### Dietary management of diabetes mellitus

By DAVID R. HADDEN and E. ANNE WILSON, The Sir George E. Clark Metabolic Unit, Royal Victoria Hospital, Belfast BT12 6BA

It seems strange that the early writers on diabetes did not seem to recognize the effect of food restriction on the symptoms of the disease (Malins, 1968). John Rollo (1797) may be regarded as the pioneer of modern dietary therapy, although his theoretical reasoning was confused. His dietary advice involved a rigorous and unpleasant lifestyle, which probably accounts for the unpopularity of dieting for diabetes at that time. 'Breakfast,  $1\frac{1}{2}$  pints of milk and  $\frac{1}{2}$  pint of lime water, mixed together; bread and butter. For noon, plain blood puddings, made of blood and suet only. Dinner, game or old meats which have been long kept; and as far as the stomach may bear, fat and rancid old meats, as pork, to eat in moderation. Supper, the same as breakfast'.

It is clear today that the basis of this diet was virtually total exclusion of carbohydrate, but the noxious effect of the high fat content would also have considerably reduced the total energy intake. Bouchardat (1875), in Paris, had arrived at a much more gastronomic compromise by eliminating the unpleasant rancid fat and concentrating instead on the value of eating little, especially of carbohydrate ('manger le moins possible'). His theories were strikingly proven during the seige of Paris in 1870, when he observed a reduction in glycosuria among his diabetic patients who were subsisting on the starvation rations available.

The success of rigorous dieting in those patients whom we would now recognize as non-insulin dependent diabetics must have been offset by much more disappointing results in those who would now be treated with insulin. Naunyn (1898) and others developed the concept with various forms of energy restriction. Just before the discovery of insulin many clinics existed primarily for the intensive dietary supervision of diabetic patients. (Ruthin Castle in North Wales was such a private clinic, where scientific and clinical studies were carried out under balance conditions with apparently encouraging results). The ultimate extension of these concepts of under-nutrition was shown by Allen et al. (1919) who extended the life expectancy of diabetic children from 1.3 to 2.9 years by virtual starvation.

The discovery of insulin produced a gradual but profound change in the dietary approach to diabetes. For example, at Ruthin Castle the carbohydrate content of the diet for the severely diabetic patients was increased from 25 to 250 g (Allison, 1978) and the effect of this increased energy intake must have been very striking. At that time the pioneer work at King's College Hospital by R. D. Lawrence (Lawrence, 1960) defined the principles for the dietary prescription for insulinrequiring diabetics which have survived virtually unquestioned to this day. The Lawrence Line Diet, with 10 g portions of carbohydrate was structured to allow 0029-6651/81/4023-5206 \$01.00 © 1981 The Nutrition Society

the useful practice of expressing the carbohydrate content in g as one tenth of the total energy content in calories. This continued the belief that this was primarily a carbohydrate restricted diet although by 1960 Lawrence himself had recognized the advantages of a carbohydrate intake of 150 g or more. With the present view of achieving a normally balanced food intake, the concept of diet for the insulin-requiring patient has moved away from restriction of carbohydrate towards simple energy planning—the 'food plan' has replaced the 'diet'. Nevertheless the underlying concept of carbohydrate restriction persists because that is the simplest nutrient to control, which has led to the statement that diets for diabetics are still high in fat and might thus be atherogenic (Joint Working Party of the Royal College of Physicians of London and the British Cardiac Society, 1977). A more correct view of this situation shows that the diabetic food plan may still contain proportionately more fat, but in absolute terms the nature of the plan would exclude the possibility of an actual excess of fat intake above that consumed by the average non-diabetic person in the United Kingdom.

