# Diet Therapy of Diabetes: An Analysis of Failure

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**A review of the available evidence shows clearly the rarity with which diabetics understand and follow their diet prescriptions. The reasons for these shortcomings and their persistence are many and complex. They include the tendency of physicians to underestimate the formidability of developing, implementing, and adjusting a diet prescription that is both acceptable and effective over a long period of time. Another problem is the limited conceptual and technical knowledge of most physicians concerning dietary principles, strategies, and tactics as they apply to the various types of diabetes. Recent research confirms the important potentials of diet regulation in mitigating diabetes and its complications. But apparently much of our effort in diet counseling is ineffective and wasteful. It seems desirable, therefore, to review in some detail the reasons for this failure and then to use candid appraisals for developing more effective approaches in the diet therapy of diabetes.**

THE CONCLUSIONS of the University Group Diabetes Program on the oral hypoglycemic agents have been the subject of lively controversy (1-8). Yet the protagonists on both sides, as well as more neutral observers, agree on one point: diet is the fundamental element of therapy in most cases of maturity-onset diabetes (2). Fortunately, several excellent publications (9-11) describing the goals, principles, and methods of diet therapy in diabetes have recently appeared. These principles have also been well summarized recently in a succinct statement by an expert committee of the American Diabetes Association (12). The new guidelines have helped to clarify these dietary principles, but the poor results of previous years are not primarily attributable to the absence of authoritative guidance. As early as 1958, for example, Daughaday (13), writing on behalf of the AMA Nutrition Council, had described principles that are surprisingly similar to those being advocated currently by experts in the field. Kaufman (14) published an excellent essay in 1964 on diet counseling. Stone (15) had shown, in 1961, an impressive relation between the effectiveness of dietary therapy and the control of diabetes; he also showed that effective dietary therapy was quite feasible. Wood and Bierman (11) have recently published an excellent account of the historical development of our concepts of the dietary management of diabetes.

Most clinicians and dietitians recognize that the practical effectiveness of diet therapy leaves something to be desired, but few fully appreciate how poor the record really is. One of my purposes is to summarize evidence that documents the ineffectiveness of our previous efforts, with the hope that better recognition of the extent and character of these shortcomings will engender sufficient humility to prompt corrective measures. The dimensions of this failure and particularly *its persistence* justify a more thorough study of its causes. If better insights can be gained about the roots of failure, we can then apply more readily and effectively the principles that have been recently promulgated (12).

### To What Degree Do Patients Follow Their Diet Prescriptions?

In 1961 Stone (15) studied the reasons for poor control in a group of 126 patients; he concluded that ineffective dietary therapy was responsible in most. He also reviewed the literature relating to studies of adherence to diabetic diets. It was apparent that,

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before 1961, successful application of diet therapy was uncommon. Unfortunately, there is no evidence of recent improvement. Perhaps the most impressive and discouraging data are those obtained in the National Health Survey (16). It was found that 22% of diabetics "stated that they were never given a diet for their diabetes." Another 25% "had received a diet but said they didn't follow it." Only 53% of this representative group of American diabetics "indicated that they followed a prescribed diet." A simple test of knowledge of some fundamentals of diet therapy showed that, even in the group who said they followed a prescribed diet, a substantial majority had a poor grasp of the fundamentals of the exchange method. Understanding of, and adherence to, diet were poor in each of three subgroups who were being treated with diet alone, insulin, or oral agents, respectively. Williams and associates (17) evaluated the knowledge and practices of another group of diabetics by using more detailed methods, with equally discouraging results: only a very small minority followed their prescriptions accurately.

Knowles and colleagues (18) summarized studies by seven different groups of investigators who had evaluated adherence by juvenile diabetics to prescribed diet. These reports described "failure to follow prescriptions in 22% to 89% of cases." In one of the more recent British studies (19) only 19 of 63 diabetics consumed within 10% of the prescribed amount of food (calories), when their actual intakes were studied for a week. It seems likely that dietary adherence of this select and cooperative subgroup was better than that of 44 other individuals from the same group, who had chosen not to participate fully in the study. Moreover, the adherence to diet of the cooperative subgroup was probably better during the period of observation than at other times. This problem seems to be an international one. In India, for instance, adherence to diet prescriptions was poor in a group of diabetics studied by Devadas, Nirmala, and Bai (20).

A final discouraging example is the result of the studies of the University Group Diabetes Program (21). A large group of maturity-onset diabetics were given standard diet prescriptions under conditions that were, in at least some respects, highly favorable (for example, specialty clinics, highly competent physicians and dietitians, and regular clinic visits). This group's most important dietary objective was reduction of caloric consumption. The mean body weight of these subjects was 133% of the standard weight. The initial mean weights were the same in each of four subgroups who were treated with the various therapeutic agents, and diet prescriptions were similar in each subgroup. During the first 6 months of the study, very little weight reduction was achived. Mean weight loss in the four subgroups ranged from 1% to 3% of the initial values. Even this very small success was short-lived. Within 9 months the mean weight of the subgroup prescribed a diet and tolbutamide had returned to the base-line value. After 4.5 years the mean weights had *increased* an average of about 1% in three of the four subgroups. The results in a subgroup prescribed "diet" and placebo were not quite as bad, but even in this subgroup mean weight after 4.5 years was only 2% below the base-line value. Mean weights for the whole group were *higher*, not lower, after 4.5 years of "diet." It is of interest that the patients on "diet" alone had no long-term improvement of blood glucose levels. This could mean that sugar intake was not reduced, but I would guess that they did eat less sugar. If so, this suggests that reducing sugar intake provides little if any long-term effect on glycemia when sugar is replaced with other calories. It is important to note that diet therapy was quite successful initially. During the first few days and weeks significant weight reduction was achieved in all subgroups, but, as the months passed, adiposity returned.

