Mean urinary glucose concentrations and insulin dose before and during one year of guar administration

Case No	Before guar	Week						
		4	8	12	16	20	24	52
Mean urinary glucose (mmol/l)								
2	83.2	7.2	5.5	11.1	2.2	10.6	22.8	22.8
3	16.6	3.9	2.2	3.9	4.4	2.8	6.7	3.3
4	27.7	26.1	69.4	43.3	55.6	55.6	55.6	—
5	27.7	15.6	31.1	23.9	22.2	20.0	49.4	25.6
6	83.3*	28.9	27.7	30.0	28.9	42.8	27.8	17.8
7	111.0	—	22.2	—	—	44.4	87.8	—
8	55.6	15.0	8.9	10.0	18.3	22.2	15.0	13.3
10	30.0	35.6	35.6	35.6	17.8	16.1	23.9	27.8
11	111.1	13.9	19.4	10.0	21.7	3.3	33.9	—
12	16.7	37.8	23.9	13.9	4.4	23.9	40.6	25.6
13	19.7	1.5	8.5	13.2	5.8	28.8	16.5	3.6
Mean	53.0	18.6	23.1	19.5	18.1	24.6	34.6	17.5
±SEM	±11.3	±4.1	±5.7	±4.1	±5.1	±5.2	±7.0	±3.5
p		<0.05	<0.05	<0.05	<0.05	<0.05	NS	<0.05
Insulin (U/24 h)								
2	0	0	0	0	0	0	0	0
3	26	19	19	20	20	20	16	17
4	58	48	31	34	32	38	36	—
5	52	39	34	34	32	32	32	32
6	36	36	35	32	28	31	34	34
7	56	—	40	40	36	34	36	—
8	44	28	30	28	28	28	28	26
10	44	38	38	38	40	40	40	40
11	0	0	0	0	0	0	0	—
12	76	66	66	64	64	58	57	56
13	0	0	0	0	0	0	0	0
Mean	49 ± 5	39 ± 6	37 ± 5	36 ± 5	31 ± 6	35 ± 4	35 ± 4	34 ± 5
±SEM		<0.01	<0.01	<0.002	<0.002	<0.002	<0.002	<0.02
p								

*This value differs from the figure in the original report,³ which has been corrected due to availability of complete first week pretreatment urine charts.

p = Significance of mean absolute difference from control values.

Conversion: SI to traditional units—Glucose: 1 mmol/l ≈ 18 mg/100 ml.

glucose and insulin values over one year are shown in the table. The case numbers are the same as those in our original publication.³

Each urinary glucose value represents the mean result of seven consecutive days' home urine tests on fresh urine specimens passed immediately before breakfast, lunch, dinner, and bed and performed by the patients using Clinitest tablets (Ames). In case 4 the values represent the results of only two to three days' testing. No changes were made in the insulin regimen, apart from changes in dose, or in the diet, except in one patient (case 11) who was given an essentially vegetarian diet while in the hospital metabolic ward before he started to take guar. His daily carbohydrate intake was increased from an average of 150 g daily to 200 g largely at the expense of fat and he was subsequently encouraged to maintain this diet while taking guar.

At six months, the fasting blood glucose of the 11 diabetics had risen non-significantly by 0.6 mmol/l (11 mg/100 ml) (4%) from 14.1±1.5 mmol/l (254±27 mg/100 ml) and their body weight fallen by 2.2 kg (3.4%; p<0.01). In the eight who were followed up for one year mean fasting blood glucose, which was 13.2±1.9 mmol/l (238±34 mg/100 ml) before guar, remained the same at 13.3±2.0 mmol/l (240±36 mg/100 ml) while their weight fell by 1.9 kg (3%; p<0.02) below the pre-guar value. Two individuals (cases 11 and 2) were withdrawn from the study at 6 and 12 months respectively and placed on 48 and 24 units of insulin. Immediately before insulin, while still on guar, their weights showed falls of 2.7 kg (4.4%) and 2.3 kg (5.4%) and fasting blood glucose values were reduced by 4 mmol/l (72 mg/100 ml) (26%) and 2 mmol/l (36 mg/100 ml) (24%) respectively. Three patients (cases 4, 5, and 12) who had always suffered from hypoglycaemic episodes and lived at a distance from the centre voluntarily reduced their insulin dose excessively and showed increased glycosuria and fasting blood glucose values. Urinary glucose concentrations were, however, lower in all those whose insulin was not reduced more than 19 U/day.