At the same time dietary measures remained necessary for those diabetics for whom insulin was not necessarily life-saving. The management of these noninsulin-requiring diabetics gradually evolved along two divergent pathways: that of continuation of strict and rigorous diabetic control contrasting with a much more free food intake which often necessitated the treatment of the resultant hyperglycaemia with insulin. The dichotomy was not often discussed in print, the written articles tending to consider only the more severe ketosis-prone juvenile form of diabetes. The 'free' diet, (Knowles 1965), was the ultimate expression of this latter view and this still finds some supporters among paediatric diabetologists. No properly controlled clinical trials have ever been carried out to test the validity of one or other pole of this argument and it is unlikely that any such study would ever be considered ethical. Current practice certainly favours more definite dietary control for both insulin and non-insulin requiring diabetics. The Committee on Food and Nutrition of the American Diabetes Association (1971) stated 'the single most important objective in dietary treatment of diabetic patients is control of total calorie intake to attain ideal body-weight'.

When the oral hypoglycaemic agents became available for clinical use in the late 1950s the dichotomy persisted. All over the world it was found that some patients who had needed insulin to maintain normal blood sugars could safely withdraw the insulin and change to sulphonylurea therapy while still maintaining adequate control. Some of these patients still remain to this day on their tablet therapy and the concept that the tablet is the main therapy of their diabetic state has led to a tendency to neglect their dietary supervision. Bloom (1972) then showed that at least one third of patients at a diabetic clinic who were taking an oral hypoglycaemic agent could safely stop this therapy without relapse into hyperglycaemia, provided they continued to attend the clinic and took some cognisance of their food intake.

In many diabetic clinics there was a gradual tendency for more and more non insulin dependent diabetics to be treated with oral hypoglycaemic agents with only

lip service being paid to dietary supervision. In Belfast 39% of the whole diabetic clinic population was taking an oral hypoglycaemic tablet in 1970 (Hadden 1973) and in other UK clinics up to half the patients were so treated. In Germany it became the exception not to take such hypoglycaemic therapy and advertising practice at that time was to suggest that virtually no dietary control was necessary provided a suitable sulphonylurea or diaguanide was taken. This course of events set the scene for the pioneering work of the University Group Diabetes Program (1970) in attempting to find the best answer to the problem of management for the maturity onset diabetic: the motivation for that study was primarily to investigate the efficacy of either insulin or tolbutamide in preventing the long term vascular complications of diabetes. The controversial outcome of that study was to implicate the oral hypoglycaemic therapy in increasing cardiovascular mortality. No detailed assessment of dietary adherence was made in the UGDP and the placebo group were not given any different or more frequent dietary instruction or supervision than any of the other three treatment groups. It is not possible therefore to consider their placebo group as a truly 'diet treatment' group and this is borne out by the finding that after 4-5 years the mean weight of the placebo group was only 2% below the baseline value and mean weights for the whole study were actually higher than at onset (West 1973).

The swing of the pendulum against the misuse of the oral hypoglycaemic agents that followed the initial UGDP report has not yet reached an equilibrium. It is not clear whether the long term vascular complications of maturity onset diabetes are more related to blood glucose levels or to the achievement of ideal dietary and weight control. These two concepts are clearly linked to some extent, but the possibility of lowering blood sugar pharmacologically without the need to diet will remain an attraction, perhaps most closely related to the human dream for a tablet to promote weight loss in simple obesity without the rigor of the diet which that disorder also demands.

### Nutritional construction of the diabetic food plan

The practical considerations which dictate the food which we eat are those of simplicity, cost and taste; scientific nutritional considerations are in the background. It is clear that during the past 50 years there has been a gradual change in the nutritional prescribing of diets for diabetics and the accepted

Table 1. Diabetic food plan

	Insulin requiring diabetic (juvenile onset)		Non insulin requiring diabetic (maturity onset)	
	(g/d)	(% energy)	(g/d)	(% energy)
Carbohydrate	271	42	162	42
Protein	93	14	60	16
Fat	125	44	73	42
	10·5 MJ (2500 kcal)		6 4 MJ (1500 kcal)	

guidelines for diabetic patients are now much closer to the normal food intake of the UK population than they used to be.