### Some Causes of Failure

Table 1 lists some of the major deterrents to successful diet therapy. There is no new information here; the purpose of the table is simply to dramatize the fact that, *in the aggregate*, these problems constitute an extremely formidable barrier. One of the principal reasons for the ineffectiveness of our system of diet therapy is our tendency to underestimate the magnitude and number of the obstacles to be overcome in achieving results. Only a few of the many deterrents listed in Table 1 will be discussed here.

#### MITIGATING OBESITY IS VERY IMPORTANT BUT VERY DIFFICULT

In the more affluent societies most diabetics are fat people with some endogenous insulin who develop manifestations of the disorder after 40 years of age. Recent research results have confirmed and emphasized the profoundly advantageous effect of weight reduction on beta cell function and sensitivity to insulin in these fat diabetics (22-24). The favorable clinical effects of dietary restrictions for these diabetics has long been known, but the more recent studies have been helpful in several respects. They have explained in part the mechanisms by which weight reduction is effective. The newer studies have also shown the *degree* of these markedly deleterious effects of adiposity, on both diabetics and potential



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**Table 1. Some Deterrents to Successful Diet Therapy: A Formidable Challenge**

1. The diet prescription recommended may lack relevance to patient's preferences and propensities. It may not fit his cultural, sociologic, or economic status. The proposed diet may differ from that of the patient's family or friends. The patient may find it difficult to give up calories or specifically proscribed foods (for example, concentrated carbohydrates, alcohol, and animal fat). He may not like low-caloric, low-carbohydrate foods in his prescription, such as A and B vegetables. He may lack sufficient intelligence, education, incentive, or self-discipline.
2. An insulin-dependent patient may find frequent feedings are not convenient or appealing. He may not like to eat the same number of total calories every day. He may find it difficult to follow the fixed time schedule for meals called for in his own prescription, or in any prescription, because of his other commitments, or vagaries of preference or of appetite. He may not like to eat the same ratios and amounts of carbohydrates, proteins, and fats in each meal every day. He may not find it convenient to balance periods of unusual exercise with additional carbohydrate in appropriate amounts.
3. The patient and his family may misunderstand the general goals and priorities of the diet, or they may not understand either the specific dietary strategies (for example, the principles of the food exchange system) or the specific dietary techniques and alternatives (for example, how much potato is equivalent to one piece of bread).
4. The physician, dietitian, or nurse may have a poor understanding of the principles, strategies, priorities, or methods of diet therapy.
5. The patient education system may be defective because of [1] limitations of acumen, enthusiasm, and time of health professionals; [2] limited teaching manpower; [3] lack of economic incentives for health professionals and institutions to teach patients; [4] patient education efforts that are seldom sufficiently systematic (for example, responsibilities of physician, dietitian, nurse, and patient may not be clearly and systematically delineated); or [5] often underestimated general and specific deficiencies in the patients' understanding.

diabetics (25). Thus, in some ways, the problem of achieving weight reduction is the most important challenge in the therapy of diabetes. Unfortunately, fat people rarely follow their diet prescriptions. Most observers estimate that significant *long-range* weight reduction has been achieved by less than 15% of those who are advised by physicians to lose weight (26). Others have discussed the problems and strategies in the management of obesity (26-29). Under ordinary circumstances results are poor, but under certain conditions impressive rates of success can be achieved (28-30). Apparently much of our present effort in the diet counseling of obese patients is ineffective and wasteful. An institution that is not prepared to conduct weight reduction programs with unusual vigor and skill might best be advised not to waste money on token efforts of the usual kind. A major difficulty is that many physicians, dietitians, and patients have only modest incentive, because they do not yet appreciate sufficiently the profoundly beneficial effects of weight reduction in fat, maturity-onset diabetics.

## Confusion about Dietary Goals, Strategies, and Priorities

### MISUNDERSTANDINGS AND UNCERTAINTIES ABOUT THE EFFECTS OF CARBOHYDRATES

In 1949, as a measure of desperation, I prescribed a rice diet for a severe diabetic with malignant hypertension. Although dietary carbohydrate was increased approximately threefold, there was no increase in insulin requirement. Like many other physicians who had this experience, I was reluctant either to accept fully the implications or to follow up the findings adequately. In 1960 I did, however, study a severely diabetic patient who was consuming a standard diabetic diet. After a suitable control period on a metabolic ward, daily carbohydrate consumption was doubled (from 180 to 360 g), under *isocaloric* conditions. It may be of some importance that the carbohydrate was divided into five feedings. There was no change in insulin requirement and no evident change in levels of blood and urine glucose. In the process of preparing a publication of this "discovery," I was surprised to find that very similar experiments had been done before 1935 by Himsworth, with the same results (31-33). Over and over again, this phenomenon had been rediscovered (34-47)—and subsequently forgotten or disregarded. Recent studies by Brunzell and associates (48) are in some respects the most impressive to date for showing the excellent tolerance of diabetics for dietary carbohydrate under certain conditions. As a matter of fact, the results of Himsworth and the more recent work in Seattle (48, 49) suggest a positive advantage for high-carbohydrate diets because, at least under some conditions, they lead to improved carbohydrate tolerance in diabetics.

Such evidence and the potential deleterious effects of saturated fat led the expert committee on nutrition to recommend, in 1971, more generous amounts of carbohydrates than have been customarily prescribed by clinicians in Western countries (12). If Western clinicians had been less parochial, we would have appreciated sooner the surprising tolerance of diabetics for dietary carbohydrate *when caloric intake is controlled.* Clinicians in certain Eastern and tropical countries have long known that both maturity-onset and juvenile-onset diabetes may be quite satisfactorily controlled on diets containing almost twice as much carbohydrate as is in standard diabetic diets of the West (50-52). For example, the diets of the diabetics in one Japanese clinic contain mainly carbohydrate (64% of calories) (53). Clinicians in India have often prescribed diets, with apparent success, that contain more than 70% of



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the calories as carbohydrate (51, 52). The American diabetic patients of Stone and Conner (47) also did well on high-carbohydrate diets containing 65% of calories as carbohydrate. These diets did not produce any elevation of serum triglyceride levels. Yet most Western clinicians, dietitians, and their patients still believe that limiting dietary carbohydrates is the *main* purpose of diet therapy. They have often observed dramatic improvement with carbohydrate restriction, without considering the possibility that improvement may have been partly or wholly attributable to the incidental reduction of caloric intake.

