Life without protein is impossible. The importance of protein is suggested by the fact that the term is derived from the Greek and means "of first importance."

All proteins are made up of carbon, hydrogen, oxygen, and nitrogen. Some contain, in addition, iron, phosphorus, or sulphur. Proteins are usually classified as high-grade proteins, which are those of high biological value, and low-grade proteins, which are those of low biological value. Proteins of high biological value are found in animal foods. High biological value proteins more nearly resemble the proteins of human tissues in chemical make-up than do the proteins of lower biological value. Obviously, it is more advantageous to eat animal protein than plant protein.

Carbohydrates consist of carbon, hydrogen, and oxygen, and are produced by all plant life from carbon dioxide and water under the influence of sunlight, a process called photosynthesis. Under the general heading of carbohydrates we distinguish sugars, starches, and forms which are

neither sugar nor starch since they do not react specifically to chemical tests for sugar and starch. This distinction is extremely important because there is evidence that only sugar and starch can cause low blood sugar. Carbohydrate foods such as carrots, lettuce, tomatoes, cabbage, which contain no sugar and no starch do not cause low blood sugar. This difference in behaviour is due, no doubt, to a difference in chemical structure.

Fats consist of carbon, hydrogen, and oxygen, and chemically are made up of glycerol in combination with stearic, palmitic, and oleic acids. Fats are found in all animal foods, dairy products, eggs, some grains, vegetables, and fruits. Fats do not have any depressant effect on the blood sugar and so there are no restrictions in their consumption. Fats may be eaten, therefore, in any quantity according to individual taste and tolerance. In humans, fats are less responsible for obesity than sugar and starch. In those persons with a tendency to obesity all excess sugar and starch consumed is readily converted to fat and stored as such throughout the body. I have observed individuals for years on a diet of protein, fat, and little or no sugar and starch, who have maintained normal weights in spite of increased fat intake. Livestock are fattened for market chiefly by feeding them corn, grain, feeds, all of which contain large amounts of starch.

At this point I should like to acquaint the reader with a most interesting phenomenon which also gives us a clue to the foods that Nature intended man and other animals should eat and the foods that he should not eat. Biochemists speak of the "optical activity" of amino acids, the end products of protein digestion, and of the monosaccharides (sugars), the end products of the digestion of sugar and starch. Solutions of amino acids and of the various monosaccharides have the property of rotating a beam of plane-polarized light to the right or to the left. Those amino acids and sugars that rotate the beam to the right are called dextrorotatory, and those that rotate the beam to the left

are called levorotatory. Most of the sugars derived from the digestion of the sugar and starch that we eat are dextrorotatory. Only a few sugars are levorotatory. The commercially prepared dextrose got its name because it is dextrorotatory. It is otherwise with the amino acids obtained from the digestion of the proteins we eat. *They are all levorotatory! Dextrorotatory amino acids are not found in nature.*

This is most interesting because from clinical observations made by studying the effects of various foods in patients, and from a study of the blood sugar effects made by these same foods in the same patients, I have concluded that the foods that had adverse effects, both clinically and biochemically, were the same foods that always yielded dextrorotatory chemicals! The foods that were found to be beneficial to the patient, both clinically and biochemically, such as the proteins, are known to yield only levorotatory amino acids, never dextrorotatory amino acids. Thus some biochemists speak of the dextrorotatory amino acids as "unnatural," implying that the body prefers and wants only the natural or levorotatory amino acids. As a matter of fact, the proteins we eat yield only levorotatory amino acids, never the dextrorotatory.

I have concluded that the body prefers only those foods that yield levorotatory end products. If one were to pick foods after this principle, one would eat only proteins, fats, and non-starch, non-sugar carbohydrates. One could eat carbohydrates that yield levorotatory sugar, but these are few in number. For example, I have found that moderate quantities of honey can be tolerated bv the human apparently because honey is largely made up of a levorotatory sugar called levulose.

Therefore, it appears clear that Nature apparently intended for all animal life, carnivorous and herbivorous, to consume only those foods that yield levorotatory end products. All the foods that yield dextrorotatory end products are not intended for animal consumption.

General Diet Instruction For Patients With Heart Disease. I advise that for the best results in the treatment of a patient with recurrent heart pain and for the prevention of the heart attack, all foods containing sugar and starch should be avoided. If this is impractical, because of the financial inability to purchase the more expensive protein foods or because the proteins are not available in sufficient quantity, then I advise the complete elimination of sugar and the reduced consumption of foods containing starch. Sugar *is the more important factor in the causation of heart disease.* The sugar and starch are not only responsible for the adverse effects on the blood sugar level, but are also responsible for the arteriosclerosis as well. Arteriosclerosis may be an aging process. If it is, then one should see it only in the aged, in people past 80 years, let us say. Certainly one should not see any arteriosclerosis, even of mild degree, in any individual below the age of 40. Yet, many young adults and occasionally children have shown mild to moderate degrees of the degenerative sclerotic changes, involving the coronary arteries as well as other arteries.

What is the cause of this disturbance in fat and cholesterol metabolism leading to this abnormal vascular sclerosis? I believe the answer is given to us by the diabetic who is generally a victim of arteriosclerosis at an earlier age than the non-diabetic. Arteriosclerosis in the diabetic is more severe, more extensive, and comes at an earlier age, than in the non-diabetic. It has been attributed to the faulty fat metabolism with high blood cholesterol which is *secondary* to the disturbance in carbohydrate metabolism. It has been well stated that "Fats burn in a carbohydrate flame." In a diabetic controlled by diet and insulin this secondary disturbance in fat metabolism is corrected very readily. In the uncontrolled diabetic the disturbance in fat metabolism is manifest in several ways, especially by the early degenerative vascular sclerosis.

