Discussion

Our results indicate that using the foam results in a good topical application of steroid to the lower rectum but that more proximal spread is limited and infrequent. One criticism that could be put forward is that we made no attempt to ensure that the rectum was clear of any hard faecal matter that might prevent the spread of the foam. All the patients, however, had loose stools, and the foam spread no further in them than in the controls.

The aim of the study was simply to assess the extent of spread of the foam after rectal administration and not to attempt to compare the product with any other in terms of extent of spread or efficacy. Certain clinical implications, however, could not be avoided. On the basis of topical application alone the product is a rational treatment for only proctitis and mild distal ulcerative colitis. It would, however, seem illogical to use it for more proximal disease, and our clinical impression has been that under these circumstances it is less effective. If it is of benefit in more proximal disease this can be assumed to be only the result of absorption of the steroid from the foam by the rectal mucosa, any effect being that of systemic steroid. Of the clinical trials quoted by the manufacturer, only that of Scherl and Scherl[4] attempted to differentiate between patients with proximal or distal disease. Unquestionably, however, the foam is more comfortable and easier to retain than a retention enema, and since the patient need not be immobilised, the foam obviously has a place in outpatient practice for patients with proctitis and distal ulcerative colitis.

We wish to thank Professor B Pullan for his advice, and Stafford Miller Limited for supplying the Colifoam.

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Improved glucose control in maturity-onset diabetes treated with high-carbohydrate-modified fat diet

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Summary and conclusions

Fourteen patients with established maturity-onset diabetes were treated as outpatients with a high-carbohydrate-(about 60% of total daily energy requirements)-modified fat diet (ratio of polyunsaturated fatty acids to other fatty acids 1:1) for six weeks. Commercially available and acceptable cereal foods and tuberous vegetables high in both digestible and non-digestible carbohydrates were used. Simple sugars were restricted. Compared with their usual, low-carbohydrate diabetic diet this diet resulted in a fall in basal plasma glucose concentration (average of values measured at 0300, 0500, and 0700), mean preprandial plasma glucose concentration (average of values measured at 0800, 1230, and 1730), and percentage of glycosylated haemoglobin. Modifying dietary fat also decreased the fasting plasma cholesterol concentration.

The findings suggest that it is no longer justifiable to prescribe a low-carbohydrate diet for maturity-onset diabetes.

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Introduction

Since insulin became routinely available over 50 years ago there has been some relaxation in restricting dietary carbohydrate in diabetic clinics. Despite this, in 1971 most clinics in Great Britain were still recommending a low-carbohydrate diet (carbohydrate constituting about 40% of total daily energy) for both maturity-onset and insulin-requiring disease.[1] Increasing evidence suggests, however, that dietary carbohydrate is not detrimental to blood sugar control.[2] An average carbohydrate-containing diet (carbohydrate providing 50-55% of total energy) gives a similar improvement in fasting blood sugar values to that obtained with the lower carbohydrate diet when recommended in newly diagnosed maturity-onset diabetes.[3] Several workers[4 5] have suggested that much higher carbohydrate-containing diets reduce the fasting blood sugar concentration. More recently it has been shown that increasing the dietary fibre,[6] particularly guar or pectin,[7] reduces the postprandial blood sugar concentration. These studies, however, used formula diets,[4-6] unacceptably high amounts of carbohydrate,[4 5] and carbohydrate foods likely to be unpalatable to some patients.[7] Furthermore, measures of diabetic control used were principally confined to fasting blood glucose and 24-hour urinary glucose values.

We therefore decided to examine in greater detail the effect in maturity-onset diabetes of an acceptable, high-carbohydrate diet composed of commercially available high-carbohydrate foods such as wholemeal bread and tuberous vegetables.

Patients and methods

Eighteen patients with established maturity-onset diabetes (15 men and three women) were recruited into the study. Their mean age ($\pm$SE of mean) was $54 \pm 2{\cdot}0$ years and mean percentage of ideal body weight ($\pm$SE of mean; Metropolitan Life Assurance Co) $106{\cdot}3 \pm 3{\cdot}3$. All were considered to be clinically and chemically stable (14 taking sulphonylureas and the remainder dietary treatment alone), their mean duration of diabetes (±SE of mean) being 4·4±0·6 years. On entry patients were interviewed by an experienced dietitian (JE) and had their current diet analysed. They were then allocated at random to one of two groups. The first group continued with their usual, low-carbohydrate (LC) diet (about 40% of daily energy from carbohydrate; ratio of polyunsaturated fatty acids to other fatty acids about 1:3). The second group was recommended an isoenergetic high-carbohydrate-(HC)-modified fat diet (about 60% of daily energy requirement from carbohydrate; ratio of polyunsaturated fatty acids to other fatty acids ≥1:1). Tables I and II give an example.[8 9] Monosaccharides and disaccharides were restricted. After six weeks the patients were admitted for a 24-hour metabolic profile starting at 1730. Thereafter the diets were reversed and a further six weeks elapsed before a second admission for another 24-hour metabolic profile.