The composition of current food plans constructed for insulin requiring and non insulin requiring diabetics are shown in Table 1. The juvenile onset diabetic with a 10.5 MJ (2500 kcal) daily energy intake and the maturity onset diabetic with a 6.4 MJ (1500 kcal) diet have a very similar proportionate distribution of carbohydrate, protein and fat. Although there are no standard nutrient analyses for Northern Ireland, it is probably relevant to examine the values available for the Republic of Ireland. In 1947, mean total energy intake was 13.0 MJ/person, with 102 g fat (29% of the energy). In 1973, mean total energy intake remained the same (13.1 MJ), but the fat intake had risen to 132 g (38% of the energy). This trend has been seen in other Western countries although it may be more pronounced in the Irish agricultural environment. In view of this increase in fat intake in the Western World, the US Senate produced a report on Nutrition and Human Needs (1977) which has been formalized as Dietary Goals for the USA (the McGovern Recommendations). The main recommendations are shown in Table 2 and compared with the percentage of total energy derived from different sources for Great Britain.

Table 2 Dietary goals for USA\*—comparison with British intake

	US goal for 'normal' diet (% total energy)	Present UK intake (% total energy)
Carbohydrate	55-60	46
Fat:	30	41
Saturated	10	2 I
Monosaturated	10	16
Polyunsaturated	10	4
Cholesterol (mg/d)	300	Not known
Sucrose (% total carbohydrate)	15	20

<sup>\*</sup>US Senate Committee Report on Nutrition and Human Needs, Washington DC, 1977.

There is much debate as to the practicality or desirability of the McGovern dietary goals. They are certainly idealistic in their return to a more starchy food intake with less animal products. They are also much closer to the food intake of the undeveloped countries and more economical in terms of a national budget. However, it is doubtful if a sudden move to this diet would achieve any general acceptance within a Western Society.

The principles and construction of the standard 6.5 MJ (1500 kcal) food plan as currently used in the Diabetic Clinic at the Royal Victoria Hospital are shown in Tables 3, 4 and 5.

The concept of carbohydrate exchanges dates back to the Lawrence Line Diet but we have limited our list of exchanges on the grounds of simplicity. A list of foods allowed in addition to the food plan, and more importantly, of those not allowed is printed on the food plan card (Table 6).

# Table 3. Details of the 6.4 MJ (1500 kcal) food plan

Carbohydrate:	Bread (white or brown) (g)	120
•	Potatoes (g)	180
	Cornflakes (g)	15
	Biscuits, plain (g)	20
	Fruit (two portions) (g)	200
	Milk allowance (ml)	300
Protein:	Lean Meat (two portions) (g)	120
	Egg (one) (g)	55
Fat:	Butter (g)	30

# Table 4. Principles of the Belfast diabetic food plan

- 1. The diet is prescribed initially on an energy basis.
- Carbohydrate content is defined as between 40 and 46% of the total energy intake.
- 3. Sucrose and sucrose-containing foods are excluded.
- 4. Glucose, lactose and fructose are excluded as additives.
- The food plan is otherwise arranged as a modification of normal food intake.

#### Table 5. Construction of the diabetic food plan

- 1. Protein foods are allowed in normal amounts.
- Fats and oils allowed in moderation except in total energy restriction.
- 3. Most vegetables allowed freely.
- 4. Fruit—two to three helpings/d.
- 5. Milk-0.5 to 1 pint/d.
- Starches—measured quantities of bread, cereals, plain biscuits and potatoes weighed on a Salter No. 18D Dietary Balance. Carbohydrate exchanges permitted on a limited basis only.