What is the cause of the arteriosclerosis in the non-diabetic? Well, one can readily implicate the same causal

mechanism which operates in the diabetic, namely, a disturbed carbohydrate metabolism in the form of a chronic hypoglycemia. In chronic hypoglycemia, there will be several hours out of the 24 hours when the blood sugar will be delivered in abnormally low amounts to the various tissues of the body, including the wall of the arteries. The net effect on the body will be similar to the net effect in the presence of diabetes, namely, a reduced sugar oxidation. And so, all the secondary metabolic disturbances which follow in the wake of a deranged carbohydrate metabolism of the diabetic, such as the deranged fat metabolism, will be found in the non-diabetic with chronic hypoglycemia. Acidosis, with acetone in the urine, can be found in the non-diabetic with chronic hypoglycemia just as it can be found in the uncontrolled diabetic with chronic hyperglycemia. The final common effect in both diabetic hyperglycemia and hypoglycemia is the same, namely, a reduced sugar-oxygen consumption. Of course, intrinsically, the final common effect is more severe and more serious in the diabetic because of the inherent nature of the disease.

Specific Dietary Instructions. The following foods should be avoided: sugar, soft drinks, ice cream, ices, sherbets, cakes, candies, cookies, wafers, pastries, pies, fruit juices, canned and preserved fruits, jams, jellies, marmalades, puddings, custards, syrups.

Coffee, tea, cocoa, lemonade, etc., may be sweetened with saccharin or other artificial sweetener. So-called "diabetic foods and desserts" and food preparations may be used. Nuts may be eaten in unlimited quantity except the starchy ones such as peanuts, cashews, chestnuts. These may be eaten sparingly.

The following foods should be eaten in reduced quantity because they contain starch:

beans, dried	tapioca	rolls		
beans, lima	macaroni	crackers		
corn		spaghetti		cereals—
peas, dried split		vermicelli		oat preparations
potatoes, white or sweet		noodles		rice preparations
yams		breads		rye preparations
lentils		buns		corn preparations
rice		biscuits		wheat preparations

The following fresh fruits should be eaten only in limited quantity because of their sugar content:

oranges	cantaloupe	pineapple	raspberries
grapefruit	watermelon	strawberries	grapes
peaches	apples	blueberries	cherries
honey dew melons	pears	blackberries	plums

Fresh fruits are allowed but only one portion should be taken with a meal, i.e., one apple or one orange. The sugar in fruits may cause low blood sugar if they are eaten in excess. Fruit juices, canned fruits, dried fruits, preserved fruits, should be avoided. Fruits may be stewed without sugar. Apples may be baked without sugar. Tomato juice is allowed since it contains no natural sugar.

The following carbohydrate foods contain little or no starch and may be eaten in unlimited quantity:

artichokes	celery	mushrooms	spinach
asparagus	chard, swiss	okra	squash, summer
avocados	collards	onions	tomatoes
bamboo shoots	cucumbers	parsley	turnips
beans, string	eggplant	parsnips	watercress
beans, wax	endive, leaves	peas, fresh	pickles
beans, soy	greens, beet	peppers	horseradish
beets, red	greens, dandelion	pumpkins	mustard
broccoli	greens, turnip	radishes	vinegar
brussels sprouts	kale	rhubarb	olives
cabbage	kohlrabi	rutabagas	capers
carrots	leeks	sorrel	mayonnaise
cauliflower	lettuce		

All animal foods may be eaten in unlimited quantity. Such foods are: beef, pork, lamb, mutton, veal, poultry, fish. These may be purchased fresh, canned, smoked, dried, etc. Eggs can be used freely whether fresh or dried. All dairy products may be eaten in unlimited quantity: milk, buttermilk, fermented milk, butter, sweet and sour cream, and all cheeses. Milk may be fresh, evaporated, or powdered.

SUGGESTED MEALS

BREAKFAST

fresh fruit or tomato juice
eggs, any style; two or more eggs if desired
bacon, ham, fish, cheese, or other meat
1 slice bread or 4 soda crackers
butter or oleomargarine
beverage: milk, coffee, tea, cocoa, without sugar; saccharin may be substituted for sugar

This breakfast may be varied according to taste, capacity, and appetite. Some individuals are satisfied with fruit, eggs, bread and butter, and a beverage. Others wish to eat meat, fish, or cheese. There is no limitation in the amounts of permitted foods. If cereals are eaten at all, the quantity should be small, and only saccharin should be used for sweetening.

LUNCH

tomato juice, broth, or soup containing permitted vegetables, but no rice, noodles or other starch
meat, fish, or poultry (as much as desired)
permitted vegetables, raw or cooked
salad, with or without dressing
1 slice bread or 4 crackers
butter or oleomargarine
beverage: milk, coffee, tea, or cocoa, with saccharin, if desired
permitted fresh fruit

or

tomato juice, or broth, or soup
combination salad: egg, sardines, salmon, cold cuts, etc.
cheese, sour cream; fresh vegetables, cut up, with sour cream
1 slice bread or 4 crackers
butter or oleomargarine
beverage, as above
permitted fresh fruit

DINNER

oysters, shrimp, tomato juice, broth, soup
meat, fish, poultry, omelet
permitted vegetables
salad
1 slice bread or 4 crackers
butter or oleomargarine
beverage, as above
nuts, permitted fresh fruit, cheese

or

oysters, shrimp, tomato juice, broth, soup
combination salad, cold cuts, cheese, eggs, sour cream

permitted vegetables
salad
1 slice bread or 4 crackers
butter or oleomargarine
beverage as above
nuts, permitted fresh fruit, cheese

Soybean muffins made from pure soy flour may be used in unlimited quantity as a substitute for bread and crackers. Natural gravies are preferred to gravies thickened with wheat flour.

BOX LUNCH SUGGESTIONS

1. Cold cuts of meat or poultry; parsnips fried in butter; dill pickles; lettuce; tomato juice.
2. Hard boiled eggs; sliced cold boiled carrots; cheese; olives; walnuts; milk.
3. Cold roast lamb or fried eggs; fried slices of eggplant dipped in egg batter and soybean flour; season; place lamb between slices of eggplant; pickles; milk or tomato juice or beef bouillon, hot or cold.
4. Slices of tripe fried in batter of egg and soybean flour; cheese or hard boiled egg; lettuce; celery stuffed with pimento cheese; milk or tomato juice.
5. Vegetable salad (no potatoes) with dressing, carried in a small jar; soybean muffins or crackers; salted nuts; clear broth, milk or tomato juice.
6. Hot soup or broth carried in a thermos bottle; soda crackers or soybean crackers or muffins; cheese; milk or tomato juice.
7. Summer squash dipped in beaten egg and fried in oil or butter and sprinkled with grated Romano or other cheese, well seasoned; cold roast chicken or turkey; milk or tomato juice or consomme.
8. Left-over meat or poultry chopped fine with grated cheese, seasoned, and stuffed into green peppers; olives; radishes; celery; milk or tomato juice.
9. Finely chopped cabbage fried slowly in bacon fat; add few green peas and some tomato paste, a dash of paprika, grated strong cheese, to make an omelette; may be carried

in a bowl covered with wax paper; olives; milk or tomato juice.