TABLE I—*Constituents of low-carbohydrate and high-carbohydrate diets*

Low-carbohydrate diet			High-carbohydrate diet		
	Item	kcal*		Item	kcal*
Breakfast					
60 g	White bread	138	120 ml	Skimmed milk	80
29 g	Butter	218	150 g	Wholemeal bread	325
60 g	Mandarins	44	30 g	Ham	62
120 ml	Milk	76	7·5 g	Flora margarine	56
10 g	Wheat crispbreads	38	10 g	Bran crispbread	38
60 g	Egg	92	120 g	Orange	40
	Total	606		Total	601
Mid-morning					
60 ml	Milk	38	60 ml	Skimmed milk	20
30 g	White bread	69	60 g	Wholemeal bread	130
13 g	Butter	98	7·5 g	Flora margarine	56
	Total	205		Total	206
Midday					
90 g	Minced beef	213	30 g	Beef, roast	71
150 g	Potatoes, mashed	115	330 g	Potatoes, boiled	253
60 g	Carrots	10	120 g	Carrots	20
60 g	Ice-cream	112	15 g	Rice } as pudding	51
60 g	Tinned fruit	44	240 ml	Skimmed milk } as pudding	80
			60 ml	Skimmed milk	20
	Total	494		Total	495
Mid-afternoon					
60 ml	Milk	38	60 ml	Skimmed milk	20
30 g	White bread	69	60 g	Wholemeal bread	130
8·5 g	Butter	64	7·5 g	Flora margarine	56
15 g	Beef	35			
	Total	206		Total	206
Evening meal					
60 g	Cheese	240	30 g	Ham	62
60 g	Tomato + salad vegetables	10	60 g	Tomato + salad vegetables	10
30 g	White bread	69	150 g	Wholemeal bread	325
15 g	Butter	113	7·5 g	Flora margarine	56
120 g	Apple	40	105 ml	Skimmed milk	35
120 g	Pear	44	120 g	Apple	40
90 ml	Milk	57	120 g	Pear	44
	Total	573		Total	572
Bedtime					
240 ml	Milk	152	300 ml	Skimmed milk	100
15 g	Horlicks	56	120 g	Orange	40
10 g	Ryvita	64	60 g	Wholemeal bread	130
14 g	Butter	106	14·5 g	Flora margarine	108
	Total	378		Total	378

*1000 kcal ≈ 4·2 MJ.

Immediately on admission for the 24-hour study a Teflon cannula was inserted into a large forearm vein for sampling free-flowing blood. While in the ward all but two patients received meals that were individually isoenergetic between the two diets. During the 24-hour stay all 18 patients individually received diets that were isocaloric between the two studies. Blood samples were drawn at half-hour to two-hour intervals. Samples were analysed for plasma glucose with a glucose oxidase method (Boehringer GOD-perid), for immunoreactive insulin with a standard radioimmunoassay,[10] and for serum triglycerides with a glycerokinase method.[11] Fasting lipoproteins (at 0800) were assayed by precipitation techniques,[12 13] and cholesterol concentrations determined by an automated Liebermann-Burchardt reaction.[14] In most patients fasting (0800) glycosylated haemoglobin was assayed by cation exchange chromatography[15] (quality control mean 6·5±SD 0·4%).

Adherence to diet was monitored by estimating the linoleic acid (18:2) content of serum triglycerides.[16] Patients adhering to a diet containing a high ratio of polyunsaturated fatty acids to other fatty acids (≥1:1) have over 20% of the total serum triglyceride fatty acid as linoleic acid. This test does not provide a direct indication that subjects have modified their carbohydrate intake, but we know of no accurate practical means of assessing this. Nevertheless, highly motivated volunteers who adhere to one aspect of an experimental diet will probably also adhere to the other component of the study. Statistical tests used were Student's paired *t* test and the Wilcoxon matched pairs test where indicated. Results are expressed as means ± SE of mean.

