FASTING AND DIET RESTRICTION

in the Treatment of Cardiovascular Disease

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The physiological state which accompanies fasting and food restriction is characterized by the use, primarily, of lipid to satisfy the body's energy needs. This tends to spare body proteins. Studies of the biochemistry and metabolism of fasting and food restriction indicate that these dietary alterations may be expected to have a beneficial effect in cases of atherosclerosis, hypertension, and selected cases of heart failure. This has been confirmed by observations during wartime food restriction, and at autopsy. A method for conducting a fast is presented together with a discussion of three case histories.

Introduction

Approximately one in five US adults has hypertension.[1] About five million Americans have ischemic heart disease, the number one cause of death in males after age 35, and in all persons after age 45.[2] This article will discuss diet therapy, specifically fasting and food restriction, in the treatment of atherosclerosis (the underlying pathology in ischemic heart disease and in most other cardiovascular diseases), hypertension (a contributory factor to many cardiovascular diseases), and cardiac insufficiency/heart failure. Covered in addition, are the metabolism and technique of application of fasting therapy, and some case reports.

Fasting is defined as complete astinence from food (solid and liquid); water is allowable. During the period in which the body is fasting, non-essential reserves stored within the body (mainly lipids) are used to supply the energy needs. This is in contrast to starvation, that metabolic state reached after a long period of fasting; the difference is that during starvation, the body, having exhausted its reserves of non-essentials, begins to break down essential tissues to supply its energy needs. Death quickly follows.

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Metabolism of Fasting

Since the 1950s, fasting therapy has been used to treat obesity. The scientific community has thus been stimulated to investigate the changes in metabolism undergone by the body in the fasting state. Researchers have attempted to explain how it is possible for the body to survive without food intake for long periods of time, 249 days in one case[3] and 382 days in another.[4] Since excellent reviews of this information have already been published,[5,6] this subject need not be covered in great depth but discussed only briefly.

When a person has abstained from food for a few hours at the start of a fast, glycogen reserves are broken down to maintain blood glucose levels. Correspondingly, glucose use becomes restricted to few tissues, the brain being the major consumer. This conserves the small glycogen stores. Instead of glucose, fatty acids, the main source of which are adipose tissue, are preferentially oxidized. When the fast has progressed until the glycogen stores have been exhausted, the body begins to form glucose from glycerol (derived from adipose tissue), lactate (formed from oxidation of glucose by red blood cells, renal medulla, bone marrow, white blood cells, and others, and recycled to the liver via the Cori cycle), and from amino acids (derived mainly from muscle). The main source of this glucose is the amino acids. Were this to continue at the same rate, the muscle mass and other stores of body protein would be rapidly depleted of protein to supply the needs of the brain and survival would not be long-lasting. To prolong survival, the brain begins to burn ketone bodies formed from fatty acids in the liver. This greatly reduces the glucose

requirement, and thence the rate of protein breakdown.[5,6]

This, in brief, is the adaptative mechanism by which the organism can survive in the fasting state. The amount of time a person may safely fast depends on the quantity of stored reserve food (primarily lipid). As noted, an obese patient fasted for 382 days with no dangerous side effects. Some investigators have theorized that a "normally fed man" may fast for more than 80 days.[5] In the therapeutic setting, such non-obese patients routinely fast for periods sometimes reaching 40 days.[7]

It should be emphasized that, while fasting, the vast majority (about 90%) of the energy needs of the body is supplied by adipose tissue, not lean tissue, breakdown.[6] Over 60 years ago, this was demonstrated[8] and has been repeatedly confirmed in recent years.[9,10,11] Various research methods have shown that fat is the primary substance oxidized in fasting. One method is by determination of the respiratory quotient (RQ), the ratio of carbon dioxide output to oxygen usage. The RQ is measured by determining the total intake of oxygen and the total output of carbon dioxide. The RQ for carbohydrate oxidation is 1.00, for protein .81 and for fat .71. Therefore, if the RQ is .71, the individual is oxidizing mainly fat to the exclusion of carbohydrate and protein.[12] While fasting, one investigator found that the RQ is .71 in both obese and non-obese subjects.[10] Another researcher noted an RQ of .72 in a non-obese patient fasting for 31 days.[8]

A research method which shows that very little protein, and therefore muscle, is being lost in a fast, is measurement of nitrogen excretion during the fast. Many studies have shown low levels of nitrogen excretion.[10,11,13,14,15,16] This reflects that fat, not protein, is the major source of energy in the fast. When protein from lean tissues is broken down to form glucose, nitrogen is released and this is found in the excretions of the body.

Occasionally, researchers have stated that lean, not adipose, tissue is the primary source of energy during a fast. This argument was presented in an article entitled "The Caloric Theory Does Not Apply to Obesity" in the January 1978, *ACA Journal*.[18] Grande[18] has reviewed research supporting the hypothesis that lean tissue mainly is oxidized in the fast; he concluded that because of errors in computation, inconsistent data, inconsistent applications of basic assumptions, and unreliable measurements, no valid conclusions can be derived from this research. One of the studies reviewed by Grande formed the foundation for the January 1978, *ACA Journal* article.

Other pertinent aspects of the fasting metabolic state are apparent. Drenick[19] found that plasma amino acids, serum albumin and globulin levels remain at prefast levels despite continuous loss of nitrogen from the body during fasts of 12 to 117 days. Other investigators have confirmed this finding.[20,21,22] During the first few days of a fast, the excretion of sodium is about three times greater than that observed with a low salt diet, but this accelerated excretion comes to a halt upon consumption of a small amount of carbohydrate.[12,23,24,25,26,27] Consequently, juices are usually not allowed on a fast since this sodium excretion is one of the most desirable aspects of the fasting metabolism, especially in heart complications and hypertension.

Hunger is not experienced after the first two to five days from the start of a fast.[28,29,30] Feeding small amounts of protein during a fast does not prevent the loss of protein from the body while fasting and, furthermore, this loss of protein leaves no lasting defects.[24,30,31] Basal metabolic rate (BMR) decreases,[16,30,32,114] probably as a result of decreased secretion of thyroid hormone.[33] The BMR is probably lowered by the organism as a further means by which food reserves may be oxidized more slowly, thereby prolonging survival. The BMR has been observed to be higher after the conclusion of than before the fast, believed by one investigator to be a reflection of rejuvenescence.[34]

Slight changes of liver functions, in some cases causing hyperbilirubinemia, are sometimes seen, but liver function becomes normal upon refeeding.[20,31,35,36,37,38] Uric acid levels rise but only rarely is this associated with symptoms of gout.[3,4,19,21,39] Some investigators have cautioned that gout or hyperuricemia with family history of gout, if present before the fast, may be contraindications to prolonged fasting.[31]

Other possible contraindications to prolonged fasting include myocardial infarction (MI), or cerebrovascular accident of recent origin, insulin-dependent diabetes (unless the insulin dosage can be adjusted as necessary), chronic renal disease, severe hepatic disease, anemia, nutritional deficiency states, porphyria, psychological disorders in some cases, pregnancy, and perhaps childhood and adolescence.[31] Many exceptions, nevertheless, exist. Epileptic children[40] and persons with acute MI[37] have fasted for prolonged time periods with benefit to their conditions. Extremely weak and debilitated patients generally should not fast. As in all clinical matters, judgment will be necessary to decide which patients may encounter problems during the course of a fast.

The metabolism in cases of extreme caloric restriction (for example an 800 calorie diet) is similar to that of fasting. Nitrogen excretion, and consequently protein breakdown, is greatly reduced.[18,30,41] This reduction constitutes a survival mechanism; by sparing protein and oxidizing fat for energy, the body is able to prolong the period of survival even with inadequate sources of food.