Asians, who consume high-carbohydrate, low-calorie diets, have very low rates of diabetes (25, 50, 54). A typical Asian meal contains 130 to 250 g of carbohydrate. This epidemiologic evidence is compatible with the idea that adiposity is a much greater insult to beta cell integrity than a high-carbohydrate diet is (25). But it should be kept in mind that, although the Asian diet is high in carbohydrate (starch), it is low in sugars. Recent studies (54-57) showing that both proteins and fats, as well as carbohydrates, stimulate insulin release may have clinical implications.

I hope that the evidence cited above and the authoritative endorsement of diets containing more liberal amounts of carbohydrate (12) will lead physicians to reconsider their dietary strategies. But history has shown that it is extremely difficult for physicians, dietitians, and patients to give up the notion that carbohydrates are bad for people with too much sugar in their blood. To most it just does not seem logical that carbohydrate would be so well tolerated. What is the root of this prejudice? One factor is a failure to appreciate the efficiency with which and the extent to which diabetics convert non-carbohydrate fuels to glucose (particularly when there is an oversupply of fuel). With a carbohydrate load the intolerance is more immediately evident (particularly when sugar is consumed), but in the long run the total fuel supply is usually more important in determining blood glucose levels. Soskin and Levine (58) many years ago demonstrated the importance of the overproduction of glucose by the diabetic liver, but too few of today's clinicians appreciate the practical significance of this general concept.

## THE REGULATION, TIMING, AND CHARACTERISTICS OF DIETARY CARBOHYDRATES

Findings confirming the tolerance of diabetics for carbohydrates might appear to make diet therapy of diabetes not only more attractive but also unnecessary; this is not so. Special diets are still important in the therapy of both the typical types of diabetes: the obese who do not require insulin and the insulin-dependent diabetics. *High-carbohydrate diets are poorly tolerated by both obese and lean patients if caloric intake is not regulated appropriately.* The major problem here is the frequent failure to understand that the need for *regulation* of carbohydrate intake does not mean that dietary carbohydrate should be reduced to the lowest possible level. The purpose of "regulation" is to assure appropriate consistency in the timing, division, amounts, and characteristics (sugar or starch) of the carbohydrates consumed and to assure that the total amounts do not increase caloric consumption above the optimal level. In insulin-dependent diabetics, for example, this regulation of carbohydrate consumption is usually quite important (*see* Table 2), even though daily carbohydrate intake may be relatively generous. There is also a tendency to assume that the need to limit the amount of carbohydrate in any single feeding reflects a priority for carbohydrate restriction in the total prescription. If, for example, a patient is told to limit carbohydrate consumption to less than 70 g in any one feeding, he may assume that if some restriction is good, more restriction may be better.

Most would agree that well-spaced feedings of modest size are preferable to large feedings and that simple, concentrated carbohydrates such as sucrose should usually be limited or avoided, particularly in the main meals. Under certain conditions simple sugars have been poorly tolerated (59, 60); under other conditions they have been tolerated very well (46, 48, 49, 61, 62). Although the clinical effects in diabetes of typical Asian diets high in carbohydrates (mainly rice) have been fairly well tested, the clinical feasibility and usefulness of well-regulated diets containing generous amounts of simple sugars have not been evaluated very well. There are several theoretical disadvantages of high-sugar diets, as contrasted with high-starch diets, including the possibility that sugar intake may favor triglyceridemia (9). Under certain conditions, however, even patients with type IV hyperlipidemia tolerate high-carbohydrate diets better than high-fat diets (63). Because foods containing sugar tend to be rich in calories, it is often difficult to limit caloric consumption in obese patients without limiting sugar intake.

Although the trend in Western countries is clearly toward increasing total carbohydrate in the diabetic diet, some recent evidence suggests that all the benefits of carbohydrate restriction in obese diabetics may not be attributable to the incidental reduction



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**Table 2. Dietary Strategies for the Two Main Types of Diabetes**

| Dietary Strategy | Obese Patients Who Do Not Require Insulin | Insulin-Dependent Nonobese Patients |
|---|---|---|
| Decrease calories | Yes | No |
| Protect or improve beta cell function | Very urgent priority | Seldom important because beta cells are usually extinct |
| Increase frequency and number of feedings | Usually no* | Yes |
| Day-to-day consistency of intake of calories, carbohydrate, protein, and fat | Not crucial if average caloric intake remains in low range | Very important |
| Day-to-day consistency of the ratios of carbohydrate, protein, and fat for each of the feedings† | Not crucial | Desirable |
| Consistency of timing of meals | Not crucial | Very important |
| Extra food for unusual exercise | Not usually appropriate | Usually appropriate |
| Use of food to treat, abort, or prevent hypoglycemia | Not necessary | Important |

* There are some theoretical advantages in dividing the diet into four or five feedings even in mild diabetes *if* this can be done without increasing caloric consumption. However, because limitation of calories has highest priority in obese diabetics, there are some potential disadvantages in providing extra feedings. Giving fat people an opportunity to eat at bedtime is particularly "risky" if weight reduction is the prime goal.

† The total daily insulin requirement is apparently not much affected when dietary constituents are changed *under isocaloric conditions*. But insulin requirement *immediately* after a high-carbohydrate meal is higher than immediately after a low-carbohydrate meal, even if the meal is isocaloric.

in calories (64, 65). Some uncertainties about the degree to which it is necessary to control the amounts, characteristics, and timing of dietary carbohydrates remain.