Results

Triglyceride fatty acid—After six weeks of taking the HC diet 14 of the 18 patients showed a linoleic acid content of serum triglyceride fatty acid of over 20% (mean 15·8±1·2% with the LC diet; 26·5±1·4% with the HC diet). Two of the 14 patients also showed a linoleic acid content exceeding 20% with their normal diet. Thus all 14 patients showed satisfactory adherence to the HC diet from this test, and the following results therefore relate only to these patients. The remaining four patients showed a low proportion of linoleic acid with both diets (15·8±0·8%, LC; 14·1±1·9 HC), suggesting that they did not adhere to the experimental diet.

Twenty-four-hour blood glucose profile (fig 1)—The plasma glucose profile with the HC diet for the 12 compliant patients who received meals isoenergetic between the two diets while in the ward was significantly lower than with the LC diet from 0500 to 0830. The two profiles otherwise differed significantly only at 2200 and 1230. Fasting glucose, mean basal glucose (average of values measured at 0300, 0500, and 0700), and mean preprandial glucose concentrations (average of values measured at 1730, 0830, and 1230) were all significantly lower after the HC diet; however, weight, mean daily glucose concentration, and Schlichtkrull's "M" value[17] did not differ between the two diets (table III).

Fasting serum triglyceride and total lipoprotein cholesterol concentrations—Fasting serum triglyceride concentrations did not differ significantly between the two diets (LC 1·30±0·09 mmol/l (115·0±8·0 mg/100 ml); HC 1·22±0·09 mmol/l (108·0±8·0 mg/100 ml)). The fasting total cholesterol concentration, however, was significantly lower with the HC diet (4·4±0·3 mmol/l; 170·0±11·6 mg/100 ml) than with the LC diet (5·1±0·3 mmol/l; 197·0±11·6 mg/100 ml) ($P < 0·001$). Although the concentrations of high-density-lipoprotein cholesterol (1·4±0·1 mmol/l; 54·1±3·9 mg/100 ml), low-density-lipoprotein cholesterol (2·6±0·3 mmol/l; 100·4±11·6 mg/100 ml), and very-low-density lipoprotein cholesterol (0·5±0·1 mmol/l; 19·3±3·9 mg/100 ml) were lower with the HC diet than with the LC diet (1·6±0·1 mmol/l (61·8±3·9 mg/100 ml); 2·8±0·3 mmol/l (108·1±11·6 mg/100 ml); and 0·7±0·1 mmol/l (27·0±3·9 mg/100 ml) respectively), the differences were not significant.

TABLE II—*Total 24-hour intakes provided by low-carbohydrate and high-carbohydrate diets listed in table I*

	Protein (g)	Fat (g)	Carbohydrate (g)	Energy (kcal*)	Fibre (g)
Low-carbohydrate diet (% of energy provided)	91·3 (16)	125·0 (50)	217·0 (34)	2462·0	35·5
High-carbohydrate diet (% of energy provided)	96·8 (16)	59·8 (23)	357·49 (61)	2458·0	78·0

*1000 kcal ≈ 4·2 MJ.

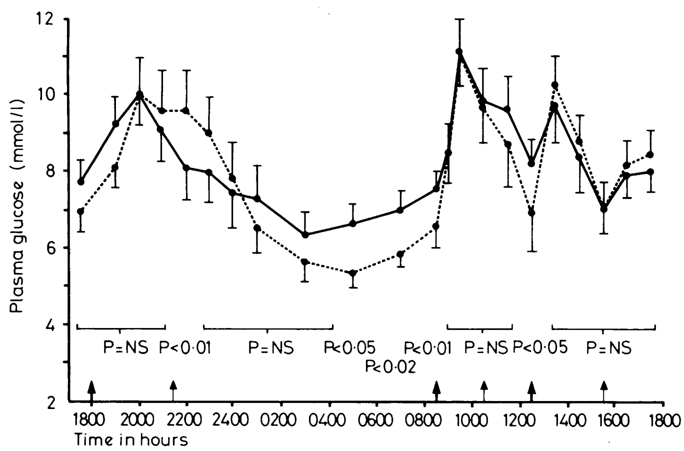

FIG 1—Twenty-four-hour plasma glucose profile for 12 patients given meals isoenergetic between the two diets. Bold arrows indicate times of main meals. Small arrows indicate times of snacks. ●——● LC (normal diabetic diet with about 40% of daily energy intake from carbohydrate). ●- - -● HC (high-carbohydrate diet with about 60% of daily energy intake from carbohydrate). Student's paired *t* test. (Plasma glucose: 1 mmol/l ≈ 18 mg/100 ml.)