Certain aspects of the psychology of fasting are of interest. Researchers have found that fasting is generally not difficult for the patient. F.M. Allen, MD, of the Rockefeller Institute, used fasting to treat severe diabetes before insulin was available. He found it necessary for patients to fast for weeks before the

diabetes was controlled but noted that "patients generally accept radical treatment with quick decisive results more readily than the weeks or months of privation heretofore used in stopping glycosuria, and the quick relief from polyphagia, polydipsia, and other symptoms aids further in securing their cooperation."[42]

Dr T.J. Thomson treated obese patients with fasting and found that "in common with other workers, we have found partial restriction of caloric intake to be relatively ineffective in the treatment of obesity. We therefore decided to adopt the method of total fasting . . . the rapid weight loss on this regimen presents to the patient, often frustrated and distressed by ineffective dieting, the solution to a hitherto intractable problem. This boost in morale is of inestimable value in ensuring their subsequent cooperation." Also, "the most surprising aspect of this study was the ease with which the prolonged fast was tolerated . . . we found that intolerance was not a problem, even in patients one and two after 236 and 249 days respectively."[3] W.L. Bloom, MD, found that "fasting is well tolerated for prolonged periods . . . patients experienced comparatively few undesirable symptoms; indeed, during the fast, many reported a marked sense of well-being, suggesting a mild euphoria . . . on resumption of eating, a 600 to 800 calorie diet was found to produce satiety, whereas, before the fast, this type of diet was unsatisfactory to the patient."[43]

E.J. Drenick, MD said: "All reports have emphasized that prolonged fasts are easily tolerated, little discomfort is experienced, and no serious adverse side effects are observed . . . the most astonishing aspect of this study, to the patient and to the physician, was the ease with which prolonged starvation was tolerated . . . it is remarkable that the fast seemed easier for the patient the longer it lasted."[14] G.G. Duncan, MD, noted that "the sense of well being and cheerfulness was surprisingly constant; anorexia was striking, notably after the first day of the fast, but in many hunger was not a complaint at any time . . . total fasts have been better tolerated than diets low in calories."[44] Other researchers concur in these findings.[45,46]

Investigators have noted certain side effects of fasting. Dr T.J. Thompson states that one patient experienced edema, one a bout of acute parotitis, and one a transient experience of postural hypotension. He says that "other symptoms were vague complaints of headache, lightheadedness, and nervousness, which could not be assigned to any physical abnormality. Since these were mentioned more commonly during the first week of fasting, we considered them to be due to anxiety arising from the absence of food."[3] Dr W. Bloom reports that his patients had few subjective symptoms other than occasional mild headaches and epigastric distress (relieved by water).[43] Dr Drenick mentions common early side effects of "mild headaches, occasional nausea, and some nervousness and tension . . . which seemed to abate readily. . . . There was no clear correlation of any of these symptoms with chemical changes observed in blood or urine." Some patients also developed orthostatic hypotension, the onset of which was usually after the third or fourth week of fasting. However, in one patient it occurred after only 12 days. With refeeding, "the postural fall in blood pressure was promptly reversed to a normal pattern." One patient developed acute arthritis of the ankle after three months of fasting; another developed arthritis in the ankle after 40 days. These attacks were associated with hyperuricemia, but no uric acid crystals were found in fluid aspirated from the joints; the arthritis responded to colchicine. Four of Drenick's 11 patients developed an anemia during the second month of fasting; this was normochromic, normocytic, was refractory to iron, folic acid, and B_{12} therapy, and gradually improved with refeeding.[19]

Dr Hollifield reports certain emotional disturbances associated with fasting but says that such problems "are not likely to occur in the absence of a history of recognized emotional disturbance."[47] Dr Duncan notes that "mild degrees of weakness, especially in those who were fairly active, were recorded but in none did this necessitate interrupting the fast. Headaches and light-headedness were complained of in a few instances. Transient waves of nausea in a mild form, except in one case in which vomiting occurred, were reported in approximately a third of these subjects."[44]

Atherosclerosis

Fasting and severe food restriction are proposed as treatments for the ubiquitous disorder of atherosclerosis. These dietary changes will, hypothetically, result in significant resorption of atherosclerotic plaques, and thus improve blood flow to the heart and peripheral tissues.

Resorption of atheromas necessitates that cholesterol, one of the main components of such lesions, must be in a labile state; it must be capable of leaving the plaque and not be inert. By administering radioactively labeled cholesterol to human subjects, scientists have been able to observe movement of cholesterol into and out of atheromas. Dr S.N. Jagannathan has done some of the more recent research in this area and he concludes that "the occurrence of definite influx of radioactivity even from obstructive atherosclerotic lesions suggests that the cholesterol in atherosclerotic lesions in man is potentially mobilizable."[48] Dr A.V. Chobanian in his studies concluded that "complete equilibration of serum and arterial intimal cholesterol does occur in normal human blood vessels."[49] Other studies have also observed turnover of the cholesterol in atheromas.[50,51]

Fasting is known to result in a decrease in the biosynthesis of cholesterol.[52] The combination of no intake plus decreased endogenous synthesis of cholesterol may be expected to result in a decreased

serum level of cholesterol since cholesterol is used for hormone synthesis, etc. This would allow increased movement of cholesterol from the arterial wall into the blood in order to establish an equilibrium, the end result being a reduction of size of atheromas.

Some studies have reported that serum cholesterol levels rise upon consumption of diets markedly restricted in lipid and calories.[53,54,55] The interpretation of this reflects a high rate of fat transport from storage.[56] Assuming a valid interpretation, the rise in cholesterol levels on such diets may reflect breakdown of atheromata with release of their contents.

The effect of severe food restriction and fasting on atherosclerosis has been studied both during and after periods of war. Conditions have caused considerable food shortages and, occasionally, imposed fasting. During World War II, food restriction resulting in decreased calorie and fat intake was a common occurrence in Europe and Russia, and available data reflects changes in health status at that time.

Dr H. Malmros studied the effect of food restriction in Germany, Norway, Sweden, Finland and Denmark. He states that "the mortality from arterio- and cardiosclerosis declined in Finland, Norway and Sweden during the lean years of the war. It is clear that this is associated with a reduced consumption of eggs, butter and other foodstuffs rich in cholesterol." Further, "it is remarkable that no latency period of appreciable length is found between changes of the composition of the diet and displacements in the course of the mortality curves. In Sweden the mortality from arteriosclerosis already fell in 1942, which was the first lean year of the war. . . . We must perhaps take into account the possibility that a meal rich in fatty matter . . . can momentarily induce small atheromatous foci. It is conceivable that such acutely appearing plaques may grow in a short time . . . if such is the case it ought to be of benefit to employ prophylactic measures. We ought to avoid all luxury consumption of high-cholesterol foodstuffs. Especially the combination eggs and cream, butter or other fats would seem to be risky. . . ."[57] Since the aforementioned high cholesterol foods are also high in saturated fats, it may be the saturated fats and not the cholesterol which is the villain.

Drs A. Strom and R.A. Jensen have also investigated the effect of food restriction on mortality from circulatory diseases. They studied this problem in Norway during World War II. They said: "Before the late war, mortality from diseases of the circulatory system was rising each year in Norway. This rise ceased during the war, and from 1941 to 1945 there was a well-marked fall in mortality from these diseases. Since the war there has been a rapid rise in mortality towards the prewar level. The wartime decline coincided with severe dietary restrictions. The supply of calories was reduced, and this reduction consisted principally of foods containing fat, including those rich in cholesterol."[58]

Dr F. Henschen echoes this finding: "The regulation of provisions was no doubt very favorable for the healthy, and the common state of health has never been as good in Sweden as during this time. The mortality decreased to a minimum, owing mainly to a very marked decrease of the mortality from arteriosclerosis and arteriosclerotic heart disease. Also, gallstones were less frequently found at autopsy."[59]

Findings from Leningrad during the siege that occurred soon after the USSR entered the war confirm the above statements. "It is interesting to note the diseases that were reported to be quite rate during the period of semi-starvation. Among these are coronary artery disease, hypertension, and congestive heart failure, gastric and duodenal ulcer and appendicitis, acute nephritis and exacerbations of chronic nephritis, cholecystitis and hepatitis, diabetes mellitus, hyperthyroidism, allergic states, acute upper respiratory infection, and rheumatic fever. Only with improvement of the nutritional state did some of these return to their former prominence in the causes for hospitalization."[60] Tzinzerling showed that clinical atherosclerotic disease became extremely rare during the siege of Leningrad and that autopsy studies including histological examination showed 'cleansing' of the lipid component in plaques.[61]

Other studies on the effect of "undernutrition" on atherosclerosis tend to confirm the observations made during World War II. S.L. Wilens, MD, found that at necropsy (performed on 1250 subjects) severe atherosclerosis was at least twice as common in obese as in undernourished persons 35 or older. This relationship was independent of sex, hypertension, and diabetes. Significantly, a large number of those found to be undernourished at necropsy had been well nourished or even obese prior to the onset of the terminal wasting disease, thus implying resorption of atheromata.[62] A further study by Wilens found that "the general state of nutrition is a factor not only in the development but also in the resolution of human atherosclerotic lesions. . . . A high incidence of severe or even moderate atherosclerotic change is seldom observed in those with protracted undernutrition. . . . Less severe degrees of atherosclerotic change are usually observed in the group with wasting disease than in those without terminal weight loss. It is inferred, therefore, that significant resorption of previously formed atheromatous lesions may occur during periods of marked weight loss . . . less lipid is demonstrable in the intimal lesions of those with terminal weight loss."[63]

Dublin reported decreased organic heart disease, arterial disease, and angina pectoris in underweight people as compared to normal and overweight individuals.[64] Ackerman found at autopsy less atherosclerosis in hearts from underweight as opposed to average or overweight individuals.[65]

Observations indicate a negative correlation between atherosclerosis and carcinoma.[66,67] "Based on material from 1935 autopsies, the authors demonstrate that atherosclerotic lesions are less pronounced in patients suffering from carcinoma than among

non-cancerous persons."[66] To quote another observer of this phenomenon: "It is suggested that this may be the result of alterations in nutrition during a prolonged terminal illness . . . it is possible to suppose that there might actually be some regression of atherosclerotic lesions in the human."[67]