10. Chopped nuts mixed with a paste of hard boiled eggs to which a little oil is added, a dash of tabasco sauce, and a pinch of chili powder; this may be rolled in tender cabbage leaves and steamed for 30 minutes; may be eaten cold with pickles and cheese; milk or tomato juice or bouillon.
11. Cocktail frankfurters; pickled beets; olives; lettuce; chopped mixed nut salad; hot consomme.
12. Pickled peppers with thin slices of roast or boiled chicken or turkey, dusted with chili powder and rolled into lettuce leaves; milk or tomato juice.
13. Pickled egg; sauerkraut; cheese; soybean crackers or soda crackers; milk, buttermilk, or tomato juice.
14. Left-over chicken, veal, roast pork, chopped fine and mixed together, well seasoned and pressed into patties, fried in oil or butter to make a sandwich surface for pimento cheese as a filler; milk or tomato juice.
15. Thin codfish cakes with a slice of ripe tomato placed between the cakes; milk or tomato juice.
16. Italian or other sausage fried in oil; celery; cheese; crackers; tomato juice.

The above suggestions may be altered to taste. Ripe tomatoes, green peppers, cucumbers, or celery, may be added to any lunch.

Chapter IX

THE CONCEPT OF GAMMA-GLUCOSE

I believe the reader will benefit by a discussion of the concept of gamma-glucose. Ordinary glucose is a mixture of two chemical forms called alpha-glucose and beta-glucose. Alpha-beta-glucose mixture is derived from the cane sugar we eat, and from the sugar present in fruit; it is an end-product of the digestion of starch. Gamma-glucose is so labile (unstable chemically) that it has never been isolated in the test tube and identified as such. The evidence for its existence however, while indirect, is strong. Many authorities in chemistry and physiology believe it exists since it helps explain certain phenomena which otherwise would be obscure. (Just as in nuclear physics where faith in the existence of protons, electrons, and mesons, helps to explain many phenomena.)

Gamma-glucose is considered to be labile, highly reactive, and readily oxidized. It is very likely that the cells of the body prefer to oxidize gamma-glucose to the exclusion of alpha-beta-glucose. Dr. Shaffer, (29) an authority on the subject, in a review of the literature on gamma-glucose, has stated that the hypothesis appears attractive that alpha-beta-glucose is converted under the influence of insulin into gamma-glucose. In this discussion, and in the present state of our knowledge, it is sufficient to believe that alpha-beta-glucose is synthesized to glycogen in the liver under the influence of insulin and that the glycogen is subsequently broken down into gamma-glucose which is then liberated into the blood.

When a human lives on meat and fish exclusively, as does the Eskimo or Arctic explorer, he is living on protein and fat. Yet glycogen is stored in the liver in normal amounts and there is a constantly normal blood sugar level. Where do the glycogen and glucose come from on

such a diet? They are derived from the protein and fat in the diet. Fifty-eight percent of protein and 10% of fat can be converted by the body to glycogen and ultimately to glucose. I want to point out that the blood sugar on such a diet may be said to exist exclusively in the gamma form. On such a diet the blood sugar is produced entirely within the body and so may be called endogenous glucose as opposed to the exogenous alpha-beta-glucose which is introduced from the outside in the foods we eat. Certainly it would appear plausible to regard the sugar derived from meat and fish (protein and fat) as being different chemically from the sugar derived from cane sugar, fruits, and starch. From my studies I have been forced to conclude that alpha-beta-glucose is so foreign to the cells of the body as to be harmful to the body's economy.

There is ample evidence for this statement from observations made by two of this country's leading researchers in metabolism, Benedict and Carpenter. (30) These investigators determined the minimum (basal) oxygen requirements of normal human subjects and then measured the oxygen consumption of these subjects after various test meals of sugar, starch, protein, and fats, alone and in combination.

After a protein meal they always observed a marked rise in oxygen absorption (consumption), a phenomenon called "the specific dynamic action" of protein and which is regarded as highly beneficial to the body. After a fat meal there was a slight rise in oxygen absorption or none at all. After sugar and starch they found a slight initial rise followed by a fall in oxygen absorption in some experiments, and in other experiments they observed a consistent fall in oxygen absorption, with no initial rise. Benedict and Carpenter were unable to explain this fall in oxygen absorption after sugar and starch. They were much surprised to discover that the ingestion of sugar and starch actually could cause a depression of total bodily oxygen absorption below basal requirements.

Here are some of the actual experimental results reported by Benedict and Carpenter. They fed one subject 400 grams of bananas (413 calories) and observed an increase in oxygen absorption of 5.5 grams during the first hour, and a fall in oxygen absorption below basal requirements during the second, third, and fourth hours. Bananas contain chiefly sugar and starch. In the same subject, after 217 grams of beefsteak (451 calories), which consists of protein and fat, there was an increase in oxygen absorption during each of four hours, with a total increase of 17.5 grams of oxygen against a net increase of only 1.5 grams of oxygen after the bananas.

With larger meals the results were even more striking. For example, after 1382 calories of a sugar mixture, there was a fall in oxygen absorption of 11 grams over an eight hour period with no initial increase; in other words the subject would have consumed 11 grams more oxygen if he had fasted for the eight hours. On the other hand, after 1305 calories of beefsteak there was an increase in oxygen absorption of 40 grams during an eight-hour period.