### FAILURE TO RELATE DIETARY OBJECTIVES TO TYPE OF DIABETES

A major deterrent to the application of appropriate diet therapy has been the limited understanding by patients, dietitians, and physicians of the special goals, strategies, and priorities of diet therapy in the different kinds of diabetes. It is not sufficiently appreciated that the objectives of the diet in the two typical types of diabetes are quite different in several respects. Table 2 summarizes and contrasts the objectives and strategies for these two main types. The differences are so marked that one may question the propriety of teaching sessions on diet that involve both groups. Although certain methods, such as the food exchange system, have potential applicability for both types, the differences in dietary objectives are so great that attempts to generalize about diabetic diets are apt to be misleading to both groups.

It should be noted that Table 2 and other parts of this discussion are relevant only to the two most typical types of diabetes. Certain other special types may require different approaches. This includes lean persons with mild diabetes who do not require insulin, insulin-dependent diabetics who are fat, and those hyperlipemic diabetics whose blood lipid levels are especially sensitive to dietary carbohydrate.

### UNCERTAINTIES ABOUT THE INFLUENCE OF DIET ON VASCULAR MANIFESTATIONS

The main challenge in the management of diabetes today is the prevention and mitigation of the vascular manifestations. Clinicians should consider the probable effects of dietary alternatives on each of these major lesions: glomerulosclerosis, retinopathy, coronary atherosclerosis, atherosclerosis of the lower extremity vessels, and cerebrovascular disease. In the past there has been an unfortunate propensity to lump "the vascular lesions of diabetes" into a single group as if they had a common etiology and pathophysiology. But there is increasing laboratory and epidemiologic evidence of differences among these lesions. For example, diabetics in Japan apparently develop retinopathy at roughly the same rate as diabetics of North America and Europe, but these Japanese diabetics have more cerebrovascular disease, far less coronary disease, and even less clinically significant atherosclerosis of the lower-extremity vessels (54). It is, therefore, quite possible that the ideal diet for mitigating or preventing one of these vascular lesions may not be optimal for another.

One of the clinician's problems is that he does not have evidence to judge with certainty the effects of various dietary alternatives on either the small- or large-vessel lesions of diabetes. Nevertheless, considerable evidence helpful in estimating probable effects of various nutrients on atherosclerosis is available. A full and fair review of this evidence is beyond the scope of this discussion, but a strong



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case has been made for limiting cholesterol and saturated fat in most diabetics to levels below the levels commonly consumed in the United States (10, 66). It is clear, however, that the dietary strategy for controlling blood lipid levels of diabetics must be individualized to some extent, depending on the serum lipid patterns, the degree of adiposity, and other considerations (9). In diabetics who live in societies where saturated fat intake is high, many experts are encouraging selectively, or even routinely, both the limitation of total fat and the substitution of polyunsaturated fat for at least some saturated fat (10, 12). Expert opinions vary about the *degree* to which it is desirable and feasible to lower saturated fat intake and to raise consumption of polyunsaturates. Issues include the theoretical possibility of toxicity of large amounts of polyunsaturates if administered for many years and the degree to which these polyunsaturates have a specific and direct effect on the inhibition of atherosclerosis. Kinsell (67) has briefly summarized the case for using polyunsaturates in the diabetic diet and has described specific simple methods for doing this. The standard diets sponsored by the American Diabetes Association, the American Dietetic Association, and the U.S. Public Health Service are still quite high in fat (about 42% of calories), but these organizations have recently made alternative fat-controlled diets available. Three recent publications have summarized for the clinician specific principles and strategies for controlling diabetes and its atherosclerotic complications (9, 10, 12).

It is useful to remember that the large vessels of the diabetics who have lived in disadvantaged countries, and who have received little or no medical attention, usually look much better than those of our patients, who have had the "advantage" of consuming the traditional diabetic diets of North America and Europe (54, 68). Most of these underprivileged societies consume a diet high in carbohydrate and low in saturated fat, which leads to the conclusion that saturated fat is the main culprit. But sugar intake is also low in most of the poor countries. This and other evidence has led some to believe that sugar is an important factor in atherogenesis (60, 69). Other considerations about dietary sugar have been discussed above.

The effects of diet on retinopathy and glomerulosclerosis are not well understood. Indeed, there is little evidence that the constituents of the diet are important in determining the rate at which these lesions develop. Sussman (70) has recently summarized the evidence for and against the hypothesis that "control" of diabetes has a mitigating effect on these lesions. There are two separate issues here that are often considered together, when they should be considered separately. The first is what dietary approaches are most effective in controlling blood glucose values. The second issue is the relation, if any, between the degree of hyperglycemia and the rates at which the lesions develop. The second issue is beyond the scope of this discussion, but it has been well reviewed recently by Fajans (71) and by Sussman (70). As pointed out above, dietary approaches to the control of blood glucose vary with the type of diabetes (*see* Table 2).

### PROBLEMS OF SEMANTICS

Another problem has been the propensity of many clinicians to misinterpret, misunderstand, or to use without adequately defining terms such as "free diet," "strict diet," and "diabetic diet." Another difficulty is the common failure to distinguish between the diet prescription and the diet (what the patient actually eats). Many patients who are said to be "on a diet" are not really following the prescription to any significant degree. Some patients said to be on "free diets" are following regimens that more adequately meet appropriate dietary objectives than other patients who "watch their diets very carefully." Patients who say they are on "free diets" often omit or limit table sugar, and they may eat less of concentrated carbohydrate such as syrup, pie, and cake. Terms such as "rigid diet" are often misunderstood or used inappropriately, particularly when applied to the dietary strategy for insulin-dependent patients. In the sense that a certain basic regularity is desirable, the term "rigid" does have some application. Yet the need to make frequent and appropriate exchanges among foods and the need for extra food to balance the effects of vigorous exercise suggest that in some respects "flexibility" may be more descriptive of what is required than "rigidity." As applied to the lean, insulin-dependent diabetics, expressions such as "careful diet," "watch your diet," and "strict diet" often suggest that the erroneous concept, deprivation —the more the better—is a prime objective.