Obviously, no doctor advises that patients contract a severe wasting disease in order to resorb atherosclerotic plaques. What we suggest is that a period of therapeutic fasting might mimic the effect of a wasting disease to the extent that atherosclerotic lesions may be resorbed to a significant degree. Dr M.L. Armstrong, of the Atherosclerotic Research Center and Cardiovascular Division, University of Iowa College of Medicine, asks: "Is it possible to accomplish generally and by more acceptable means what has been reported to occur as a result of wartime privations, cancer, and wasting disease?"[68] Wilens says: "If spontaneous resorption can occur, it is possible that the conditions leading to this event might be simulated."[63]

The possibility of resorption of atheromas is obviously of great interest to scientists today. Dr G.A. Gresham has reviewed the literature to 1976. He describes studies on animals, showing that atheromas may regress, and reviews the literature regarding regression of human lesions in wartime and during the course of chronic wasting diseases. He concludes that "it seems likely that atherosclerosis can slowly regress."[69] Studies with non-human primates have clearly shown that atherosclerosis may be induced by a high cholesterol, high saturated fat diet, and that the atheromata regress upon consumption of a cholesterol free, low saturated fat diet.[70,71,72]

Studies have indicated that fibrinolytic activity may be decreased in atherosclerotic diseases,[73,74] and it has been suggested that the decreased fibrinolytic activity plays a part in the causation of atherosclerosis and thrombosis.[75,76] A 1962 study has shown, that after 36 hours of fasting, there is a statistically significant increase in the fibrinolytic activity of the blood which continued during the following 24 hours, after which the fast was terminated.[77] A 1967 study confirms these findings.[78] Increased fibrinolysis in fasting may partially account for the dramatic improvement in well-being of patients suffering from thrombophlebitis who were fasting for obesity.[39]

Dietary restriction of cholesterol, saturated fats, refined sugars, and salt in the treatment of cardiovascular diseases is a controversial subject, and can be considered but superficially. The physician attending cardiovascular patients should have a plan of dietary intervention to treat such conditions. A discussion of this subject will therefore be presented.

As an introduction, the following quote from an article by W. Dock, MD, entitled, "Atherosclerosis — Why do we pretend the pathogenesis is mysterious?" is appropriate. The author states that "there is no mystery about why the incidence of vascular disease continues to rise . . . human beings, including physicians and informed laymen, are eager for excuses not to face annoying facts and so they continue to do things which are agreeable but hazardous. Like most of my fellow students, I ignored the facts on atherosclerosis in daily life long after I accepted them as proved. Osler, in 1869, concluded that 'angeiosclerosis is the Nemesis through which Nature exacts retributive justice for the violation of her laws.' He considered diet as the most important feature of management, but few heeded the wisdom of the world-famous Hopkins professor. Our incidence of coronary disease will continue to rise as long as our professors seek and expound ephemeral theories of pathogenesis and our profession denies the importance of 'stuffing, sitting, smoking and sipping'. . . . It is almost impossible for most men to accept any suggestion that it might be wise to give up agreeable habits such as smoking or eating favorite foods . . . that is why we who had been shown exactly how to produce it a half a century ago continue to pretend that atherosclerosis is mysterious."[79]

A considerable body of research and a wide spectrum of opinion in the scientific community supports dietary restriction of cholesterol, saturated fats, refined carbohydrates, and excess calories. The risk factor hypothesis is used as a guideline in formation of recommendations to patients; it states that the following factors, in rough order of their importance, are positively correlated with cardiovascular diseases: hyperlipidemia, hypertension, cigarette smoking, diabetes mellitus, physical inactivity, obesity, emotional stress, and positive family history of premature atherosclerosis.[80] W.B. Kannel, MD, a principal architect of the Framingham study which established coronary risk factors, states: "Who can seriously argue with the general merit of a program that advocates maintenance of lean body weight, a less rich diet, physical fitness, giving up cigarettes, and control of hypertension, diabetes and hyperlipidemia?"[81]

Dr W. Walker has shown that a weight reduction diet, not extremely low in lipids, will, regardless, result in decreased serum cholesterol and lipoprotein levels. This reflects the importance of total calorie intake.[82,83]

E. Corday, MD, while indicating that the mechanism of atherosclerosis and risk factors is not understood, states that "most physicians agree that there is no harm in advising the patients to follow a prudent diet and life style . . . most investigators agree that the risk factor hypothesis has provided valuable information and to be successful must be applied well before clinical evidence becomes manifest."[84]

D.S. Fredrickson, MD, director of the National Institutes of Health, says: "The risk factors have been shown to be associated with an increased incidence of premature ischemic heart disease (IHD). Being amenable to some control, they are considered in preventive management of IHD."[80]

In February 1977, the Select Committee on Nutrition and Human Needs of the US Senate published a report entitled *Dietary Goals for the US*. In this report, Senator George McGovern says that "our dietary changes in the past 50 years represent as great a threat to public health as smoking. Too much fat, too much sugar or salt, can be and are linked directly to heart disease, cancer, obesity, and stroke, among other killer diseases." Also in the report, Dr D.M. Hegsted, professor of Nutrition, Harvard School of Public Health, states that "the risks associated with eating this diet (rich in meat and other sources of saturated fat and cholesterol, and in sugar) are demonstrably large. The question to be asked, therefore, is not why should we change our diet, but why not?" He also says: "Ischemic heart disease, cancer, diabetes and hypertension are the diseases that kill us. They are epidemic in our population. We cannot afford to temporize. We have an obligation to inform the public of the current state of knowledge and to assist the public in making the correct food choices. To do less is to avoid our responsibility."

Dr G. Briggs, professor of Nutrition at the University of California, Berkeley, in his testimony before the senate committee in 1972, estimated, based on a study by the department of Agriculture, that "improved nutrition might cut the nation's health bill by one-third." The general conclusion of the report states that "there is a great deal of evidence . . . which strongly implicates, and, in some instances, proves, that the major causes of death and disability in the United States are related to the diet we eat. . . . The over-consumption of fat, generally, and saturated fat in particular, as well as cholesterol, sugar, salt and alcohol have been related to six of the ten leading causes of death: heart disease, cancer, cerebrovascular disease, diabetes, arteriosclerosis, and cirrhosis of the liver."[85]

Recently, with the development of the lipoprotein hypothesis, dietary recommendations have become more specific. This hypothesis states lipoprotein levels, not serum cholesterol levels, are more accurate in reflecting risk of cardiovascular disease. For example, if serum cholesterol levels are elevated along with increased levels of high density lipoproteins, there is much less risk of atherosclerosis than if high serum cholesterol levels exist with increased levels of low density lipoproteins.[86] This new outlook also supports the risk factor hypothesis. Type II and type IV hyperlipoproteinemias, the most common types, are treated correspondingly with the risk factor hypothesis. Type II is treated with a low-cholesterol, low saturated fat diet. Type IV is treated with a controlled carbohydrate, moderately restricted cholesterol diet with substitution of polyunsaturated fats for saturated fats and elimination of most concentrated sweets; also, these patients are advised to achieve and maintain ideal weight.[87]

Zelis, by measuring peripheral blood flow, has reported objective evidence of improved circulation in patients following therapy aimed at reduction of serum lipids. He believes this improvement resulted from resorption of cholesterol-rich atheromata and not improved collateral circulation since the patients were not on an exercise program. Improved blood flow was accompanied by improvement in angina symptoms.[88]

Other investigators have reported similar results following surgical portacaval shunt[89] and partial ileal bypass.[90]

No doubt can exist of the effect of postprandial hyperlipidemia in patients with cardiovascular pathologies. By direct observation of bulbar conjunctival vessels, researchers found that "following the ingestion of fat-enriched meals, there was increased intravascular agglutination, slowed blood flow, plugging of some small vessels, venule sacculations, occasionally petechial hemorrhage, and anginal pain in some cases. Two of these patients placed on low fat diet more than two months ago report no recurrence of angina."[91] A later paper by the same researchers also presents evidence that ingested fat can contribute to reduction of coronary blood flow by an **acute** effect, "an immediate altering of the physical consistency of the blood, by red cell agglutination, causing the blood to become more resistant to passage through the narrowest vessels. This is in addition to the well-known slowly developing atherosclerotic narrowing of coronary arteries of various sizes."[92] Direct microscopic examination of bulbar conjunctival vessels was also employed in this study.

Other scientists induced lipemia in patients with angina pectoris by a "standard fat meal" and noted that "attacks of anginal pain would invariably occur at or near the peak of the lipemic curve."[93] Further research shows that postprandial lipemia causes decreased coronary blood flow and decreased heart oxygen consumption in normal individuals.[94] Many other studies support these findings.[95,96,97,98,99,100]

With this evidence of an acute effect of a high fat meal in patients with atherosclerotic cardiovascular disease, there should be no hesitation in recommending that such individuals consume a low fat diet.