Now, how does the ingestion of sugar and starch depress oxygen absorption? There are two ways by which this can be brought about: first, by causing low blood sugar, and second, by depressing the liver output of gamma-glucose. As has been previously explained, sugar and starch may cause low blood sugar about an hour after eating, and this period of low blood sugar may last from one to three hours. During the period of low blood sugar there will be a reduced delivery of glucose to all the cells of the body with a resultant reduced oxidation of glucose; as a result, less oxygen will be absorbed by the body since cells utilize oxygen in proportion as they utilize glucose. Also, during the period of reduced glucose-oxygen consumption, less heat will be generated and so the body temperature may fall. This reduced heat production during low blood sugar readily explains the presence of sub-

normal body temperature in certain individuals in the morning before breakfast, and even after meals. Such individuals tolerate cold weather very poorly.

The second mechanism whereby sugar and starch may cause a depressed oxygen consumption involves the concept of gamma-glucose. During fasting, the blood sugar is derived entirely from the breakdown of liver glycogen and may be regarded as existing in the readily oxidizable gamma form. Now Dr. Soskin and his co-workers (31) at Michael Reese Hospital, Chicago, have found that when ordinary alpha-beta-glucose is injected into the blood stream of a dog, the liver output of glucose is depressed, and the greater the amount of alpha-beta-glucose injected the greater the depressant effect on the liver output. This reduced output by the liver occurs because there is no need for the liver to pour out endogenous glucose as long as the exogenous alpha-beta-glucose is being introduced from the outside. The injection of exogenous glucose raises the blood sugar level and, to prevent too great a rise, the liver responds by lowering its output. The oxygen absorption of the body may fall, however, in spite of a rise in blood sugar level which followed the injection of glucose, because of the reduced output from the liver of the more readily oxidizable form which I believe to be the gamma form.

The influx of alpha-beta-glucose into the blood drives the gamma-glucose out of the circulation, just as bad money drives good money out of circulation. The oxygen absorption of the body falls in proportion as the output of gamma-glucose falls. This concept explains why Benedict and Carpenter observed a moderate fall after small sugar meals and a greater fall after larger sugar meals. After a pure protein and fat meal (beefsteak) there is no introduction of alpha-beta-glucose and hence there will be no depressant effect on the liver output of gamma-glucose and there will be no fall in oxygen absorption. The marked increase in oxygen absorption after beefsteak may be readily explained by the oxidation of the products

of digestion resulting from the digestion of the protein and fat.

Summarizing, the ingestion of sugar and starch may depress oxygen absorption by causing low blood sugar and/or depressing the output of gamma-glucose from the liver. In some instances only one of these mechanisms may be operating, and in other instances both mechanisms may be operating. In the latter, the fall in oxygen absorption may be so great and so prolonged as to cause a marked lowering of the body's ability to resist an acute infection, such as polio or pneumonia.

I wish to impress the reader with this concept of gamma-glucose. Gamma-glucose is to be regarded as the "natural sugar," preferred by the cells of the body for energy purposes because it is more readily oxidized with greater release of energy. Alpha-beta-glucose is to be regarded as a "foreign sugar," and, in a sense, artificial. Alpha-beta-glucose may be oxidized directly but not as efficiently or as rapidly as gamma-glucose. The reader should remember that the ingestion of sugar and starch with resultant liberation of alpha-beta-glucose after digestion exerts a depressant effect on the liver output of the preferred gamma-glucose.

Knowledge in medicine and physiology is in a constant state of flux. New discoveries open new doors and confirm or alter previous concepts. In the light of our present knowledge, the concept of gamma-glucose presented here is fundamentally sound and warranted by supporting data, and it explains certain phenomena that would otherwise be baffling. Regardless of whether or not the above explanation is the correct one, the fact remains that the ingestion of sugar and starch may readily cause a fall in total bodily oxygen absorption through adverse effects on the blood sugar regulatory mechanism, and it is this state of oxygen lack that is responsible for, let us say, a lowering of the body's resistance to invasion by a virus, or for the sequence of events that lead to a fatal heart attack.

Fig. a

In the fasting state the blood glucose (or blood sugar) is derived from the breakdown of liver glycogen and may be said to exist in the endogenous gamma-glucose form.

Fig. b

Following the ingestion of a moderate amount of exogenous alpha-beta-glucose the blood sugar level will rise, and the blood glucose value will now be the sum of the gamma-glucose plus the alpha-beta-glucose. However, the exogenous alpha-beta-glucose inhibits the output of endogenous gamma-glucose from the liver, and the concentration of gamma-glucose will fall as shown by the heavy line in the figure. Thus there may be a fall in total body oxygen absorption because of the fall in gamma-glucose concentration and in spite of the rise in blood glucose level.

Fig. c

Following the ingestion of a larger amount of exogenous alpha-beta-glucose the blood glucose level rises to a high value and the liver output of endogenous gamma-glucose is proportionately depressed. Under such circumstances an individual may experience hypoglycemic symptoms even though the blood sugar actually rises. The gamma-glucose output may be subnormal for 1 to 3 hours. After such a meal there may be a fall in total body oxygen consumption.

Fig. d

Following the ingestion of proteins and fat the blood glucose level rises moderately. Since no sugar or starch is ingested no exogenous alpha-beta-glucose is available to inhibit the output of endogenous gamma-glucose from the liver. The blood sugar will thus consist of the gamma-glucose form exclusively. The rise in blood sugar after protein and fat is due to the rapid conversion of the digested protein and fat to gamma-glucose. A meal consisting of protein, fat, and carbohydrate foods containing no sugar or starch will also cause a moderate rise in blood sugar and with no inhibition of liver output of gamma-glucose. After such a meal there will be a rise in total body oxygen absorption.

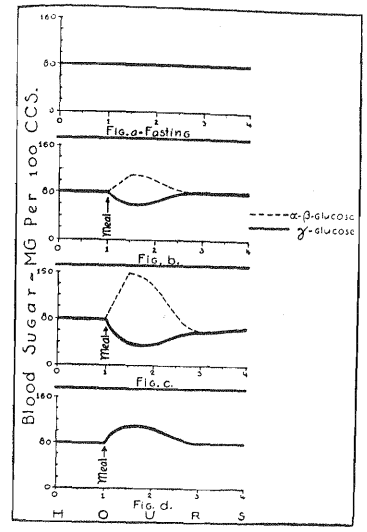


Fig. 16

Chapter X

TOBACCO AND THE HEART

Tobacco contains nicotine which is a chemical with a powerful pharmacological action in the human body. It is the nicotine in tobacco which accounts for the "satisfaction" that is derived from smoking. De-nicotinized tobacco does not satisfy the habitual or occasional smoker. Chain smokers are actually nicotine addicts and are no different from other drug addicts. Nicotine addiction is not regarded as seriously as narcotic and alcoholic addiction. Tobacco addiction is apparent when a chain smoker attempts to stop abruptly. He exhibits a variety of withdrawal symptoms which are chiefly nervous in origin and resemble the symptoms of low blood sugar.