### INAPPROPRIATE OR INDEFINITE DIET PRESCRIPTIONS

Often the irregularity of dietary intake in insulin-dependent diabetics is attributable to a diet prescription that is inappropriately restrictive in calories. Insulin-dependent nonobese men, for example, who typically require 2500 to 3200 kilocalories per day are often prescribed diets containing about 1800 kilocalories daily. This kind of error is all too often made with children. There are several reasons for the frequency of this error. The doctor, dietitian,



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and the patient may misunderstand the purpose of a diet prescription for lean diabetics. Most physicians do not know much about caloric requirements and the degrees to which these are affected by factors such as body size and exercise. Many patients are left on a prescription such as 1800 kilocalories because, while inactive in the hospital, they may do well on this and complain little. Most diabetics are fairly well satisfied *initially* with prescriptions containing 70% to 80% of their caloric requirements. The total bulk of food and the size of the main meals may be as large as those to which they were accustomed. This is because the ratio of bulk to calories is a little higher in most diabetic diets and because extra calories were previously available in snacks (beer, liquor, cookies, candy, soft drinks) and in occasional feasts at meal time. The patient who needs 2800 calories may follow his 1800 calorie diet faithfully, while just adding a bit here and there to achieve satiety; or he may follow his diet carefully, every other week. The disadvantage of this in lean patients who take insulin is that the food intake tends to be less constant than with a prescription that keeps them "filled up." As shown in Table 2, the situation is quite different for obese patients with typical maturity-onset diabetes.

Even the knowledge of dietitians about diabetic diets leaves much to be desired (72). The shortcomings of the nutrition-related teaching programs of medical schools have been documented and roundly criticized (73, 74).

Another problem is the failure to tailor the diets to fit the propensities and life style of the patient (75). The patient may be given a midafternoon feeding, which is taken only irregularly because the food prescribed is difficult to obtain at that hour in his job situation or because he does not like it. Most physicians do not know enough about nutrition to construct a diet that will be attractive and feasible as well as effective. On the other hand, dietitians often do not have sufficient authorization from the physician to formulate the appropriate "individualized" prescription. Also, the dietitian who thoroughly understands the fundamental tactics (for example, the food exchange system) may not grasp the strategic priorities applicable to the particular patient. And the physician who does not know how much carbohydrate, protein, and fat is in common foods such as milk is at a disadvantage in the dietary management of diabetes, even if he understands the general principles.

It is, of course, possible for the physician to delegate authority to the dietitian to effect appropriate adjustments in the diet prescription dictated by factors such as changes in the patient's job, domestic situation, exercise, and food preferences. He may even find it feasible and appropriate to authorize the dietitian to formulate the initial dietary prescription after he has suggested general guidelines. Dietitians often *have* to do this when faced with an enigmatic order on the chart, such as "diabetic diet." Unfortunately, there are seldom systematically determined, clearly understood, and specific agreements between physicians and their dietitians about who is responsible for what in designing, implementing, and modifying the diet prescription. The dietitians "play it by ear" as best they can. As a result of this unsystematic approach, the specifics of dietary goals, strategies, and methods are not often clearly formulated (76). In an attempt to remedy this difficulty, I have developed a check list (Table 3) that provides an opportunity for the physician to consider systematically, with the dietitian, the needs of each patient and to determine who (doctor, dietitian, or patient) should do what. This check list has teaching value; it may prompt the physician (or medical student) and the dietitian (or dietetic intern) to appreciate the many factors that ought to be considered in formulating and implementing the diet prescription of a specific patient.

In such a consultation the necessity for considering every question on the list could be obviated by prior agreement on standard operating procedures that might be applied in common situations. For example, it might be decided that patients who take insulin will be given bedtime feedings routinely, or that all diabetic patients will be restricted to a certain level of saturated fat unless a specific order to the contrary is received.

Another frequent problem in implementing dietary prescriptions is that they are often either insufficiently detailed or unnecessarily complex. Attempts are frequently made to teach patients the details of the exchange system who do not need to know it (for example, those with very mild diabetes and most elderly patients). On the other hand, other patients with severe and labile diabetes, who would benefit from a detailed knowledge, do not receive the kind of instruction that would permit a maximum variety of dietary alternatives within their fixed prescription of carbohydrates, proteins, and fats.

### DISAGREEMENTS ABOUT THE IMPORTANCE OF DIETARY THERAPY

Disagreement among the experts over certain aspects of diet therapy fosters some uncertainty and confusion in patients, dietitians, and physicians. Some of the disagreements are real; some of the differences are mainly semantic. The excellent sum-



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Table 3. A Check List for Dietitians and Physicians: Formulating and Implementing a Diet Prescription for a Specific Patient