Hypertension

This discussion is concerned with primary (essential) hypertension; that for which no cause can be found. Approximately 90% of all hypertensive patients suffer from the primary form.[101]

A period of therapeutic fasting will lower blood pressure even where severe hypertension exists. In a review of the application of fasting for obesity, the conclusion regarding blood pressure is: "Fasting is usually accompanied by a gradual decrease in systolic and diastolic blood pressure, and even severely hypertensive patients may become normotensive."[31] Another study with 683 obese patients noted that: "Arterial hypertension was present prior to the fast in 48% of the subjects. It was rare that these patients did not become normotensive within two to four days

after embarking on the fast."[102] Other studies have reported this same finding with both obese[14,29,37,103] and non-obese[20,104,105] hypertensives.

Much information is available on the effect of food restriction on hypertension. This dietary manipulation has been shown to lower blood pressure. Brozek, *et al*, fed 16 hundred calories to 34 normal young men for six months; body weight declined 23.0%, systolic pressure fell 11.1%, diastolic pressure, 7.7%, basal metabolic rate by 39.9%. The conclusion was: "That drastic dietary restriction causes a fall in blood pressure in most normal persons and persons with hypertensive disease seems to us to be proved. . . . this provides considerable support for the treatment of certain cases of hypertension by vigorous dietary limitation."[106]

A report from Holland after World War II states that, "during the severest hunger period the average systolic as well as the average diastolic blood pressure were definitely lowered. In case of considerable loss of weight, decrease of the systolic pressure was found to be most frequent in hypertensive subjects and least in hypotensive subjects. The diastolic pressure showed corresponding changes."[107]

Dr J. Hartsilver treated 48 cases of hypertension with a "semistarvation diet" consisting of 600 calories from fruit juices, vegetable soup, and fruit. His conclusion: "The effect of the diet has been, in a very short time (a few weeks only), to reduce the BP to little above normal, with complete disappearance of symptoms (headache, dyspnea, insomnia, and giddiness)."[108]

Kempner treated hypertensive patients with a "rice-fruit" diet containing about 2000 calories, 15 to 20 grams of protein, .15 gram of sodium, and no more than five grams of fat per day. This diet was successful in lowering the BP in 60% of the patients.[109]

A few possible mechanisms exist whereby fasting might cause the blood pressure to drop. First, during a fast the heart size[110] and the cardiac output[111] fall. Since the BP is equal to the sum of the cardiac output times the total peripheral resistance, decreasing the cardiac output will decrease the BP. Second, during a fast there is a tremendous diuresis of water and sodium (as previously discussed) and this causes a decrease in the blood volume.[110,111] Accumulation of sodium and the water which osmotically follows it is considered a major cause of hypertension,[112] and, for this reason, hypertensives are put on a low sodium diet.[113] It is important to note again that the loss of sodium during a fast exceeds by three times the loss during a low salt diet, and that this loss is abruptly halted by the ingestion of a small amount of carbohydrate (see above).

Another explanation why the BP drops during a fast is the decreased heart rate which occurs.[32,33,114] Cardiac output, on which BP depends is equal to the heart rate times the stroke volume; therefore, with a decrease in the heart rate, the cardiac output will also decrease and this, in turn, will cause a decrease in the BP. A final reason why BP drops as a result of fasting is the concommitant weight loss. A recent report shows that 75% in one group and 61% in another group regained normal BP after losing weight, and this was independent of any effect from decreased sodium intake.[115]

It may be concluded from this section that, in the treatment of essential hypertension, an initial period of fasting will lower the blood pressure to normal levels, and a subsequent diet greatly restricted in calories will maintain the BP at this decreased level.

Heart Failure — Cardiac Insufficiency

Heart failure is defined as "the pathophysiologic state in which an abnormality of cardiac function is responsible for the failure of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues."[116] Treatment is essentially three-pronged: reduction of cardiac work load, enhancement of myocardial contractility, and control of excessive fluid retention.[117]

The cardiac work load may be effectively reduced through the use of fasting and food restriction. Accompanying both of these dietary changes is a decrease in the basal metabolic rate; this results in slowing of the heart rate[118,119,120] which, in turn, decreases the cardiac output. The blood pressure also decreases while fasting or restricting food intake. These changes in the cardiovascular system reduce the cardiac workload.

Enhancement of myocardial contractility is accomplished medically by use of cardiac glycosides, mainly digitalis. This therapy increases contractility by stimulating the heart muscle and, in some severe cases, is not effective.[121] In a life-or-death situation, digitalis often preserves life, but is effective only to the degree that the heart has the functional reserve to respond to the drug with increased contractility; digitalis does not add to the functional reserve of the myocardial cells. For this reason, digitalis should ideally be withdrawn as soon as some degree of normal activity may be undertaken without it.

Concurrent with the use of digitalis, food intake should be greatly restricted; nevertheless, fasting may be dangerous. Over a period of time, the needs of the circulation will often decrease to the point that the heart will be able to handle its workload without the stimulation provided by digitalis; fasting therapy may now be instituted. If resorption of atheromata does occur with the aforementioned dietary restrictions, and the evidence strongly indicates that it does, the heart muscle will gradually be supplied with an increased amount of nutrients and oxygen; this may be expected to increase myocardial contractility.

Control of excessive fluid retention, the third aspect of heart failure treatment, is accomplished quite well by fasting. The loss of sodium and water in the fast has been discussed extensively.

A.J. Merrill, MD, applied fasting in the treatment of

intractable heart failure. Sometimes, he says, "digitalization, bed rest, low-salt diet, and diuretics no longer suffice to maintain compensation in patients who are in heart failure. The body begins to retain water . . . recognized as the terminal phase in patients with fixed, severe chronic heart failure." One patient was given only coffee and water in addition to her previous therapy and lost 21½ pounds in five days; she became fully compensated at rest. Another patient, an 84 year old woman, also was failing to maintain compensation on digitalis and diuretics. Her heart was enlarged, and she was experiencing many of the accompaniments of the terminal phase of heart failure.

She was given only water and in five days progressed from a state of "disoriented, almost comatose" to being "quite bright." The patient was then placed on a 200 mg sodium diet with limited water intake, and was discharged 12 days later "alert and well oriented." The change in this patient resulting from five days of fasting was reported as "spectacular." Dr Merrill reports the results with two other patients with severe congestive heart diseases. He says: "One showed an excellent response. The other exhibited very little change. The latter, however, was jaundiced, possibly from a combination of congestive heart failure with pulmonary emboli, and had a low serum albumin."[121]

D. Stechschulte, MD, and Marvin Dunn, MD, FACP, also used fasting to treat four patients with severe heart trouble. These patients "were selected for study only after they became refractory to usual diuretic management." While fasting, digitalis administration was continued. The results of this therapy in most cases were positive; the authors concluded that "we wish to emphasize the efficacy of the starvation diet in the treatment of selected cases of congestive failure." Nevertheless, it is also noted that an additional patient with severe heart decline fasted with "resultant diuresis, hemoconcentration, uremia, and death." This investigation indicates the hazards of applying fasting therapy plus digitalis to patients with severe heart complications. A low calorie, low salt diet would probably be a safer procedure in these severely ill cases, with fasting therapy reserved for cases of milder heart trouble.[122]

Japanese physicians employed fasting and reported an 87% efficacy rate in treatment of mild coronary insufficiency, extrasystoles, and the Wolff-Parkinson-White syndrome.[20] Obese patients with "cardiovascular embarrassment," fasting to lose weight, experienced prompt improvement in their symptoms.[19] Another study reports significant improvement in patients with angina pectoris and chronic myocardial insufficiency.[102]

Other researchers have studied the effect of dietary restriction on cardiac pathology. Kempner, employing the rice-fruit diet (see section on hypertension) in the treatment of heart failure, noted that "the enlarged heart became smaller, the inverted T waves became upright, and the signs and symptoms of cardiac failure disappeared."[123]

A.M. Masters, MD, employed for many years an 800 calorie diet in the treatment of ischemic heart disease. In two articles, Masters reports his results with 127 patients; the majority had just experienced an acute myocardial infarction; the others were suffering from angina but were ambulatory.

Clinical improvement was observed in all patients. In the first series of MI patients, most patients were able to resume normal work after a period of rest. He says: "The low calorie diet is not merely a scientific theory, for it has been observed clinically that patients lose their pain more quickly and are less apt to have gastrointestinal complaints. Their vital capacity, blood velocity, venous pressure and blood pressure return more quickly to normal."[124] In the second article, Masters shows that there was a reduction of 49% in the workload of the heart with the low calorie diet, and this "often relieved the symptoms of heart disease." He notes that the lowered basal metabolic rate achieved by the diet "had a beneficial effect on the cardiovascular system, resulting in slowing of the pulse rate, decrease in blood pressure, and pulse pressure and diminution of the cardiac output and work of the heart." No ill effects of the diet were found.[125]

Other aspects of the physiology of food consumption affect the heart. The cardiac output "rises immediately after the ingestion of food, reaches a maximum of 0.5 to 2.0 liters per minute over the fasting level, and remains at this high level for one to three hours. Four to five hours after the ingestion of a light meal, the cardiac output has returned to its fasting level."[126] Normal cardiac output is about 5.5 liters per minute, so the rise after eating is significant. It is important to emphasize that the rise even occurs with a light meal. This is another reason why a person with a failing heart would benefit from fasting.