TABLE III—*Mean (±SE of mean) weight, blood glucose concentration, and Schlichtkrull "M" values[17] after treatment with the two diets*

	High-carbohydrate diet	Low-carbohydrate diet	Significance*
Weight (kg)	69·3±2·3	69·6±2·2	NS
Fasting glucose (0830) (mmol/l)	6·5±0·4	7·4±0·5	P<0·01
Basal glucose (mmol/l)†	5·5±0·4	6·7±0·4	P<0·002
Preprandial glucose (mmol/l)‡	7·0±0·6	7·8±0·5	P<0·01
Daily glucose (mmol/l)	7·7±0·6	8·1±0·6	NS
Schlichtkrull "M" value	34·7±8·2	33·2±8·5	NS

*Significance of difference in "M" values assessed with Wilcoxon matched pairs test; other differences assessed with Student's paired *t* test.
†Average of three early-morning measurements.
‡Average of three evening and night measurements.
Conversion: SI to traditional units—Blood glucose: 1 mmol/l ≈ 18 mg/100 ml.

Percentage of glycosylated haemoglobin (fig 2)—The proportion of glycosylated haemoglobin fell significantly from 9·5±0·4% with the LC diet to 8·5±0·3% with the HC diet ($P < 0·01$) in the 12 compliant patients for whom we have data. The normal value was 7·1±0·1% (115 non-diabetics).

Twenty-four-hour insulin profile—In the 12 compliant patients the mean average insulin concentrations (LC diet 19·7±5·3 mU/ml; HC diet 16·7±3·2 mU/ml), mean basal insulin concentrations (LC 6·0±1·6 mU/ml; HC 4·5±0·8 mU/ml), and mean preprandial insulin concentrations (LC 13·1±1·4 mU/ml; HC 10·6±1·8 mU/ml) did not differ significantly between the two diets.

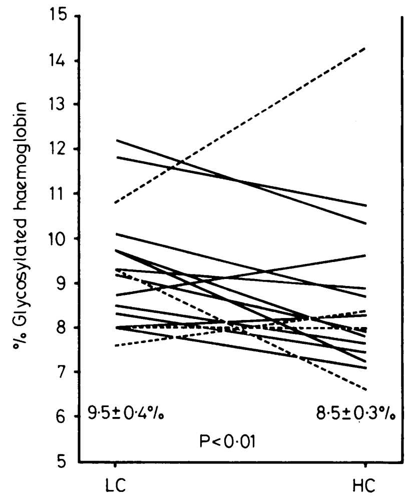

FIG 2—Change in percentage concentration of glycosylated haemoglobin after six weeks on high-carbohydrate (HC) diet. Mean ±SE of mean results refer only to compliant patients. ●——● Compliant patients. ●- - -● Non-compliant patients. Wilcoxon matched pairs test. LC=Low-carbohydrate diet.

Discussion

Epidemiologically there is no evidence that diets high in carbohydrate increase diabetic morbidity or mortality.[18 19] This has led to increasing uncertainty in Britain about the optimal proportion and type of carbohydrate for the diet in maturity-onset diabetes. Furthermore, diabetics changed from diets low to high in digestible carbohydrate with or without increased non-digestible carbohydrates apparently have a lower fasting blood sugar concentration and either unchanged or reduced 24-hour urinary glucose excretion values.[4 5] Diabetics given diets high in fibre[6 7] have been shown to have reduced post-prandial glucose concentrations and again reduced 24-hour urinary loss. Many of these studies were relatively short-term, however, and the indices of diabetic control used (single fasting blood glucose estimations and 24-hour urinary glucose excretion) are not very sensitive indicators of carbohydrate metabolism. Moreover, the studies were not confined to readily available and acceptable carbohydrate-containing foods such as the cereal foods and tuberous vegetables used here.

We found that a diet high in both digestible and non-digestible polysaccharides (monosaccharides and disaccharides were avoided) taken for six weeks does not cause a deterioration in diabetic control. Indeed, 24-hour glucose profiles showed a reduction in the basal glucose concentration, so that the post-prandial rises started from a lower value. Furthermore, the fall in glycosylated haemoglobin observed during the HC diet suggests that this diet may improve control in the long term.