The pleasant effects of smoking are due largely to the ability of nicotine to elevate the blood sugar and thus give the smoker a "lift." This explains why smoking is in reality an artificial stimulant. However, the rise in blood sugar is transient and is soon followed by a fall which calls for another smoke and which will also be followed by a rise in blood sugar. The desire for a smoke is actually due to the fall in blood sugar. This explains the chain smoker who smokes 20 to 40 cigarettes a day. Each fall in blood sugar calls for another "pick-up." Smokers will tell you that they "enjoy" smoking. They are not aware of the fact that they are really addicted to and dependent on nicotine. Instead of eating some wholesome food to elevate and stabilize the blood sugar they resort to a quick, satisfying, artificial form of blood sugar elevation. The rise in blood sugar is always at the expense of stored liver glycogen and this is part of the price paid for the dubious pleasure of smoking.

Since many medical men believe that tobacco is harm-

ful to the patient with angina pectoris, it was logical for me to investigate the effect of smoking on the blood sugar. It had already been known that nicotine could cause a rise in blood sugar. The rise in blood sugar has been attributed to an increased output of adrenalin which then stimulated the liver cells to put out glucose. Dill, Edwards, and Forbes (32) state that the effect of tobacco smoking on the blood sugar depends on how much nicotine is absorbed. If the amount is small no effect may be observed; if sufficient is absorbed there may be a rise; if a large amount is absorbed it may be depressed. Ssalischtscheff (33) induced toxic symptoms by having subjects smoke until such symptoms appeared. He tested 11 subjects, non-smokers and habitual smokers. He took a blood sugar specimen just before smoking commenced and a second one when toxic symptoms appeared. He described the following symptoms: general weakness, "adynamie," salivation, sweating, nausea, vomiting, tremors, cramps, and dizziness. The falls in blood sugar varied from 18 to 61 percent. One subject, a habitual smoker aged 41, had a decline from 81 mg. to 31 mg.

There are patients with angina pectoris who state that smoking sometimes causes an attack of chest pain similar to the spontaneous attacks. The term "tobacco angina" has been applied to this type of pain after smoking. No satisfactory explanation for the tobacco pain has been offered that is consistent with the usual explanation of angina pectoris.

Since I believed that attacks of chest pain in angina pectoris were due to sharp falls in blood sugar it was logical for me to study the effects of smoking on the blood sugar. I suspected that nicotine could cause a sharp fall in blood sugar and thus cause an attack of pain. Like other investigators I found that nicotine could very readily cause a considerable fall in blood sugar in a short period of time, measured in minutes. In addition to causing the chest pain, nicotine can cause other classic symptoms of

low blood sugar such as dizziness, headache, nervousness, tremors, palpitation, rapid pulse, peripheral vasoconstriction, etc. Nicotine is more apt to cause a fall in blood sugar when the subject has not eaten for several hours, as in the morning before breakfast. Hiestand and co-workers (34) reported that nicotine had more marked effects on the metabolism and cardio-respiratory function when the subject was in the basal (fasting) state. Smokers will tell you that they enjoy smoking best directly after finishing a meal. This can be readily explained by the fact that there is little likelihood of nicotine causing a fall in blood sugar with its train of unpleasant symptoms at that time because the blood sugar is rising due to the absorption of nutrients from the digestive tract. Smokers usually learn which times during the day are best for smoking and may confine their smoking to those periods. Some smokers know that a cigarette on an "empty stomach" doesn't taste as good and doesn't satisfy as well, as at other times when the stomach is full. Of course, an empty stomach means a blood sugar that is at or near the fasting level and hence more apt to be depressed than elevated by a smoke.

Method Used in Testing Effects of Tobacco on the Blood Sugar. Since symptoms occurred after smoking more frequently in the morning before breakfast, the subjects were tested at around 9:00 a.m. after an all night fast. A control blood sugar specimen was drawn at the start of the test and the subject was told to smoke a cigarette, or cigar or pipe, according to habit. The subject was instructed to smoke as he would ordinarily, and no attempt was made to bring on symptoms by forced and rapid inhalations. The duration of the tests varied from 5 to 20 minutes, according to the reaction and co-operation of the subject. Blood sugar specimens were drawn at 2 to 5 minute intervals. The subjects were told to describe symptoms as soon as any were felt. They were up and about during the test. They ranged in age from 23 to

68 years. Twenty-two tests were performed on 21 subjects; one subject was tested twice on two different days. Four subjects stated that they had no symptoms and enjoyed the smoke. All four subjects showed either a slight rise in blood sugar or no significant change. In the remaining 18 tests, the subjects reported moderate to severe symptoms. In all 18 subjects the symptoms coincided with a fall in blood sugar, the magnitude of which usually corresponded with the severity of the symptoms. Relief of symptoms always coincided with either a rise in blood sugar or a cessation in decline.

Illustrative Experiments.