Experiments have shown that distending the stomach will cause a 15 to 35% decrease in coronary blood flow through a vagovagal reflex and, by this mechanism, "an attack of angina pectoris might be precipitated."[127] Another report has noted that a myocardial infarction may occur because of overeating in the presence of abnormal coronary arteries.[128] Ischemic heart disease patients should be warned of these possibilities.

Clinicians should be warned that certain variations in the fasting program presented in this paper may be dangerous. Diuretics given during fasting have occasionally resulted in death.[129] Fasting with the addition of small amounts of protein has been fatal; this is, however, not a true fast but a low protein diet.[130] Exercise, a definite contraindication during a prolonged fast, has resulted in onset of arrhythmias, but the heart rhythm returned to normal after refeeding.[102,131]

Method of Therapeutic Fasting

The details of this method of fasting come from the

in-patient institute owned and operated by D.J. Scott, DC, in Cleveland, Ohio.[7] Since 1957, more than 10,000 patients have been treated by fasting in this institution to treat a wide range of problems.

In preparation for fasting, a careful drug history must be taken. Patients who cannot give up their medications for the period of the fast, such as those dependent on adrenal steroids and insulin, are not accepted as subjects for this treatment. To prepare for fasting, patients should eat a diet of raw fruits and vegetables for a few days. This promotes elimination and makes the transition into the fasting state a little easier; however, it is not essential.

After a thorough diagnostic workup, patients are admitted to the institute. Fasting is begun immediately, thus no food or juice is allowed; complete physical and psychological rest is prescribed. Drinking distilled water, to relieve thirst, is allowed.

During the fast, vital signs and weight are checked once each day and multiple blood chemistries plus CBC once each week. Distilled water enemas are used if the patient has an impacted bowel or is vomiting and needs hydration. Elimination is greatly attenuated during the fast; it is normal for the patient to have few or no bowel movements, however signs and symptoms of constipation or toxemia are not observed. After the fast, the bowel movements usually become normal within one to five days if the patient is on a diet of raw fruit and vegetables; the only exception to this is in the presence of remaining pathology (especially rectal). The quantity of urine decreases during the fast, the result of decreased water drinking; no discomforts associated with urinary function are experienced in the absence of previous GU pathology.

When the fast is broken depends on many factors including the amount of time the patient has available for fasting (determined by work schedule, family demands, etc), the amount of money the patient has, presence of psychological crises, or onset of various physiological signs and symptoms. The fast is terminated either when the body has completed its self-cure, or when signs of shortage of reserves are observed. Such shortage is reflected in changes in the vital signs: BP below 70/50 in the supine position (this value obviously depends on the levels of the BP at the start of the fast — it may be necessary to terminate the fast at a BP of 150/100 in a patient who began fasting with a BP of 200/150); heart rate below about 35 beats per minute (this, as with BP, must be evaluated on an individual basis — the presence of signs of circulatory failure with shock, not the level of heart rate or BP, is the important criteria); decrease in body temperature (this is not usually significant unless the decrease is of a great magnitude — patients are normally intolerant of cold during the fast because of the decreased basal metabolic rate, and may only need exposure to an external source of warmth). Evidence that the body has completed its self-healing process comes through observation of the blood chemistries or signs and symptoms. The return of metabolic efficiency may be reflected by onset of hunger after a long period of anorexia (often this hunger is 'compulsive' in nature — the patient may even dream about food), and reappearance for the first time in many days of a pleasant taste in the mouth accompanied by an unoffensive breath.

The fast is broken with undiluted, freshly squeezed orange juice, four ounces every three hours for the first day. California, not Florida, citrus is used as this has been found to be less acidic. The juice supplies the patient with a rapidly absorbed source of glucose, the primary need at this time. On the second day, eight to ten ounces of freshly squeezed juice are fed every three hours; orange juice is alternated with vegetable juice (3 ounces carrot, 4 ounces celery, 1 ounce romaine). Bean sprout juice may be added if the doctor determines that the patient has a need for extra vitamins. The juices restore fluid and electrolytes lost during the fast.

On day three, whole food is usually served. This would definitely be the case if the fast had lasted about three weeks; if longer than this, it may be desirable to extend the juice diet a few days. Breakfast consists of citrus: two oranges, one grapefruit, and one slice of pineapple; these must be totally ripe and not sour. Lunch consists of vegetable juice, subacid fruit (two apples, one pear, and one-half pound of grapes), plus one whole avocado (this supplies easily used fat). Dinner is a tossed salad consisting of a base of lettuce (anything but iceberg; this has been found to disagree with many patients; romaine is generally used) with celery, cucumber, red sweet pepper, mung bean and alfalfa sprouts, shredded carrots or beets, and avocado dressing. At least one pound of salad is eaten; the maximum amount allowable is to the point of satisfaction (patients almost never overeat after a long fast). Additional foods are added in the following days according to the metabolic type and energy expenditures.

Certain variations in the fast-breaking regimen are occasionally necessary. With persistance of inflammatory conditions, especially of the gastrointestinal tract, orange juice may intensify symptoms; thus, other juices are used. If the patient has a renal problem with oliguria, melon juice may be used; this has been found to have the best effect on urine output. In the case of a peptic ulcer, the fast may be broken with whole avocado and potato with warm vegetable juices.

Case Reports

The following cases are from Dr. Scott's institution.[7]

- Case 1: M.Z., a 63 year-old female, entered the institution on March 14, 1977 to be treated for hypertension, hyperlipidemia, and cardiomegaly. History: herpes zoster one month prior to admission, diagnosis of diverticulosis in 1975, obesity for ten years, chronic ear-nose-throat infections. On ad-

mission, weight was 168 pounds, height 5 feet 4 inches, BP 160/90, triglycerides (TG) 601, cholesterol (C) 338. Cardiac monitoring revealed an arrhythmia. Patient fasted for 13 days; weight now 151 pounds, BP 120/85. Patient then ate a diet of raw fruit and vegetables for eight days; weight now 153 pounds, BP 110/75. Patient was discharged; returned May 16, 1977 (at 147 pounds) to fast again. This fast lasted ten days; weight now 136 pounds, BP 115/80. Another period of eating ensued and lasted for nine days; weight now 139 pounds, BP 110/70. Cardiomegaly as evidenced by radiography was corrected, and normal heart rhythm was restored. TG 374 and C 264 at discharge.

- Case 2: A.B., a 60 year-old female, entered the institution April 14, 1977 to be treated for angina pectoris. Medical physicians previously attending A.B. had considered her a candidate for coronary bypass surgery, but had rejected this possibility as too risky in this case. On April 14 her weight was 98 pounds, height 5 feet 4 inches, BP 130/70, glucose 61mg%, BUN 22, uric acid 9.5. Fast was begun and lasted for 24 days. At completion, weight was 82 pounds, BP 100/50, glucose 64, BUN 24, uric acid 9.8. Patient remained at the institution on raw fruit and vegetables for 19 days; weight now 88 pounds, BP 115/50, glucose 86, BUN 9, uric acid 4.0. Patient was discharged at this time having experienced a complete recovery, and was now capable of assuming normal activities. Followup examination eight weeks later: BP 130/70, gains achieved during therapy had been maintained.
- Case 3: A.Q., a 34 year-old male, entered the institution on October 7, 1976 with severe angina pectoris. History — patient had experienced severe pain with even mild exertion, such as walking from car to house; also familial hypercholesterolemia — daughter had C level of 1000 at age 10. On admission, weight was 142 pounds, height 5 feet 8 inches, BP 120/80, C 494, uric acid 8, calcium 7.9. Patient fasted for 37 days; weight now 112 pounds, BP 95/60, C 467 (with peak of 666 on day eight of fast), uric acid 10, calcium 10. Patient fed raw fruit and vegetable diet for 12 days; weight now 115½ pounds, BP 130/70, C 442, uric acid 4.3, calcium 9.2. Patient was discharged; condition now characterized by complete absence of angina pectoris pains, running for 0.6 mile was accomplished painlessly.