1. *In order of priority* what are the main general purposes (not strategies or methods) of this patient's prescription?
2. How much should the patient weigh? How much does the doctor, the dietitian, and the patient think he should weigh? How much would the patient *like* to weigh?
3. What is the appropriate level of caloric consumption for this patient?
4. Does the patient require insulin? If so, is the blood glucose relatively stable, moderately labile, or severely labile? What kind of insulin is to be given, at what time, in what amounts?
5. What and when and how much would the patient *like* to eat if he did not have diabetes? Are there any special considerations relating to economic factors or to family or cultural dietary propensities?
6. Is the level of carbohydrate to be limited? To what level or range? To what extent and under what conditions, if any, are concentrated carbohydrates to be used?
7. Any special requirements concerning levels of protein?
8. Are there any specific or general requirements with respect to levels of dietary fat, either saturated or unsaturated?
9. How much alcohol is to be permitted? Under what conditions? Should alcohol be exchanged for another food? If so, what kind and in what amounts?
10. If time distribution of food is of any importance, are there specific requirements concerning [1] the relative size and timing of each of the three main meals? [2] the timing, size, and characteristics of any extra feedings?
11. To what degree is day-to-day consistency required in [1] total calories? [2] size and characteristics of specific feedings such as lunch?
12. Are dietary adjustments to be made for exercise or marked glycosuria? Of what nature?
13. Are there any special conditions unrelated to diabetes requiring special diet (for example, gout, hyperlipidemia, and renal or cardiac failure)?
14. Can all elements of the prescription be reconciled, and how should this be done? (For example, it is usually not feasible to construct a palatable diet for a lean diabetic if the prescription calls for a diet that restricts both carbohydrate and fat.)
15. What kinds and what degree of changes are to be made subsequently by the dietitian without consulting the physician?
16. What should this patient do if he finds it necessary to postpone or modify a meal (for example, attending a dinner meeting or social affair)? How should he adjust diet if his appetite fails (for example, during illness)?
17. Tactical questions: [1] How much precision is required in the various elements of this prescription? [2] What foods can be freely allowed? [3] What foods, if any, are to be weighed or measured? [4] Are any modifications of the standard exchange system appropriate, such as simplification? [5] In general, is food to be unmeasured, estimated, measured, or weighed? [6] Is it necessary or desirable to teach this patient the carbohydrate, protein, and fat content of the common foods? [7] Under what circumstances are artificial sweeteners and diet drinks to be used?
18. Has this patient's understanding of dietary principles and methods been systematically evaluated?

maries on diet therapy of diabetes in the two most widely read medical textbooks advocate therapeutic approaches that are quite similar (77, 78). Yet one begins with the sentence, "Dietary treatment of diabetes still constitutes the basis for management" (77); the other essay begins, "The role of diet in treating diabetes has been greatly exaggerated" (78).

Some physicians are led to minimize the importance of diet therapy in insulin-dependent diabetes by several different motives. They may believe that regularity of consumption is most important, and therefore restrictive measures are more likely to do harm than good. They may believe that the traditional diabetic diets enhance atherogenesis. They may believe in the theoretical benefits of diet prescriptions but assume that the prescriptions will be followed only negligibly. Others may acknowledge the usefulness of diet therapy in controlling blood glucose levels but doubt the importance of such control. I suspect that a major reason for the de-emphasis of diet therapy is the insecurity of physicians with a method of therapy concerning which they know little (73, 76). The need here is for devising and continuously modifying a prescription that best fits both the propensities of the patient and the therapeutic goals. Few physicians are highly proficient in this, even though the skills required are not very complex.

### Other Deterrents

Some sociologic and economic factors are important deterrents to the effective implementation of diet therapy. In the United States most costs are now paid by third parties such as Blue Cross, Blue Shield, and Medicare. Only rarely do such policies cover directly the full costs of an optimal program of dietary instruction for outpatients. This situation provides positive incentives for unnecessary hospitalization and negative incentives for patient education. Even when therapy takes place in a hospital it is seldom possible to recover from third parties the full costs of a good counseling program, which involves appropriate investments of time from the dietitian and the physician. In small hospitals these services are often not available. The teaching hospital clinic designed by Krall and Joslin (79) and the community program model of Etzwiler (72) show potential ways to mitigate these problems, by better and more efficient economic and organizational arrangements.

### Concluding Remarks

The limitations of this review are acknowledged. Its purpose is to deal with certain practical clinical considerations. Many relevant and important scientific issues are treated superficially or ignored, includ-



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ing the possible relation between nutrition and neuropathy and the question of the prevalence, character, and management of the hyperlipoproteinemias. My objectives have required that the review's general tone be critical and, perhaps by implication, even somewhat negative and pessimistic. I therefore conclude by emphasizing two points. I know that the deficiencies to which I refer do not occur universally. There are many physicians and dietitians who are highly skilled in both the formulation and execution of diet therapy for their diabetic patients. There are also many patients who understand and follow their prescribed diets in detail. My purposes are only to show that these latter circumstances are still decidedly exceptional and to analyze the underlying reasons for these shortcomings. The second point is that, although uncertainties about some aspects of dietary therapy remain, we now have a great deal of evidence that shows the very considerable potentials of diet in treating diabetes.