Subject No. 1. (Fig. 17) Cigarette smoker. During the first test on 6/22 the subject did not experience any unpleasant symptoms. He enjoyed the smoke and the blood sugar shows a slight rise during the period of observation. During the second test on 6/29 which was carried out

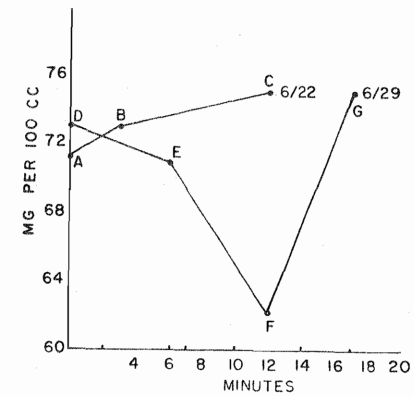


Fig. 17

Subject No. 1. On 6/22/40 the fasting blood sugar was 71 mg. just before smoking started. Three minutes later, at "B," the blood sugar was 73 mg. and patient said he was enjoying the smoke. At "C," 12 minutes after start of the experiment, the blood sugar was 74 mg. and the subject had no symptoms. The subject enjoyed the smoke during the whole period of observation. On 6/29/40 the blood sugar fell from 73 mg. to 71 mg. after 6 minutes of smoking, ("E") at which time the subject complained of slight headache. The blood sugar then fell rapidly from 73 mg. to 62 mg. ("F") at which time he complained of low abdominal pain. The blood sugar then rose spontaneously to 74 mg. ("G") at which time he felt better.

under similar fasting conditions, the subject complained of headache and abdominal cramps. The headache began about 4 minutes after smoking began. Low abdominal pain began about 9 minutes after smoking started and lasted for about 5 minutes. Note that pain disappeared coincident with a rise in blood sugar. This rise in blood sugar may be attributed to either the stimulation of the adrenal-sympathetic system or to direct stimulation of the adrenal glands by nicotine, or both. The significance of both these actions is discussed in the text.

Subject No. 2. (Fig. 18) Cigarette smoker; angina pectoris patient. The test was started at 10:08 a.m., at which time the fasting blood sugar was 105 mg. and the subject said he felt all right. He began smoking directly after the fasting specimen was drawn and remained sitting in a chair. At 10:12 a.m. he got up from the chair and felt so dizzy that he almost fell over backwards. A blood sugar

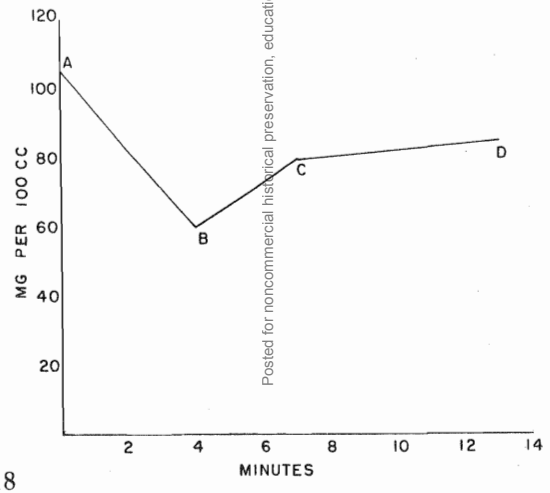


FIG. 18

Subject No. 2. At 10:08 a.m. the fasting blood sugar was 105 mg. and the subject started to smoke a cigarette. He felt all right. At "B" he got up from the chair and was so dizzy he almost fell over backwards; at this time the blood sugar had fallen sharply from 105 mg. to 60 mg. A third specimen at "C" showed a rise in blood sugar to 78 mg. and the subject said he felt better. At "D" the blood sugar was 84 mg. and the subject said he felt better.

drawn at 10:12 showed 60 mg. A third specimen at 10:15 while he was up and about and had slight dizziness was 78 mg. At 10:21 he felt much better and the blood sugar was 84 mg.

Master, Jaffe, and Dack, (35) observed pain and electrocardiographic changes during smoking. These changes were completely reversible. Since tobacco may cause a sharp fall in blood sugar level one may justifiably attribute the pain and electrocardiographic changes observed during smoking to the depressant effect of tobacco on the blood sugar.

Just how tobacco causes a fall in blood sugar can only be surmised at this time. However, I believe that the fall in blood sugar is due to an inhibitory effect on the liver output of glucose into the blood stream. The sharp fall stimulates the adrenal-sympathetic system which, by effecting the breakdown of liver glycogen, reverses the fall and restores the blood sugar to its previous level. The rapid pulse, palpitation, tremors, and peripheral vasoconstriction observed during smoking are due to such adrenal-sympathetic activity.

Chapter XI

THE INFLUENCE OF EMOTIONS, MENTAL STRESS AND STRAIN, ON THE ANGINAL SYNDROME

All authors on the subject of heart disease agree that prolonged unpleasant psychic experiences such as anxiety states, worry, fear, as well as acute unpleasant emotional experiences, affect adversely the anginal syndrome and help precipitate the eventual heart attack. That pleasant and unpleasant psychic experiences strongly influence disease is evident in many other conditions with a significant psychosomatic component such as peptic ulcer and colitis. In some of the medical articles published by me I have attempted to show that unpleasant psychic experience disrupts the mechanism controlling the carbohydrate metabolism and eventually causes abnormal blood sugar levels. A disrupted carbohydrate metabolism thus becomes a link between unpleasant psychic experience and the genesis of disease.

Aware of the fact that patients with angina pectoris often had aggravation of symptoms following psychic trauma, mental upsets, financial and domestic difficulties, it occurred to me that such unpleasant environmental factors could bring about visceral and mental symptoms by deranging the carbohydrate metabolism. Sudden bad news, for example, could bring on abdominal pain, chest pain, or even syncope (fainting) by causing an immediate sharp fall in blood sugar. The following observations which I made on patients illustrate the effect of unpleasant physical and psychic experiences on the blood sugar.

Hypoglycemia and Syncope Following Venipuncture. (Fig. 19) A white male, aged 50 years, reported to the clinic for a glucose tolerance test in the morning after

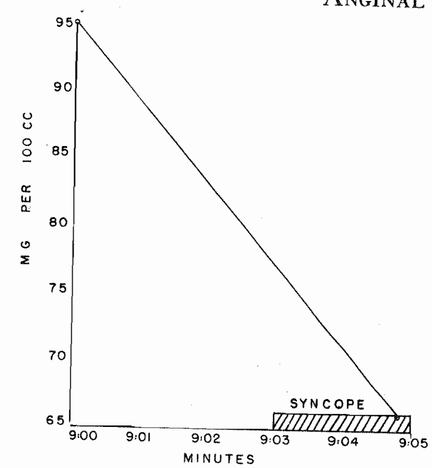


FIG. 19

Syncope following venipuncture at start of a glucose tolerance test. At 9:00 a.m. the fasting blood sugar specimen was drawn. At 9:01 the patient was given 100 gm. of glucose in 250 cc. of iced water. At 9:02 he appeared pale and nervous. At 9:03 he fell unconscious to the floor. At 9:05 he recovered spontaneously, at which time the second blood sugar specimen was drawn. The cross-hatched area shows the duration of syncope.