Discussion

Fasting is not a new therapy. It was used by Plato and Socrates to "attain mental and physical efficiency," by Pythagoras to prepare for examinations at the University of Alexandria, by ancient Egyptians to treat syphilis, by Hippocrates, Asclepiades, Thessalus, Celsus, Avicenna, Tertullian, and Plutarch. Paracelsus called fasting "the great remedy." Hundreds of scientists have studied fasting. Since the nineteenth century, clinicians have used fasting in the US; such therapists include Jennings, Shew, Trall, Walter, Tanner, Dewey, Graham, Hazzard, Carrington, Bergholtz, Tilden, Hay and Weger. At the present time Drs Shelton, Esser, Benesh, McEachen, Gross, Scott, Cinque, Vetrano and others are employing fasting therapy.[132]

This paper has attempted to establish the scientific rationale for the use of fasting by discussing the metabolism of this physiological state, and the basis for its use in the treatment of cardiovascular diseases. Findings reported herein are significant enough that the therapies of fasting and food restriction should, at least, be seriously investigated. Nevertheless, with the weight of evidence presented, perhaps the clinician would be wise to immediately but carefully begin to apply these dietary therapies. If the diseases discussed can be treated with success in the absence of surgery and drugs with their attendant hazards, a milestone in health care will have been achieved. Fasting and food restriction are valuable in that they allow the body to remove the causes of disease; these are not symptomatic treatments.

Summary

1. The metabolic state of fasting and food restriction is such that approximately 90% of the energy needs are satisfied by the oxidation of lipid, thus sparing the body's protein and prolonging survival. Fasting is generally well tolerated despite the presence of minor side effects.
2. Biochemical and metabolic studies, together with observations of populations during wartime and at autopsy indicate that fasting and dietary restrictions may be expected to have a beneficial effect in cases of atherosclerosis, hypertension, and heart failure.
3. A method for conducting a therapeutic fast and three cases so treated have been presented. □

Bibliography

1. Thorn, G.W., Adams, R.D., Bruanwald, E., Isselbacher, K. J. and Petersdorf, R.G.: *Harrison's Principles of Internal Medicine*, 8th ed, McGraw-Hill Book Company New York, 1977, p. 1307.
2. Thorn, *ibid*, p. 1301.
3. Runcie, J. and Miller, V.: "Treatment of Obesity by Total Fasting for up to 249 Days," *Lancet*, Vol 2, 1966, pp 992-996.
4. Stewart, W.K. and Fleming, L.W.: "Features of a successful therapeutic fast of 382 days' duration," *Postgraduate Medical Journal*, Vol 49, 1973, pp 203-209.
5. Saudek, C.D. and Felig, P.: "The Metabolic Events of Starvation," *American Journal of Medicine*, Vol 60, 1976, pp 117-126.
6. Cahill, G.F. and Owen, O.E.: "Starvation and Survival," *Transactions of the American Clinical and Climatological Association*, Vol 79, 1967, pp 13-20.
7. Scott, D.J.: personal communication.
8. Benedict, F.G.: "Chemical and Physiological Studies of a Man Fasting Thirty-One Days," *Proceedings of the National Academy of Sciences*, Vol 1, 1915, pp 228-231.
9. Felig, P., Owen, O.E., Morgan, A.P. and Cahill, G.F.: "Utilization of Metabolic Fuels in Obese Subjects," *American Journal of Clinical Nutrition*, Vol 21, 1968, pp 1429-1433.
10. Owen, O.E., Morgan, A.P., Kemp, H.G., Sullivan, J.M., Herrera, M.G. and Cahill, G.F.: "Brain Metabolism During Fast-

ing," *Journal of Clinical Investigation*, Vol 46, 1967, pp 1589-1595.
11. Runcie, J. and Hilditch, T.E.: "Energy Provision, Tissue Utilization, and Weight Loss in Prolonged Starvation," *British Medical Journal*, Vol 2, 1974, pp 352-356.
12. Mountcastle, V.B.: *Medical Physiology*, 13th ed, C.V. Mosby Company, St Louis, 1974, p. 1239.
13. Cahill, G.F., Herrera, M.G., Morgan, A.P., Soeldner, J.S., Steinke, J., Levy, P.L., Reichard, G.A. and Kipnis, D.M.: "Hormone-Fuel Interrelationships during Fasting," *Journal of Clinical Investigation*, Vol 45, 1966, pp 1751-1769.
14. Drenick, E.J., Swendseid, M.E., Blahd, W.H. and Tuttle, S.G.: "Prolonged Starvation as Treatment for Severe Obesity," *JAMA*, Vol 187, 1964, pp 100-105.
15. Folin, O. and Denis, W.: "On Starvation and Obesity, With Special Reference to Acidosis," *Journal of Biological Chemistry*, Vol 21, 1915, pp 183-192.
16. Rapoport, A., From, G.L.A. and Husdan, H.: "Metabolic Studies in Prolonged Fasting. I. Inorganic Metabolism and Kidney Function," *Metabolism*, Vol 14, 1965, pp 31-46.
17. Schauf, G.E.: "The Caloric Theory Does Not Apply to Obesity," *ACA Journal of Chiropractic*, Vol 15, 1978, pp 31-33, 50-51.
18. Grande, F.: "Energy Balance and Body Composition Changes," *Annals of Internal Medicine*, Vol 68, 1968, pp 467-480.
19. Drenick, *op cit*, pp 100-105.
20. Suzuki, J., Yamauchi, Y., Horikawa, M. and Yamagata, S.: "Fasting Therapy For Psychosomatic Diseases With Special Reference to Its Indication and Therapeutic Mechanism," *Tohoku J Exp Med*, Vol 118 (Suppl), 1976, pp 245-259.
21. Hoeffel, G. and Moriarty, M.: "The Effect of Fasting on the Metabolism of Epileptic Children," *American Journal of Diseases of Children*, Vol 28, 1924, pp 16-24.
22. Adibi, S.A.: "Influence of dietary deprivations on plasma concentration of free amino acids of man," *Journal of Applied Physiology*, Vol 25, 1968, pp 52-57.
23. Bloom, W.L. and Mitchell, W.: "Salt Excretion of Fasting Patients," *Archives of Internal Medicine*, Vol 106, 1960, pp 321-326.
24. Gamble, J.L.: "Physiological Information Gained From Studies on the Life Raft Ration," *The Harvey Lectures*, Vol 42, 1946-1947, pp 247-273.
25. North, K.A.K., Lascelles, D. and Coates, P.: "The Mechanisms By Which Sodium Excretion is Increased During a Fast But Reduced on Subsequent Carbohydrate Feeding," *Clinical Science and Molecular Medicine*, Vol 46, 1974, pp 423-432.
26. Weinsier, R.L.: "Fasting—A Review with Emphasis on the Electrolytes," *American Journal of Medicine*, Vol 50, 1971, pp 233-240.
27. Spark, R.F., Arky, R.A., Boulter, P.R., Saudek, C.D. and O'Brian, J.T.: "Renin, Aldosterone and Glucagon in the Natriuresis of Fasting," *New England Journal of Medicine*, Vol 292, 1975, pp 1335-1340.
28. Carlson, A.J.: "Contributions to the Physiology of the Stomach," *American Journal of Physiology*, Vol 45, 1918, pp 120-145.
29. Duncan, G.G., Jenson, W.K., Cristofori, F.C. and Schless, G.L.: "Intermittent Fasts in the Correction and Control of Intractable Obesity," *American Journal of the Medical Sciences*, Vol 245, 1963, pp 515-520.
30. Keys, A.: "Caloric Undernutrition and Starvation, with Notes on Protein Deficiency," *JAMA*, Vol 138, 1948, pp 500-511.
31. Bray, G.A., Davidson, M.B. and Drenick, E.J.: "Obesity: A Serious Symptom," *Annals of Internal Medicine*, Vol 77, 1972, pp 797-805.
32. Young, D.R.: "Effect of food deprivation on Treadmill Running in Dogs," *Journal of Applied Physiology*, Vol 14, 1959, pp 1018-1022.
33. Palmblad, J., Levi, L., Burger, A., Melander, A., Westgren, U., von Schenck, H. and Skude, G.: "Effects of Total Energy Withdrawal (Fasting) on the Levels of Growth Hormone, Thyrotropin, Cortisol, Adrenaline, Noradrenaline, T4, T3, and rT3 in Healthy Males," *Acta Med Scand*, Vol 201, 1977, pp 15-22.
34. Kunde, M.: "The After-effect of Prolonged Fasting on the Basal Metabolic Rate," *Journal of Metabolic Research*, Vol 3, 1923, pp 448-449.
35. Bloomer, J.R., Barrett, P.V., Rodkey, F.L. and Berlin, N.I.: "Studies on the Mechanism of Fasting Hyperbilirubinemia," *Gastroenterology*, Vol 61, 1971, pp 479-487.
36. Barrett, P.V.D.: "Hyperbilirubinemia of Fasting," *JAMA*, Vol 217, 1971, pp 1349-1353.
37. Runcie, J., Thomson, T.J.: "Prolonged Starvation—A Dangerous Procedure?" *British Medical Journal*, Vol 3, 1970, pp 432-435.
38. Rozental, P., Biava, C., Spencer, H. and Zimmerman, H.J.: "Liver Morphology and Function Tests in Obesity and During Total Starvation," *American Journal of Digestive Diseases*, Vol 12, 1967, pp 198-208.
39. Lawlor, T., Wells, D.G.: "Metabolic Hazards of Fasting," *American Journal of Clinical Nutrition*, Vol 22, 1969, pp 1142-1148.
40. Hoeffel, G., *op cit*, pp 16-24.
41. Benedict, F.G. and Roth, P.: "Effects of a Prolonged Reduction in Diet on 25 Men. I. Influence on Basal Metabolism and Nitrogen Excretion," *Proceedings of the National Academy of Sciences*, Vol 4, 1918, pp 149-159.
42. Allen, F.M.: "Prolonged Fasting in Diabetes," *American Journal of Medical Sciences*, Vol 150, 1915, pp 480-485.
43. Bloom, W.L.: "Fasting as an Introduction to the Treatment of Obesity," *Metabolism*, Vol 8, 1959, pp 214-220.
44. Duncan G.G., *op cit*, pp 515-520.
45. Sievers, M.L. and Hendrikx, M.E.: "Therapeutic Starvation," *British Medical Journal*, Vol 3, 1974, pp 524-525.
46. Suzuki, J., *op cit*, pp 245-259
47. Hollifield, G., Owen, J.A., Lindsay, R.W. and Parson, W.: "Effects of Prolonged Fasting on Subsequent Food Intake in Obese Humans," *Southern Medical Journal*, Vol 57, 1964, pp 1012-1016.
48. Jagannathan, S.N., Connor, W.E., Baker, W.H. and Bhattacharyya, A.K.: "The Turnover of Cholesterol in Human Atherosclerotic Arteries," *Journal of Clinical Investigation*, Vol 54, 1974, pp 366-377.
49. Chobanian, A.V. and Hollander, W.: "Body Cholesterol Metabolism in Man. I. The Equilibration of Serum and Tissue Cholesterol," *Journal of Clinical Investigation*, Vol 41, 1962, pp 1732-1737.
50. Field, H., Swell, L., Schools, P.E. and Treadwell, C.R.: "Dynamic Aspects of Cholesterol Metabolism in Different Areas of the Aorta and Other Tissues in Man and Their Relationship to Atherosclerosis," *Circulation*, Vol 22, 1960, pp 547-558.
51. Gould, R.G., Jones, R.J. and Wissler, R.W.: "Lability of Cholesterol in Human Atherosclerotic Plaques," *Circulation*, Vol 20, 1959, p. 967.
52. Orten, J.M. and Newhaus, O.W.: *Human Biochemistry*, 9th ed, C.V. Mosby Company, St Louis, 1975, p. 285.
53. Kartin, B.L., Man, E.B., Winkler, A.W. and Peters, J.P.: "Blood Ketones and Serum Lipids in Starvation and Water Deprivation," *Journal of Clinical Investigation*, Vol 23, 1944, pp 824-835.
54. Ende, N.: "Starvation Studies With Special Reference to Cholesterol," *American Journal of Clinical Nutrition*, Vol 11, 1962, pp 270-280.
55. Ende, N.: "Serum Cholesterol in Acute Starvation: A Report of 20 Cases," *Journal of Nutrition*, Vol 71, 1960, pp 85-90.
56. Keys, A.: "Dietary Factors in Atherosclerosis," in *Cowdry's Arteriosclerosis*, H.T. Blumenthal (Ed), Charles C. Thomas, Springfield, Illinois, 1967, p. 585.
57. Malmros, H.: "The Relation of Nutrition to Health," *Acta Med Scand*, Vol 246 (suppl), 1950, pp 137-153.
58. Strom, A. and Jensen, R.A.: "Mortality from Circulatory Diseases in Norway 1940-1945," *Lancet*, Vol 1, 1951, pp 126-129.
59. Henschen, F.: "Pathogenesis of Arteriosclerosis in the Light of Food Restriction in Scandinavia During the War," *Journal of Gerontology*, Vol 6, 1951, p. 102.