The reduced glucose baseline from which the postprandial surges occurred is consistent with the increased insulin sensitivity found in normal[20] and diabetic[21] subjects and the decreased fasting glucose values in patients with maturity-onset diabetes[4 5] treated with HC diets. We believe that this reduced glucose baseline is the main effect of digestible carbohydrate on blood glucose control.

Jenkins *et al*[7] found that guar and pectin, both non-digestible carbohydrates, decrease the peak value but increase the duration of the postprandial glucose surge. From our study, although non-digestible carbohydrate—mostly tuberous and cereal in origin—was increased in the HC diet, we are uncertain whether the improved diabetic control was in any part due to this increased amount of dietary fibre. Certainly bran, a cereal fibre, is much less efficacious than the viscous gelling fibres such as guar in reducing postprandial glucose values in short experiments.[22]

Our results also confirm that modifying dietary fat by both decreasing daily cholesterol and total fat intake and increasing the polyunsaturated to saturated fatty acid ratio reduces serum cholesterol. Diabetics have an increased incidence of cardiovascular disease,[23] and although there is no definitive evidence that lowering the cholesterol concentration is beneficial, it may well be worth while in these particularly susceptible patients. An important negative finding was that triglyceride concentrations were similar with the two diets, since some workers regard HC diets as hypertriglyceridaemic. Hypertriglyceridaemia may well be transient,[24] because it does not persist when such a diet is continued longer.

Thus we have shown that an acceptable diet composed of increased amounts of commonly available cereal and tuberous foods and therefore high in carbohydrate is not detrimental to diabetic control. Indeed, the lower fasting blood glucose and glycosylated haemoglobin values recorded during the experimental diet suggest that over a longer period a diet high in carbohydrate may well improve diabetic control. Whether this small improvement in glucose control and reduction in serum cholesterol will benefit the diabetic in the longer term can be evaluated only by long-term prospective studies. From our evidence, however, it no longer seems justifiable to continue routinely to prescribe low-carbohydrate diets for maturity-onset diabetes.

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ONE HUNDRED YEARS AGO Dr James Stevenson, Medical Officer of Health for Paddington, has performed useful service in preparing a careful and convincing memorandum for the Vestry of Paddington upon the propriety of making suitable public latrine accommodation in the metropolis, and of course, so far as that vestry is concerned, especially in their district. Medical men are aware of the peculiar necessity which exists for the provision of such accommodation for women, who are by circumstances especially in need of it, and to whom the want of it is not unfrequently a cause of extreme suffering and chronic disease. Special provision is made by the Public Health Acts of 1848 and 1855, authorising, and therefore in a certain sense requiring, vestries and district boards to provide and maintain such public conveniences. In Glasgow, there are three female lavatories, which, the Sanitary Inspector reports, were used during thirty-four weeks of last year by 7,981 persons. In Nottingham, there is one lavatory for females, with five closets and one washstand on the ground floor, for the use of which one penny is charged; upstairs, there are four washhand-basins, with additional toilet articles, for the use of which twopence is charged. During the year ended December 30th, 1878, 1040 females used the lavatories and 18,649 the closets, at a total charge of £82 2s 11d. Dr. Seaton, the Medical Officer of Health for Nottingham, says: "In speaking of the success of the public closets, I am more especially looking to the very substantial sanitary advantages which have resulted from their establishment. They are generally regarded as a great public boon." Nor need such accommodation be a heavy burden, if any burden at all, upon the rates. By making suitable arrangements for the accommodation of the classes who would be willing to pay a very small fee, a considerable return is easily obtained for the use of such conveniences. Those at the Waterloo Station, it is stated, yielded £1000 a year, and those at Cannon Street and Charing Cross each a still larger sum. Such places might be attached to public baths and wash-houses, or other buildings belonging to the parish, in the form of lodges at the entrance of parks, public gardens, recreation grounds, open spaces, disused burial-grounds, bridges, or in suitable recesses on vacant plots of ground in the streets. Such public lavatories for women, or waiting-rooms for women, marked first and second class, might be made readily distinguishable by a coloured lamp; and there is every reason to believe that, if suitably placed and arranged, they would prove a very great boon to the immense population of London which is at present ill provided in this respect, and that in fulfilling the duty thus imposed upon them by Acts of Parliament, which they have hitherto neglected, the vestries and district boards will do an act which is at once highly popular and useful, and which will probably be found to be in the end costless. Dr Stevenson deserves public thanks for so thoroughly investigating the subject and furnishing so able a report. (*British Medical Journal*, 1879.)