Other mean values measured before and after six months of guar treatment (in only eight patients) were: total cholesterol 5.0±0.2 and 4.5±0.3 mmol/l (193±8-178±12 mg/100 ml) (p<0.05); high density lipoprotein cholesterol 1.2±0.2 and 1.3±0.1 mmol/l (46±8-50±4 mg/100 ml); low density lipoprotein cholesterol 3.2±0.2 and 2.6±0.3 mmol/l (123±8-100±12 mg/100 ml) (p<0.01); triglyceride 1.2±0.2 and 1.3±0.3 mmol/l (106±18-115±27 mg/100 ml); zinc 1.20±0.06 and 1.15±0.05 mmol/l (7.8±0.4-7.5±0.3 mg/100 ml); copper 1.14±0.13 and 1.07±0.06 mmol/l (7.3±0.8-6.8±0.4 mg/100 ml); calcium 2.30±0.02 and 2.36±0.03 mmol/l (9.4±0.08-9.4±0.12 mg/100 ml); urea 7.0±0.4 and 7.6±0.4 mmol/l (42±2.4-46±2.4 mg/100 ml); and creatinine 83±5 and 84±5 mmol/l (0.9±0.06-1.0±0.06).

Comment

We conclude, therefore, that providing the insulin dose is not reduced by more than about a quarter, long-term guar treatment is associated with reduced urinary glucose concentrations, as in short-term studies.³ In addition, at six months there was a 15% reduction in the low density lipoprotein fraction of the total cholesterol without a

reduction in the high density lipoprotein cholesterol. A reduction in this fraction is thought to protect against cardiovascular disease.⁴ We find no evidence from blood values that guar causes trace-element deficiency.

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¹ Cohen M, Martin FIR. Guar crispbread in the diabetic diet. *Br Med J* 1979;ii:616.

² Reinhold JG, Faradji B, Abadi P, Ismail-Beigi F. Decreased absorption of calcium, magnesium, zinc, and phosphorus by humans due to increased fibre and phosphorus consumption as wheat bread. *J Nutr* 1976;106:493-503.

³ Jenkins DJA, Wolever TMS, Nineham R, et al. Guar crispbread in the diabetic diet. *Br Med J* 1978;ii:1744-6.

⁴ Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. *Am J Med* 1977;62:707-14.

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The occupational therapist and the patient with Parkinson's disease

Levodopa has transformed the management of patients with Parkinson's disease, but drug treatment is not wholly effective and leaves many patients with substantial disability. The place of physiotherapy in improving function in Parkinson's disease is undisputed, but there is little guidance on the role of occupational therapy.¹⁻³ This study was undertaken to establish whether assessment at home by an occupational therapist and the provision of simple aids could assist in the management of patients with Parkinson's disease.

Patients, methods, and results

Patients were contacted through the local branch of the Parkinson's Disease Society and through neurologists at the Glasgow Institute of Neurological Sciences and asked whether they wanted a visit from an occupational therapist. After the general practitioner's permission had been obtained, they were visited and a detailed account of their daily activities compiled. Their need for aids was assessed, and any needed but not already provided were supplied free by the branch. Aids supplied were costed at 1979 prices. Patients were visited again six to 18 months later, their condition reassessed, the usefulness of the aids inquired into, and any additional aids needed supplied.

Eighty-one patients (40 women and 41 men) were seen; 68 were over 60 years of age; 66 were under the care of consultants; and 29 had been seen by an occupational therapist at some time in the past. Twenty-three were considered not disabled, 20 disabled (incapable of living at home without help, but not needing help for self-care), and 37 dependent (needing help

with self-care).⁴ Impaired mobility was the main reason for disability and difficulty with feeding or dressing for dependence. The frequency of dependence increased with age, 33 of the dependent patients being over 60 years.

The table shows the aids considered necessary and those supplied as a result of the assessment; the difference represents aids already provided. Aids to bathing were most often considered necessary, and most of these and virtually all the feeding aids had to be supplied. Toilet aids were needed by a few patients, and many miscellaneous aids were supplied to a few patients. Bath aids were considered useful by virtually all of those to whom they were supplied, as were the feeding aids except for the suction egg cup.

Aids needed and supplied

	Aids needed	No (%) of aids supplied after assessment	No* (%) useful	Cost per item (£)
Bath aids:				
Rail	46	31 (67)	28/29 (97)	4.25
Mat	38	23 (61)	21/21 (100)	1.60
Board	14	5 (36)	3/3 (100)	6.25
Seat	21	4 (19)	4/4 (100)	5.40
Stool	1	0		
Feeding aids:				
Manoy plate	22	20 (91)	10/15 (67)	Large 1.90, small 1.30
Manoy cup	10	8 (80)	4/5 (80)	0.96
Sunflower knife	22	21 (95)	13/15 (87)	1.95
Sunflower fork	17	17 (100)	13/15 (87)	1.17
Sunflower spoon	15	14 (93)	11/13 (85)	1.90
Dycem mat	33	33 (100)	23/25 (92)	1.40 or 2.20
Suction egg cup	15	15 (100)	6/14 (43)	0.19
Other	3	2		
Toilet aids:				
Rail	5	2		15.60
Seat	5	3		4.62
Grab rail	8	3		1.30
Commode	1	0		

*Denominators are numbers of patients followed up.