60. Brozek, J., Wells, S. and Keys, A.: "Medical Aspects of Semistarvation in Leningrad," *American Review of Soviet Medicine*, Vol 4, 1946-7, pp 70-86.
61. Tzinzerling, V.D., Zakhar'evskaia, M.A. and Volkova, K.G., in A.L. Myasnikov, *Atherosclerosis*, p. 72, 1962. Translated from the Russian. Bethesda, Maryland, National Institutes of Health.
62. Wilens, S.L.: "Bearing of General Nutritional State on Atherosclerosis," *Archives of Internal Medicine*, Vol 79, 1947, pp 129-147.
63. Wilens, S.L.: "The Resorption of Arterial Atheromatous Deposits in Wasting Disease," *American Journal of Pathology*, Vol 23, 1947, pp 793-804.
64. Dublin, L.I.: "The Influence of Weight on Certain Causes of Death," *Human Biology*, Vol 11, 1930, pp 159-184.
65. Ackerman, R.F., Dry, T.J. and Edwards, J.E.: "Relationship of Various Factors to the Degree of Coronary Atherosclerosis in Women," *Circulation*, Vol 1, 1950, pp 1345-1354.
66. Wanscher, O., Clemmesen, J. and Nielsen, A.: "Negative Correlation Between Atherosclerosis and Carcinoma," *British Journal of Cancer*, Vol 5, 1951, pp 172-174.
67. Lober, P.H.: "Pathogenesis of Coronary Sclerosis," *AMA Archives of Pathology*, Vol 55, 1953, pp 357-383.
68. Armstrong, M.L.: "Evidence of Regression of Atherosclerosis in Primates and Man," *Postgraduate Medical Journal*, Vol 52, 1976, pp 456-461.
69. Gresham, G.A.: "Is Atheroma a Reversible Lesion?" *Atherosclerosis*, Vol 23, 1976, pp 379-391.
70. Armstrong, M.L., Warner, E.D. and Connor, W.E.: "Regression of Coronary Atheromatosis in Rhesus Monkeys," *Circulation Research*, Vol 27, 1970, pp 59-67.
71. Tucker, C.F., Catsulis, C., Strong, J.P. and Eggen, D.A.: "Regression of Early Cholesterol-Induced Aortic Lesions in Rhesus Monkeys," *American Journal of Pathology*, Vol 65, 1971, pp 493-514.
72. Kokatnur, M.G., Malcom, G.T., Eggen, D.A. and Strong, J.P.: "Depletion of Aortic Free and Ester Cholesterol by Dietary Means in Rhesus Monkeys with Fatty Streaks," *Atherosclerosis*, Vol 21, 1975, pp 195-203.
73. Nestel, P.J.: "Fibrinolytic Activity of the Blood in Intermittent Claudication," *Lancet*, Vol 2, 1959, p. 373.
74. Gajewski, J.: "Effect of the Ingestion of Various Fats On the Fibrinolytic Activity In Normal Subjects and Patients With Coronary Heart Disease," *Journal of Atherosclerosis Research*, Vol 1, 1961, p. 222.
75. Duguid, J.B.: "Pathogenesis of Atherosclerosis," *Lancet*, Vol 2, 1949, p. 925.
76. Astrup, T.: "The Biological Significance of Fibrinolysis," *Lancet*, Vol 2, 1956, p. 565.
77. Miettinen, M.: "Effect of Fasting on Fibrinolysis and Blood Coagulation," *American Journal of Cardiology*, Vol 10, 1962, pp 532-534.
78. Menon, I.S.: "Fasting and Non-fasting Fibrinolytic Activity," *Laboratory Practice*, Vol 16, 1967, pp 469-470.
79. Dock, W.: "Atherosclerosis—Why Do We Pretend the Pathogenesis is Mysterious?", *Circulation*, Vol 50, 1974, pp 647-649.
80. Petersdorf, R.G., *op cit*, pp 1302-1303.
81. Kannel, W.B.: "Prevention of Coronary Heart Disease by Control of Risk Factors," *JAMA*, Vol 227, 1974, p. 338.
82. Walker, W.J.: "Relationship of Adiposity to Serum Cholesterol and Lipoprotein Levels and Their Modification by Dietary Means," *Annals of Internal Medicine, New Series*, Vol 39, 1953, pp 705-716.
83. Walker, W.J., Lawry, E.L., Love, D.E., Mann, G.V., Levine, S.A. and Stare, F.J.: "Effect of Weight Reduction and Caloric Balance on Serum Lipoprotein and Cholesterol Levels," *American Journal of Medicine*, Vol 14, 1953, pp 654-664.
84. Corday, E. and Corday, S.R.: "Prevention of Heart Disease by Control of Risk Factors: The Time Has Come to Face the Facts," *American Journal of Cardiology*, Vol 35, 1975, pp 330-333.
85. Select Committee on Nutrition and Human Needs, US Senate, *Dietary Goals For The US*, US Government Printing Office, Washington, DC, 1977.
86. Goodhart, R.S., and Shils, M.E.: *Modern Nutrition in Health and Disease*, 5th ed, Lea and Febiger, Philadelphia, 1973, p. 901.
87. Goodhart, R.S., *ibid*, p. 911.
88. Zelis, R., Mason, D.T., Braunwald, E. and Levy, R.I.: "Effects of Hyperlipoproteinemias and their Treatment on the Peripheral Circulation," *Journal of Clinical Investigation*, Vol 49, 1970, pp 1007-1015.
89. Starzl, T.E., Chase, H.P., Putnam, C.W. and Porter, K.A.: "Portacaval Shunt in Hyperlipoproteinemia," *Lancet*, Vol 2, 1973, pp 940-944.
90. Knight, L., Scheibel, R., Amplatz, K., Varco, R.L. and Buchwald, H.: "Radiographic Appraisal of the Minnesota Partial Ileal Bypass Study," *Surgical Forum*, Vol 23, 1972, pp 141-142.
91. Higginbotham, A.C., Williams, A.V. and Knisely, M.H.: "Some Acute Effects Of Dietary Fats on Intravascular Agglutination in Cardiac Patients," *Anatomical Record*, Vol 121, 1955, pp 310-311.
92. Williams, A.V., Higginbotham, A.C. and Knisely, M.H.: "Increased Blood Cell Agglutination Following Ingestion of Fat, A Factor Contributing to Cardiac Ischemia, Coronary Insufficiency, and Anginal Pain," *Angiology*, Vol 8, 1957, pp 29-40.
93. Kuo, P.T. and Joyner, C.R.: "Angina Pectoris Induced by Fat Ingestion in Patients with Coronary Artery Disease," *JAMA*, Vol 158, 1955, pp 1008-1013.
94. Regan, T.J., Binak, K., Gordon, S., DeFazio, V. and Hellems, H.K.: "The Modification of Myocardial Blood Flow and Oxygen Consumption During Postprandial Lipemia and Heparin-Induced Lipolysis," *Journal of Clinical Investigation*, Vol 38, 1959, pp 1033-1034.
95. Swank, R.L.: "Changes in Blood Produced by a Fat Meal and by Intravenous Heparin," *American Journal of Physiology*, Vol 164, 1951, pp 798-811.
96. Fullerton, H.W., Davie, W.J.A. and Anastasopoulos, G.: "Relationship of Alimentary Lipaemia to Blood Coagulability," *British Medical Journal*, Vol 2, 1953, pp 250-253.
97. Cullen, C.F. and Swank, R.L.: "Intravascular Aggregation and Adhesiveness of the Blood Elements Associated with Alimentary Lipemia and Injections of Large Molecular Substances," *Circulation*, Vol 9, 1954, pp 335-346.
98. Swank, R.L. and Nakamura, H.: "Oxygen Availability in Brain Tissues After Lipid Meals," *American Journal of Physiology*, Vol 198, 1960, pp 217-220.
99. Swank, R.L.: "Blood Viscosity in Cerebrovascular Disease," *Neurology*, Vol 9, 1959, pp 553-560.
100. Nakamura, H. and Swank, R.L.: "Electrocardiogram in Hamsters after Large Fat Meals," *Proceedings of Society for Experimental Biology and Medicine*, Vol 105, 1960, pp 195-197.
101. Petersdorf, R.G., *op cit*, p. 1309.
102. Duncan, G.G., Cristofori, F.C., Yue, J.K. and Murthy, M.S.J.: "The Control of Obesity by Intermittent Fasts," *Medical Clinics of North America*, Vol 48, 1964, pp 1359-1372.
103. Krotkiewski, M., Ruzyllo, E. and Kotowska, A.: "Obesity and Arterial Hypertension. II. Effect of a Reducing Diet and Fasting," *Polish Medical Science and History*, Vol 10, 1967, pp 58-62.
104. Boehme, D.H.: "Preplanned Fasting in the Treatment of Mental Disease: Survey of Current Soviet Literature," *Schizophrenia Bulletin*, Vol 3, 1977, pp 288-296.
105. Lennox, W.G. and Cobb, S.: "Studies in Epilepsy. VIII. The Clinical Effect of Fasting," *Archives of Neurology and Psychiatry*, Vol 20, 1928, pp 771-779.
106. Brozek, J.B., Chapman, C.B. and Keys, A.: "Drastic Food Restriction — Effect on Cardiovascular Dynamics in Normotensive and Hypertensive Conditions," *JAMA*, Vol 137, 1948, pp 1569-1574.
107. Lups, S. and Francke, C.: "On the Changes in Blood Pressure During the Period of Starvation (September 1944 to May 1945) and after the Liberation (May 1945 to September 1945)