# Table 6. Foods allowed in addition to the food plan and foods which are not allowed

Allowed	Not allowed
Beef and chicken stock cubes	Sugar, glucose and lactose
Diabetic fruit squash	Biscuits, cakes and pastries
Lemon juice	Confectionery of all kinds
Meat and vegetable extracts	Fruit loaf, fancy bread and malted bread
Saccharine preparations for sweetening	Ice cream, iced lollies
Soda water	Milk drinks
Tea and coffee	Minerals and squashes
Pickles	Pickles and chutney
Vinegar	Preserves—jam, marmalade and honey
'Diabetic' preparations may be used on	Puddings and desserts
the dietitian's advice	Sauces and salad cream
	Tinned and dried fruit
	Tinned and packet soups
	Wines, beer and spirits

Prospective life-table analysis of intensive dietary therapy of maturity-onset diabetes

All diabetic patients aged 40–70 years with classical symptoms of diabetes mellitus who presented to the Diabetic Clinic at the Royal Victoria Hospital, Belfast, between October, 1972 and October, 1976 were eligible for inclusion in this study. Only those who had classical symptoms of diabetes mellitus or those few asymptomatic patients who had two blood glucose levels >18 mmol/l on the initial oral glucose tolerance test (OGTT) were enrolled. In general this included patients whose random plasma glucose was >10 mmol/l and 2 h plasma glucose after 50 g glucose by mouth >10 mmol/l. This would exclude so called 'border-line diabetics'. Of the 450 eligible patients seen during the four year enrolment period, 224 were entered in the prospective study. Of these, 153 have been followed up alive to 1st December, 1978 and fourteen had died before that date.

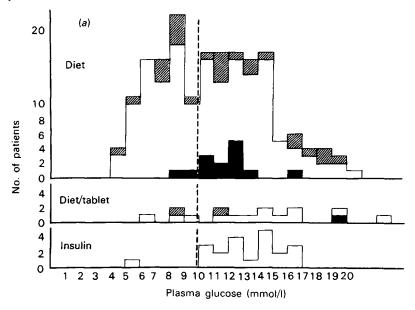
All patients were initially treated with a food plan using the principles stated earlier. During the follow up period twenty-two patients were changed to treatment with insulin because of continuing weight loss, ketoacidosis and uncontrolled fasting hyperglycaemia (>20 mmol/l). A further fourteen patients in whom fasting blood sugars at routine review remained considerably elevated (15-20 mmol/l) with or without symptoms due to the hyperglycaemia were started on treatment with an oral hypoglycaemic agent (tolbutamide 1 5 g/d or metformin 1 5 g/d). Before a patient was considered for oral hypoglycaemic therapy, every effort was made to ensure dietary adherence and these patients were often admitted to the diabetic ward both for supervision and demonstration that even if they did keep to their diet, adequate blood sugar control could not be maintained. Twenty-two patients did not complete this review for various reasons including moving to another area or refusal to reattend the clinic. However, we know that all these twenty-two patients were alive on 1st December, 1978.

In previous reports on this study (Hadden et al. 1975; Wilson et al. 1980) it has been demonstrated that while the mean weight of the patients fell gradually from about 79.4 to 72.6 kg during the first 6 months, mean fasting plasma glucose decreased much more rapidly, from over 12 mmol/l to approximately 8 mmol/l within one month and then remained fairly constant throughout the further period of follow up. It was also noted that those who were graded as poor in dietetic adherence had significantly increased plasma insulin and triglyceride values, although plasma glucose was not significantly higher, either fasting or 2 h after an oral glucose load.

Fig. 1(a) shows the frequency distribution of (a) initial fasting plasma glucose compared to (b) the mean follow up fasting plasma glucose for the three groups of patients (166 on diet only; twenty-two on insulin and fourteen on an oral hypoglycaemic agent) at the 1st December, 1978. The shaded areas on the graph represent results for the twenty-two patients for whom complete follow-up information was not available. The majority of patients had an initial fasting plasma glucose above 10 mmol/l. Four patients had an initial fasting plasma glucose of between 4 and 5 mmol/l. Those patients who were subsequently treated