Miscellaneous aids were: Monkey pole 2, rope ladder 4, electric can opener 3, jar opener 3, tap turner 1, trolley 1, telephone amplifier 1, door bell 2, ramp 1, wheel-chair 2, shower 1, Zimmer frame 1, bridge card holder 1, shoe horn 1, helping hand 1, chair raise 2, ejector seat 1, high back chair 1, incontinence pads 1 (20 out of 30 had to be supplied).

The non-disabled patients often required bath aids (they were thought to need 25 items), but few needed feeding aids (7 items); most of these were supplied. Disabled patients required bath aids more often (32 items), but most had already been provided; they required feeding aids infrequently (15 items). The dependent patients required bath aids even more often (63 items), and half of these were supplied as a result of the survey. Nearly half the dependent patients needed feeding aids (106 items), and almost all of these were supplied; two-thirds of the other aids needed were also supplied after the survey. The mean cost of aids needed was £23.10 per patient.

Sixty two patients were followed up after an average of 10 months. Of the remainder, nine had died, five were in hospital or other institutions, and five had been seen less than six months previously. Nine of the 62 had changed their category of disability: three of those initially not disabled had become disabled and five dependent. Six of the nine required further aids not considered necessary at the first visit.

Comment

Several of the patients approached thought that their state did not warrant a visit from an occupational therapist, so the group studied was probably biased towards the more disabled. Despite presumably optimal drug treatment, three-quarters were disabled or dependent. Our findings suggest that this group of patients is considerably underprovided with simple aids to daily living, despite the fact that a third had already seen an occupational therapist. Only seven of the 29 patients already seen by an occupational therapist had had all their needs provided. The greater prior provision of bath aids to the disabled than to the dependent patients, despite the latter's greater needs, and the number of feeding aids supplied to the dependent, suggest that previous assessment of needs for aids may not have been totally realistic, though some of the feeding aids (the Sunflower cutlery) only became available in early 1978.

The high proportion of aids supplied that were considered valuable by their users confirms their practical usefulness. The cost of the aids supplied was small, and on average equalled the cost of 26 days' treatment with levodopa.

The proportion of patients who developed disability or dependence in the short follow-up, and with it a need for further aids, shows that assessment must be repeated if the patient's abilities deteriorate. Our findings thus indicate that all patients with Parkinson's disease who have any substantial degree of disability should be assessed,

preferably at home, by an occupational therapist as part of their routine management.

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¹ Davis JC. Team management of Parkinson's disease. *Amer J Occup Therap* 1977;31:300-8.

² Macdonald EM. *Occupational therapy in rehabilitation*. 4th ed. London: Bailliere Tindall, 1976.

³ Shopland A, Hardial P, Unwin A, Vickers S, Westmore V, Williams J. *Occupational therapy*. 2nd ed. Edinburgh: Churchill Livingstone, 1979.

⁴ Akhtar AJ, Broe GA, Crombie A, McLean WMR, Andrews GR, Caird FI. Disability and dependence in the elderly at home. *Age Ageing* 1973;2:102-10.

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Costs of unnecessary tests: nonsense urines

Sandler¹ investigated the cost of unnecessary laboratory tests and reported that the incidence of abnormal results on routine urine examinations was 2 to 3%. He considered that routine urine examinations were of minimal value. In this laboratory, after examining a series of urine specimens, we concluded that only exceptionally did urine samples that were crystal clear contain significant numbers of either cells or bacteria. We therefore ceased to examine them bacteriologically unless clinical reasons for doing so were given on the request form.

Analysis of our urine reports suggested that there were some specimens from which bacteria might have been expected to grow but which were sterile on culture. After the obvious laboratory causes, such as inadequacies of culture media, had been excluded, we looked at the possible presence of undisclosed inhibitory substances (antibiotics) in the specimens.

Methods and results

Two hundred and four urine specimens, each of which was accompanied by a request form which indicated either by a positive statement or by the absence of information that antibiotics were not being administered at the time of collection, were examined for the presence of antibiotics. Antibiotics were present in 21% of samples (see table). These results led us to examine similarly 92 urine samples from patients alleged on the request form to be receiving antibiotics, with the results shown in the table.

Results of testing urine specimens for antibiotics in patients supposedly not taking them and in patients supposedly taking them

	Patients not taking antibiotics		Patients taking antibiotics	
	No of specimens	No (%) positive for antibiotics	No of specimens	No (%) negative for antibiotics
District general hospitals	114	32 (28)	49	3 (6)
Other hospitals	20	1 (5)	11	4 (36)
General practice	70	9 (13)	32	12 (38)
Total	204	42 (21)	92	19 (21)

Comment

It is disturbing that a fifth of patients said not to be receiving antibiotics were excreting them in the urine and that a fifth of those allegedly taking antibiotics were not excreting them in the urine. Of the several possible explanations for these results, the most likely are that antibiotics prescribed for non-urinary conditions were not recorded on the request form and that some patients were not taking the antibiotics prescribed for them.