in Utrecht, Holland," *Acta Med Scand*, Vol 126, 1947, pp 449-458.
108. Hartsilver, J.: "A Note On High Blood Pressure," *British Medical Journal*, Vol 2, 1935, pp 449-450.
109. Kempner, W.: "Treatment of Hypertensive Vascular Disease with Rice Diet," *American Journal of Medicine*, Vol 4, 1948, pp 545-577.
110. Bloom, W.L., Azar, G. and Smith, E.G.: "Changes in Heart Size and Plasma Volume during Fasting," *Metabolism Clinical and Experimental*, Vol 15, 1966, pp 409-413.
111. Haxhe, J.J.: "Experimental Undernutrition. I. Its Effects on Cardiac Output," *Metabolism Clinical and Experimental*, Vol 16, 1967, pp 1086-1091.
112. Robbins, S.L.: *Pathological Basis of Disease*, W.B. Saunders Company, Philadelphia, 1974, p. 1129.
113. Petersdorf, R.G., *op cit*, p. 1313.
114. Lusk, G.: "The Physiological Effect Of Undernutrition," *Physiological Reviews*, Vol 1, 1921, pp 523-552.
115. Reisin, Efrain, Abel, R., Modan, M., Silverberg, D.S., Eliahou, H.E. and Modan, B.: "Effect of Weight Loss Without Salt Restriction on The Reduction of Blood Pressure in Overweight Hypertensive Patients," *New England Journal of Medicine*, Vol 298, 1978, pp 1-6.
116. Petersdorf, R.G., *op cit*, p. 1178.
117. Petersdorf, R.G., *ibid*, p. 1183.
118. Master, A.M., Jaffe, H.L. and Dack, S.: "Undernutrition in the Treatment of Coronary Artery Disease (Particularly Thrombosis). Effect on the Basal Metabolism and Circulation." *Journal of Clinical Investigation*, Vol 15, 1936, pp 353-367.
119. Master, A.M., Jaffe, H.L. and Dack, S.: "Low Basal Metabolic Rates Obtained by Low Calorie Diets in Coronary Artery Disease," *Proceedings Society Experimental Biology and Medicine*, Vol 32, 1934-5, pp 779-783.
120. Young, D.R.: "Effect of Food Deprivation on Treadmill Running in Dogs," *Journal of Applied Physiology*, Vol 14, 1959, pp 1018-1022.
121. Merrill, A.J.: "Intractable Heart Failure-Management With 5 to 7 Days of Fasting. A Preliminary Trail." *American Heart Journal*, Vol 67, 1964, pp 433-436.
122. Stechschulte, D. and Dunn, M.: "Starvation and Heart Failure," *Journal of the Kansas Medical Society*, Vol 66, pp 500-502.
123. Kempner, W.: "Treatment of Cardiac Failure with the Rice Diet," *North Carolina Medical Journal*, Vol 8, 1947, pp 128-131.
124. Master, A.M.: "Coronary Artery Thrombosis," *JAMA*, Vol 105, 1935, pp 337-341.
125. Master, A.M., Jaffe, H.L. and Dack, S.: "Undernutrition in the Treatment of Coronary Artery Disease (Particularly Thrombosis). Effect on The Basal Metabolism and Circulation," *Journal of Clinical Investigation*, Vol 15, 1936, pp 353-367.
126. Grollman, A.: "Physiological Variations in the Cardiac Output of Man," *American Journal of Physiology*, Vol 89, 1929, pp 366-370.
127. Gilbert, N.C., LeRoy, G.V. and Fenn, G.K.: "The Effect of Distension of Abdominal Viscera on the Blood Flow in the Circumflex Branch of the Left Coronary Artery of the Dog," *American Heart Journal*, Vol 20, 1940, pp 519-524.
128. Smith: *Annals of Internal Medicine*, Vol 17, 1942, p. 681; and Boas: *American Heart Journal*, Vol 23, 1942, p. 1.
129. Spencer, I.O.B.: "Death During Therapeutic Starvation for Obesity," *Lancet*, Vol 1, 1968, pp 1288-1290.
130. Garnett, E.S., Barnard, D.L., Ford, J. and Woodehouse, M. A.: "Gross Fragmentation of Cardiac Myofibrils after Therapeutic Starvation for Obesity," *Lancet*, Vol 1, 1969, pp 914-916.
131. Sandhofer, F., Dienstl, F., Bolzano, K. and Schwingshackl, H.: "Severe Cardiovascular Complication Associated With Prolonged Starvation," *British Medical Journal*, Vol 1, 1973, pp 462-463.
132. De Vries, Arnold: *Therapeutic Fasting*, 4th ed, Chandler Book Company, Los Angeles, 1963, pp 5-7.

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