# Nutrition and diabetes



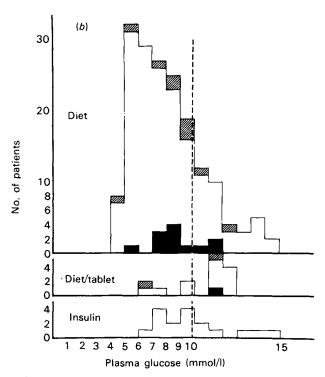


Fig. 1. (a) Frequency distributions of initial fasting plasma glucose for the three treatment groups at 1st December, 1978 (166 patients on diet only, twenty-two on insulin and fourteen on an oral hypoglycaemic agent); (b) frequency distributions of the mean of all the available follow-up fasting plasma glucose values for the same groups. The shaded areas represent results from twenty-two patients for whom complete follow-up was not available. The solid areas represent results from the thirteen patients who had died during the course of the study.

with oral hypoglycaemic agents or insulin had initial fasting plasma glucose values in the higher range. The solid bars in the histograms represent those patients who had died by 1st December, 1978. The distribution of this smaller group is similar to that of the whole population. Fig. 1(b) shows the very considerable improvement in mean fasting plasma glucose during the review period. Only a small proportion had values above 10 mmol/l, although those who were treated with oral hypoglycaemic agents or insulin showed proportionately more in that area in spite of their attempted therapeutic control. Only one patient in the oral hypoglycaemic treated group and none in the insulin group died so no conclusions are possible on the relationship between blood sugar control by either of these agents and longevity.

Life table analysis was carried out for those patients treated by diet only and compared with the expected mortality at 5 years of an age and sex matched population in Northern Ireland. This preliminary analysis of the Belfast Diet Study shows a five year survival for those diabetic patients treated by diet only of 87.6% compared to 91.7% for a matched population from Northern Ireland, which is not significantly different (P>0.05). Thus dietary management for a very large proportion (about 80%) of symptomatic maturity onset diabetic patients is possible, and extended analysis up to the present suggests that long-term survival is similar to that found in the normal population. The six year assessment of this study is now in progress and analysis will be directed at the question of whether the mildly elevated fasting plasma glucose levels which have been observed in some of these patients in spite of reasonable dietary adherence have any effect on survival.

#### REFERENCES

Allen, F. M., Stillman, E. & Fitz, R. (1919). Monogr. Rockefeller Inst. Med. Res. No. 11.

Allison, R. S. (1978). Ulster Med. J. 47, 10.

Bloom, A. (1972). Il R. Coll. Physicians Lond. 7, 61.

Bouchardat, A. (1875). De la glycosurie ou diabete sucré, vol. II. Paris: Gemer-Balliere.

Committee on Food and Nutrition of the American Diabetes Association (1971). Diabetes 20, 633.

Hadden, D. R. (1973). Ir. J. med. Sci. Suppl. 11.

Hadden, D. R., Montgomery, D. A. D., Skelly, R. J., Trimble, E. R., Weaver, J. A., Wilson, E. A. & Buchanan, K. D. (1975). Br. Med. J. iii, 276.

Joint Working Party of the Royal College of Physicians, London, and the British Cardiac Society. (1977). Jl R. Coll. Physicians Lond. 10, 213.

Knowles, H. C., Grest, G. M., Lampa, R. N., Kessler, M. & Stillman, T. O. (1965). Diabetes 14, 239.

Lawrence, R. D. (1960). The Diabetic Life (16th ed). London: J & A Churchill.

Malins, J. M. (1968). Clinical Diabetes Mellitus. London: Eyre and Spottiswoode.

Naunyn, B. (1898). Der Diabetes Mellitus. Wien: A. Holder.

Rollo, J. (1797). An account of two cases of the diabetes mellitus; to which are added a general view of the nature of the disease, and its appropriate treatment. London: T. Dilly at the Poultry.

University Group Diabetes Program (1970). Diabetes 19, Suppl. 2, 789.

US Senate Committee (1977). Report on Nutrition & Human Needs. Washington D.C.

West, K. M. (1973). Ann. intern Med. 79, 425. Wilson, E. A., Hadden, D. R., Merrett, J. D., Montgomery, D. A. D. & Weaver, J. A. (1980). Br. Med. J. i, 1367.

Printed in Great Britain