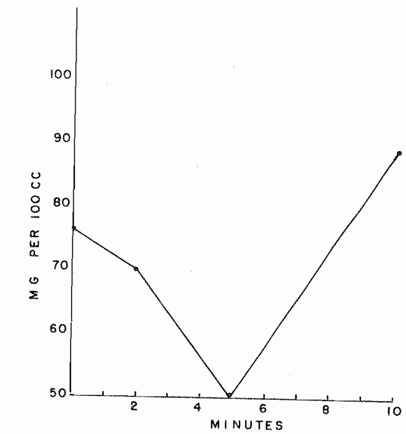


FIG. 20

The fasting blood sugar was 75 mg. when the subject was told that he was to be operated upon the next day. At two minutes the blood sugar had fallen to 70 mg. and the subject said he had a "weak feeling" in the region of the heart and stomach. At five minutes the blood sugar was 50 mg. and the subject said he felt worse. He appeared anxious and pale. At ten minutes the blood sugar was higher and the subject said he felt all right. He had regained composure.

an all-night fast. The patient fell unconscious about three minutes after the venipuncture, was unconscious for two minutes, and recovered spontaneously. The fasting blood sugar was 95 mg. On recovery from syncope, it was 65 mg. It is very likely that the blood sugar had fallen to even lower than 65 mg. and was on the rise when the second specimen was drawn since regain of consciousness must have depended on recovery from hypoglycemia. Such recovery may be attributed to the liberation of adrenalin. The pallor and cold sweat in syncope are evidence of such adrenal-sympathetic activity. I suggest that the syncope was due to the sudden fall in blood sugar precipitated by the unpleasant experience; namely, the venipuncture. Such a sharp fall in blood sugar may result from a cessation in the liver output of glucose mediated by a humoral or nervous mechanism.

Hypoglycemia Following Sudden Bad News. (Fig. 20) A white male, aged 17 years, with a long history of chronic hypoglycemia, was suddenly informed that he was to be operated upon the next day for an associated condition. He was obviously upset, appeared pale, and described a feeling of faintness in the precordium and abdomen. He regained composure after several minutes concomitant with a rise in blood sugar. The fall in blood sugar was not as great or as rapid as in the patient who fainted after venipuncture, but it was of sufficient magnitude to cause moderate visceral discomfort.

It has long been known that emotional disturbances may cause hyperglycemia and even glycosuria. Such hyperglycemia has been regarded as a defense mechanism in that it increases the amount of glucose available for energy purposes during a period of stress. It is very likely that immediately following a sudden unpleasant change in environment there is a transient hypoglycemia which is responsible for the unpleasant visceral and mental sensations, such as faintness, abdominal discomfort and pain, nervousness, tremors, pallor, rapid pulse, and palpitation,

which go to make up what we call fright and fear. Recovery from this phase and efforts made to protect the organism depend on a subsequent hyperglycemia. The individual who faints or who makes no attempt to defend himself probably has no such hyperglycemia.

Through a similar mechanism, chronic psychic trauma may cause chronic symptomatology by derangement of the mechanism controlling carbohydrate metabolism and blood sugar regulation. It is a matter of common knowledge that diabetes is often aggravated by worry or other unpleasant emotional states. This adverse effect is manifest by an increase in hyperglycemia and glycosuria, signifying that the glucose is not being utilized and is accumulating in the blood. In severe cases the diabetes becomes uncontrollable and leads to acidosis and coma. The final common effect, in both diabetics and nondiabetics, of such emotional states will be the same; namely, a decreased glucose and oxygen consumption. Abdominal and chest pain, headache, dizziness, occurring in both diabetics and nondiabetics, will be due to this final common effect. Thus may be explained the abdominal pain frequently encountered during diabetic acidosis; it is due to reduced glucose utilization and disappears when such utilization is restored with insulin.

It is thus apparent that the mechanism governing carbohydrate metabolism and blood sugar regulation is a sensitive one and is especially influenced by the emotional state of the patient. Carbohydrate metabolism may thus be unfavorably influenced by a high carbohydrate intake and psychic trauma. Since it is often difficult to remove the latter, much may be done by dietary management. If, then, a patient presents himself with symptoms obviously due to psychic trauma, he may be relieved of such symptoms by either removing the cause, i.e., the trauma, or by elevation and stabilization of blood sugar levels with a low carbohydrate diet. On such a diet the unfavorable effect on the blood sugar brought on by the psychic

trauma is neutralized and symptoms are prevented. We have, therefore, a medium whereby unpleasant emotional states may give rise to a variety of functional symptoms. By depression of blood sugar levels such emotional states may reduce the amount of glucose available for cellular utilization and indirectly reduce the oxygen consumption in the organism. The function of every cell that utilizes glucose as a source of energy will therefore be affected. The severity of the functional disorder will depend on the magnitude of the derangement of the carbohydrate metabolism, and whether or not the changes in cellular function are reversible.