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## References


1. UNIVERSITY GROUP DIABETES STUDY PROGRAM: A study of the effects of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. *Diabetes* 19:747-815, 1970
2. RICKETTS HT: Editorial statement on University Group Diabetes Program results. *Diabetes* 19(suppl 2):iii-v, 1970
3. AMA COUNCIL ON DRUGS: Statement regarding the University Group Diabetes Program study. *Ibid.*, pp. vi-vii
4. EDWARDS CC: Oral hypoglycemic agents: report of the food and drug administration. *Ibid.*, pp. viii-ix
5. SELTZER HS: Avoiding the pitfalls of long-term therapeutic trials: lessons learned from the UGDP study. *J Clin Pharmacol* 12:393-398, 1972
6. PAASIKIVI J, WAHLBERG F: Preventive tolbutamide treatment and arterial disease in mild hyperglycaemia. *Diabetologia* 7: 323-327, 1971
7. KEEN H: Minimal diabetes and arterial disease: prevalence and the effect of treatment, in *Early Diabetes*. New York, Academic Press, 1970, p. 437
8. LEIBEL B: An analysis of the University Group Diabetes Study Program: data results and conclusions. *Can Med Assoc J* 105:292-294, 1971
9. ALBRINK MJ, DAVIDSON PC: Dietary therapy and prophylaxis of vascular disease in diabetics. *Med Clin North Am* 55:877-887, 1971
10. CHRISTAKIS G, MIRIDJANIAN A: The nutritional aspects of diabetes, in *Diabetes Mellitus: Theory and Practice*, edited by ELLENBERG M, RIFKIN H. New York, McGraw-Hill Book Co., Inc., 1970, p. 594
11. WOOD FC JR, BIERMAN EL: New concepts in diabetic dietics. *Nutr Today* 7:4-10, 1972
12. BIERMAN EL, ALBRINK MJ, ARKY RA, et al: Special report: principles of nutrition and dietary recommendations for patients with diabetes mellitus: 1971. *Diabetes* 20:633-634, 1971
13. DAUGHADAY WH: Dietary treatment of adults with diabetes mellitus. *JAMA* 167:859-862, 1955
14. KAUFMAN M: The many dimensions of diet counseling for diabetes. *Am J Clin Nutr* 15:45-49, 1964
15. STONE DB: A study of the incidence and causes of poor control in patients with diabetes mellitus. *Am J Med Sci* 241:64-70, 1961
16. HOLLAND WM: The diabetes supplement of the National Health Survey. *J Am Diet Assoc* 52:387-390, 1968
17. WILLIAMS TF, ANDERSON E, WATKINS JD, et al: Dietary errors made at home by patients with diabetes. *J Am Diet Assoc* 51:19-25, 1967
18. KNOWLES HC JR, GUEST GM, LAMPE J, et al: The course of juvenile diabetes treated with unmeasured diet. Clinical monograph. *Diabetes* 14:239-273, 1967
19. TUNBRIDGE R: Reliability and cost of diabetic diets. *Br Med J* 2:78-80, 1970
20. DEVADAS RP, NIRMALA PS, BAI RG: A study of the dietary habits of diabetic patients in Coimbatore, in *Diabetes in the Tropics*, edited by PATEL JC, TALWALKAR NG. Bombay, Diabetic Association of India, 1966, p. 542
21. GOLDNER MG, KNATTERUD GL, PROUT TE: Effects of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. *JAMA* 218:1400-1410, 1971
22. KARAM JH, GRODSKY GM, FORSHAM PH: Excessive insulin response to glucose in obese subjects as measured by immunochemical assay. *Diabetes* 12:197-204, 1963
23. CAHILL GF JR: Obesity and insulin levels. *N Engl J Med* 284:1268-1269, 1971
24. BORTZ WM: Metabolic consequences of obesity. *Ann Intern Med* 71:833-843, 1969
25. WEST KM, KALBFLEISCH JM: Influence of nutritional factors on prevalence of diabetes. *Diabetes* 20:99-108, 1971
26. ALBRINK MJ: Human obesity, in *Duncan's Diseases of Metabolism*, edited by BONDY PK. Philadelphia, W. B. Saunders Co., 1969, p. 1274
27. BRAY GA, DAVIDSON MB, DRENICK EJ: Obesity: a serious symptom. *Ann Intern Med* 77:779-805, 1972
28. SOLOMON N: The study and treatment of the obese patient. *Hosp Pract* 4:90-94, 1969
29. NIDETCH J: *The Story of Weight Watchers*. New York, a Signet book published by The New American Library, Inc., 1972
30. LOVELL H: Weight reduction neighborhood health program. *JAMA* 218:1049, 1971
31. HIMSWORTH HP: Action of insulin. *Clin Sci* 1:1-38, 1933
32. HIMSWORTH HP: High carbohydrate diets and insulin efficiency. *Br Med J* 2:57-60, 1934
33. HIMSWORTH HP: The dietetic factor determining the glucose tolerance and sensitivity to insulin of healthy men. *Clin Sci* 2:67-94, 1935
34. SANSUM WD, BLATHERWICK NR, BOWDEN R: The use of high carbohydrate diets in the treatment of diabetes mellitus. *JAMA* 86:178-181, 1926
35. GIBSON RB: Latent tolerance in diabetes mellitus: a study of the effect of high sugar diets with insulin on controlled diabetes. *J Lab Clin Med* 14:597-598, 1929
36. RICHARDSON R: High carbohydrate diets in diabetes mellitus. *Am J Med Sci* 177:426-430, 1929
37. STROUSE S, SOSKIN S: Treatment of same diabetic patient with widely varying diets. *Trans Assoc Am Physicians* 47: 317-322, 1932
38. ELLIS A: Increased carbohydrate tolerance in diabetics following hourly administration of glucose and insulin over long periods. *Q J Med* 3:137-153, 1934
39. JACKSON RL, KENEFICK J: Dietary ratios for child with diabetes mellitus. *Am J Dis Child* 64:807-814, 1942
40. GEYELIN HR: The treatment of diabetes with insulin (after ten years) contrasting the effects of normal and of the older diets. *JAMA* 104:1203-1208, 1935
41. CAIRD WI: The effect of different diets on the insulin sensitivity of diabetics. *Clin Sci* 3:105-117, 1937
42. RABINOWITCH IM: Effects of a high carbohydrate-low calorie diet upon carbohydrate tolerance in diabetes mellitus. *Can Med Assoc J* 33:136-144, 1935
43. RABINOWITCH IM: Experiences with high carbohydrate-low calorie diet for treatment of diabetes mellitus. *Can Med Assoc J* 23:489, 1930
44. KEMPNER W, PESCHEL RL, SCHLAYER C: Effect of rice diet on diabetes mellitus associated with vascular disease. *Postgrad Med* 24:359-371, 1958
45. ERNEST I, LINNER E, SVANBORG A: Carbohydrate-rich, fat-poor diet in diabetes. *Am J Med* 39:594-600, 1965
46. RODGER NW, SQUIRES BP, DU E: Improved glucose tolerance in asymptomatic diabetics on diets high in simple sugar content. *Diabetes* 19:399, 1970




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47. Stone DB, Conner WE: The prolonged effects of a low cholesterol high carbohydrate diet upon the serum lipids in diabetic patients. *Diabetes* 12:127-132, 1963
48. Brunzell JD, Lerner RL, Hazzard WR, et al: Improved glucose tolerance with high carbohydrate feeding in mild diabetes. *N Engl J Med* 284:521-524, 1971
49. Lerner RL, Brunzell JD, Hazzard WR, et al: Mechanism of improved glucose tolerance on high carbohydrate diets in normals and mild diabetics (abstract). *Diabetes* 20:342-343, 1971
50. Patel JC, Talwalkar NG (editors): *Diabetes in the Tropics.* Bombay, Diabetic Association of India, 1966
51. Singh I: Low-fat diet and therapeutic doses of insulin in diabetes mellitus. *Lancet* 1:422-425, 1955
52. Patel JC, Metha AB, Dhirawani MK, et al: High carbohydrate diet in the treatment of diabetes mellitus. *Diabetologia* 5:243-247, 1969
53. Bush OB, Moriwaki T: Diet and diabetes mellitus, in *Diabetes in the Tropics*, edited by Patel JC, Talwalkar NG. Bombay, Diabetic Association of India, 1966, p. 533-539
54. Tsuji S, Wada M: *Diabetes Mellitus in Asia, 1970.* Amsterdam, Excerpta Medica Foundation, 1971
55. Floyd JC Jr, Fajans SS, Pek S, et al: Synergistic effect of essential amino acids and glucose upon insulin secretion in man. *Diabetes* 19:109-115, 1970
56. Bottger I, Faloona GR, Unger RH: Insulin, glucagon, and GLI responses to triglyceride meals (abstract). *Clin Res* 20: 84, 1972
57. Crespin SR, Greenough WB III, Steinberg D: Effect of sodium linoleate infusion on plasma free fatty acids, glucose, insulin, ketones in unanesthetized dogs. *Diabetes* 21:1179-1184, 1972
58. Soskin S, Levine R: A relationship between the blood sugar level and the rate of sugar utilization, affecting the theories of diabetes. *Am J Physiol* 120:761, 1937
59. Antar MA, Little JA, Lucas C, et al: Interrelationship between the kinds of dietary carbohydrate and fat in hyperlipoproteinemic patients. III. Synergistic effect of sucrose and animal fat on serum lipids. *Atherosclerosis* 11:191-201, 1970
60. Cohen AM, Teitelbaum A, Balogh M, et al: Effect of interchanging bread and sucrose as main source of carbohydrate in low fat diet on the glucose tolerance curve of healthy volunteer subjects. *Am J Clin Nutr* 19:59-62, 1966
61. Birchwood BL, Little JA, Antar MA, et al: Interrelationship between the kinds of dietary carbohydrate and fat in hyperlipoproteinemic patients. II. Sucrose and starch with mixed saturated and polyunsaturated fats. *Atherosclerosis* 11: 183-190, 1970
62. Anderson JW, Herman RH, Zakin D: Glucose tolerance and insulin response to prolonged high carbohydrate feeding in normal men. *Am J Clin Nutr* 21:529-539, 1968
63. Schlierf G, Reinheimer W, Stossberg V: Diurnal patterns of plasma triglycerides and free fatty acids in normal subjects and in patients with endogenous (Type IV) hyperlipoproteinemia. *Nutr Metab* 13:80-91, 1971
64. Grey N, Kipnis DM: Effect of diet composition on the hyperinsulinemia of obesity. *N Engl J Med* 285:827-831, 1971
65. Kipnis DM: Nutrient regulation of insulin secretion in human subjects. *Diabetes* 21(suppl 2):606-616, 1972
66. AMA Council on Foods and Nutrition, Food and Nutrition Board of the National Academy of Sciences—National Research Council: Diet and coronary heart disease. *JAMA* 222:1647, 1972
67. Kinsell LW: The diabetic diet, in *Diabetes Mellitus: Diagnosis and Treatment*, vol. 2, edited by Hamwi GJ, Danowski TS. New York City, American Diabetes Association, Inc., 1967, p. 97
68. West KM: Epidemiologic evidence linking nutritional factors to the prevalence and manifestations of diabetes, in *Nutrition and Diabetes Mellitus, VI Capri Conference*, edited by Froesch ER, Yudkin J. Italy, Il Ponte, 1972, p. 405
69. Yudkin J: Relation between diabetes, obesity, arteriosclerosis and nutrition. *Ibid.*, p. 395
70. Sussman KE: Control and its relationship to vascular complications, in *Juvenile-Type Diabetes and Its Complications.* Springfield, Charles C Thomas, Publisher, 1971, p. 360
71. Fajans SS: Current unsolved problems in diabetes management. *Diabetes* 21(suppl 2):678-684, 1972
72. Etzwiler DD: Who's teaching the diabetic? *Diabetes* 16: 111-117, 1967
73. Phillips MG: The nutrition knowledge of medical students. *J Med Educ* 46:86-90, 1971
74. Frankle RT, Williams ER, Christakis G: Nutrition education in the medical school: experience with an elective course for first-year medical students. *Am J Clin Nutr* 25: 709-719, 1972
75. Hinkle JE Jr: Customs, emotions, and behavior in the dietary treatment of diabetes. *J Am Diet Assoc* 41:341-344, 1962
76. Barnes RH: Doctors' dietary antics. *Nutr Today* 3:21-25, 1968
77. Steinke J, Thorn GW: Diabetes mellitus, in *Harrison's Principles of Internal Medicine*, 6th ed., edited by Wintrobe MM, Thorn GW, Adams RD, et al. New York, McGraw-Hill Book Co., Inc., 1970, p. 529
78. Bondy PK: Disorders of carbohydrate metabolism. Diabetes mellitus, in *Cecil-Loeb Textbook of Medicine*, 13th ed., edited by Beeson PB, McDermott W. Philadelphia, W. B. Saunders Co., 1971, p. 1647
79. Krall LP, Joslin AP: Hospital teaching unit, in *Joslin's Diabetes Mellitus*, 11th ed., edited by Marble A, White P, Bradley RF, et al. Philadelphia, Lea & Febiger, 1971, p. 256



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