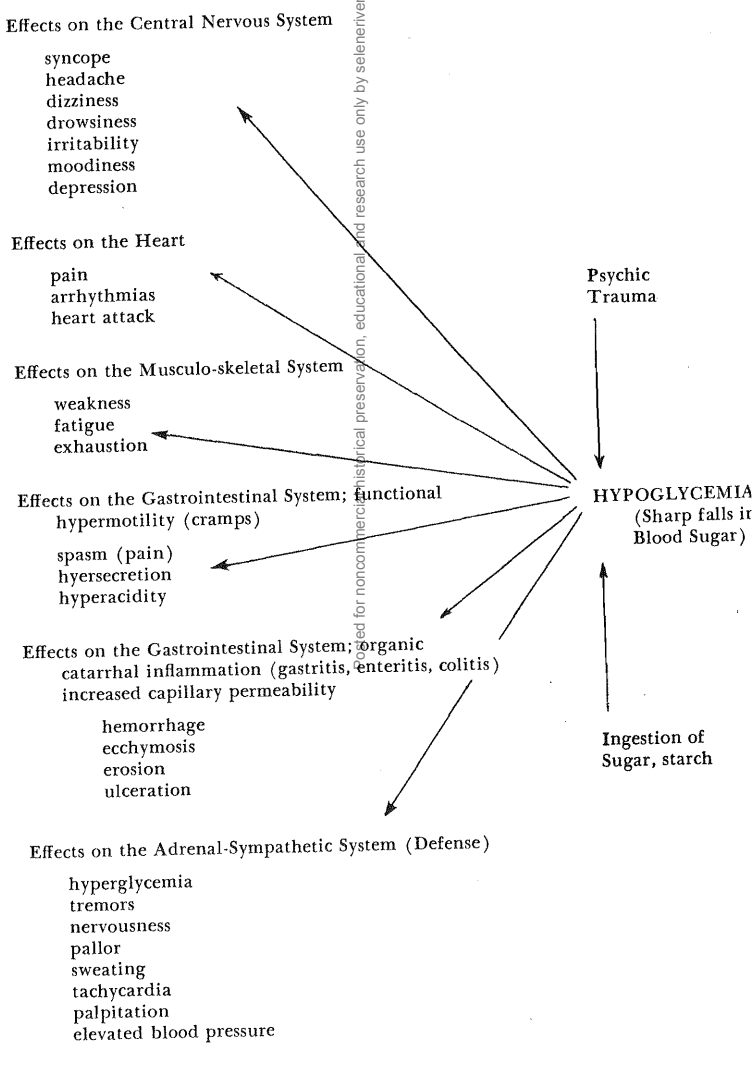
The Relationship Between Fluctuations in Blood Sugar and the Hunger Mechanism. Normally, after a meal, the blood sugar rises to around 120 mg. in the first hour, 130 mg. in the second hour, 140 mg. in the third hour, and then falls to previous fasting levels, around 80 to 100 mg., during the fourth hour. The only symptom that should occur during the period of decline is the sensation of hunger. The fall in blood sugar stimulates the vagus nerve which in turn causes an increase in gastric motility and tone. Cannon and Washburn (36) showed that "strong contraction of the muscle fibers of the wholly empty stomach, whereby its cavity disappears, makes a part of the sensation which we call hunger." Dickson and Wilson (37) injected insulin into human beings and noted that there was an increase in tone, depth, and rate of peristalsis of the stomach about an hour after the injection, when the blood sugar was around 70 mg. As was shown by Bulatao and Carlson (38) in dogs, and by La Barre and Destree (39) in human beings, gastric motility was replaced by atony when the blood sugar was depressed still further. This explains the loss of hunger and the sensation of epigastric hollowness and emptiness encountered in patients with chronic hypoglycemia where the blood sugar may be maintained for long periods at hypoglycemic levels. Further, in patients who give the flat type of curve in the

tolerance test, as an additional cause for the lack of hunger, is the absence of adequate fluctuation in blood sugar levels necessary to stimulate the hunger mechanism.

A most common complaint in individuals after psychic trauma or during periods of worry is loss of hunger sensation. In the neurotic disorder, anorexia nervosa, the glucose tolerance is abnormal and the curves obtained are chiefly of the flat type. Return of hunger, gain in weight, and improvement in mental state, regardless of the therapeutic measures employed, are associated with an improvement in the carbohydrate metabolism. The following is from a recent paper by Ross. (40) He writes, "The conception of anorexia nervosa put forward is that . . . the physiological factor of impaired glucose tolerance with a slowly falling alimentary hyperglycemia leads to a failure of hunger and the impairment of one element of appetite — a normal gastric tonicity. . . . It seems clear, then, that there is a relationship, be it causal or coincident, between the gastric manifestations of hunger and a fall in blood sugar level, and it would be strange if it were merely coincident. If it is causal, then a reasonably rapid fall of alimentary hyperglycemia back to fasting level is necessary for the regular occurrence of hunger; this fall we know is dependent on a reasonably good glucose tolerance."

One may state that the psychic trauma responsible for anorexia nervosa has disrupted the blood sugar regulatory mechanism which now brings in its wake loss of hunger and a variety of symptoms. Complete removal of the psychic trauma, if this is possible, usually cures the patient. One may say that its removal restores the regulatory mechanism to its normal status. Where complete removal is impossible, other measures, such as psychotherapy, psychoanalysis, suggestive therapy, or time, with its ability to make one forget, may cure the patient. These varied therapeutic measures apparently accomplish a final common effect; namely, a restoration to normal of the carbohydrate metabolism.

The following diagram shows how Psychic Trauma and the ingestion of Sugar and Starch, by producing acute and chronic Hypoglycemia, may cause many and varied functional (neurotic) signs and symptoms in the important organ systems of the body. It is also suggested that the prolonged abnormal functional changes may ultimately lead to organic changes."



Mainzer and Krause (41) studied the effects of fear states on the electrocardiogram and reported that changes were noted both in healthy subjects and in individuals with cardiac disturbances. They report, "Fear may induce considerable changes in the electrocardiogram, even in healthy hearts. In two persons with cardiac disturbances the changes were particularly marked." They observed an increase in amplitude of the P and T waves with pointing in some subjects, and diminution in size of the ventricular complex, negative T deflection, depression of the S-T segment below the iso-electric level in others.

Summarizing the data now at hand one may justifiably conclude that unpleasant emotional states may readily cause precordial pain and heart attacks because these emotional states may disrupt the mechanism controlling the carbohydrate metabolism and precipitate a fall in blood sugar.

A psychic trauma of short duration will produce acute symptoms and these will disappear when the trauma or unpleasant environmental factor is removed. Chronic psychic trauma, such as unemployment, anxiety states, stress and strain of daily living, will result in chronic derangement in the carbohydrate metabolism with chronic symptoms and eventually chronic changes of an organic nature. A low carbohydrate diet with its resultant stabilization and elevation of blood sugar levels to more normal range will tend to offset or neutralize the depressant action of psychic trauma and hence may be regarded as a buffer between such trauma and the genesis of pathological states.

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