"High Liquid Glucose" for Oral Use in Renal Failure.* (31303)

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Patients with acute or chronic renal failure are in a state of "hypercatabolism" in that the rate of protein breakdown exceeds protein intake. In such patients, it is desirable to minimize the rate of protein metabolism because this will delay progression of uremia and reduce the incidence of hemodialysis. Many authors advocate the use of low-protein, high-carbohydrate diet to achieve this (1-4). Evidence of the protein-sparing activity of a high carbohydrate intake in normal individuals as well as patients with severe renal disease has been presented(5). This effect is partially due to reduced protein breakdown(6). However, Thaysen(7) found that although dextrose has a very definite protein-sparing effect in patients with a low metabolic rate, it has little or no effect in the hypercatabolic state.

In 1963, Dr. F. M. Parsons(8) of the Renal Research Unit at Leeds General Infirmary developed a high-carbohydrate drink based on liquid glucose BPC‡ for dietary management of patients with renal failure. The ingredients of the drink were demineralized liquid glucose, citric acid monohydrate, benzoate, flavoring and coloring, and demineralized and carbonated water (Table I). A flavored concentrate is prepared from all but the carbonated water which is added just before sealing the bottles. The drink is presented to the patient as a carbonated beverage in 6½ oz. bottles so that 4 such bottles contain 1700 calories. Analysis of the electrolyte content of the drink as prepared shows that sodium had the highest concentration—6 mEq/liter. The potassium content was less than 3 mEq/liter.

TABLE I. Ingredients of "High Liquid Glucose."

	ml
Demineralized liquid glucose*	390
Citric acid monohydrate—50% w/v solution	10.3
Sodium benzoate—20% w/v solution	.5
Flavoring and coloring	2.0
Water	114
Carbonated water (containing 3-4 vol of carbon dioxide)	187
Total volume	703.8

* Equivalent to 454 g dextrose and 1700 calories.

The present paper reports the effect of "high liquid glucose" (HLG) drink in addition to the anuric diet in anephric patients as well as individuals in acute renal failure. Rate of urea increase, blood levels of glucose, fecal nitrogen excretion and undesirable side effects were noted before and during the use of HLG.

Material. "High liquid glucose" as a form of high carbohydrate oral intake was used in 6 patients with renal failure. Four patients with irreversible renal disease had undergone bilateral nephrectomy and were on the waiting list for cadaver kidney transplantation. The other 2 patients had acute renal failure. This had developed in one following cholecystostomy for a gangrenous gallbladder with peritonitis. The other patient had acute renal failure of obscure etiology. The uremic state of 6 patients was controlled by periodic hemodialysis.

During the first observations on these patients they were on a low-potassium, restricted protein diet which was otherwise not controlled. Subsequently these patients were placed on a control anuric diet for a period of 2 weeks during which time they were given approximately 1500 calories per day. The diet consisted of 30 g of protein, 1 g each of sodium and potassium chloride, and a fluid intake of 600 ml. The remainder of the calories in the diet were made up by fat intake of 50 g and carbohydrate of 250 g. This diet was prepared by the Dietetic Department of University of Minnesota Hospitals. After the

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‡ BPC—British Pharmaceutical Codex; drink prepared by Beecham Food and Drink Division, England.

control period, daily caloric intake was increased to approximately 2200 to 2500 calories by supplementing the original diet with 300 to 400 ml of "high liquid glucose" for the next 2 weeks. The high liquid glucose was at first offered on a P.R.N. basis so long as at least a 300 ml intake in 24 hours was obtained. Because of the extremely sweet taste, patients often would not accept the drink alone, and therefore, it was given routinely with meals plus any additional amounts that they would take to obtain a 300 ml intake per day.

Daily blood urea nitrogen (BUN), serum creatinine, serum osmolality and urine osmolality were measured in all cases. In the chronic renal failure patients, fecal nitrogen excretion and fasting blood sugar were estimated daily also. The development of gastrointestinal symptoms and the palatability of the drink were evaluated by frequent, careful questioning of the patients.

Results. In the 4 patients with chronic renal failure, the rate of increase of blood urea nitrogen prior to instituting the control anuric diet or the use of HLG averaged 20 to 30 mg%/24 hours. Consequently, they needed frequent hemodialysis (every 2 to 3 days). When these patients were on the anuric control diet, the rate of daily blood urea nitrogen increase was still in the range of 15 to 25 mg% per day and the incidence of hemodialysis remained about the same. When "high liquid glucose" was given, in combination with the anuric diet, the rate of increase of the blood urea nitrogen fell to 5.5 to 17.5 mg% per day (Table II). Hemodialysis in these patients became necessary only every 6 to 7 days. In one patient, M. F., in acute renal failure, diuresis began 3 days after he was started on HLG. In 7 days his BUN went from 112 to 19 mg%.

There was only a very mild increase in the daily fasting blood sugar values and the highest value obtained was 133 mg% (Table II). The rate of increase in serum creatinine and osmolalities did not show any appreciable change on any diet. Fecal nitrogen fell to or remained at negligible values (Table II). Similar results were seen in the patients with

TABLE II. BUN, Blood Glucose, Fecal Nitrogen, Average Caloric and Protein Intake, and Weights of 6 Patients in Renal Failure.

Patient	Avg BUN rise/24 hr, mg %		Highest blood glucose, mg %		Fecal N, g/24 hr		Avg calories/24 hr		Avg protein intake/24 hr, % 30 g		Avg wt, kg	
	No HLG*	HLG	No HLG	HLG	No HLG	HLG	No HLG	HLG	No HLG	HLG	No HLG	HLG
B.B.	23.4	9.7	134	132			650	1650	45	30	59.3	51.7
H.G.	17.2	5.5	125	132	.5	.1	1500	2250	50	50	53.4	52.2
D.L.	22.6	11.6	123	133	1.2	.7	688	1767	25	25	55.1	56.4
H.V.	26.2	17.5	83	105	.8	.9	1300	2250	100	100	37.5	35.8
M.F.†	31.3	See text	110	110	.15	.1	1500	2500	100	100	93.4	88.6
P.J.†	30.5	15.4	93	105			500	2000	75	75	72.2	71.1

* HLG = "High liquid glucose" drink.

† Acute renal failure.

acute renal failure. No glycosuria was seen in these 2 patients.

The drink was available in 6 flavors: black currant, lemon, pineapple, orange, raspberry and lime. The majority of the patients preferred the lemon, lime and orange flavors. While 4 of the patients thought the drink was palatable, 2 complained of excess sweetness. Diluting the drink made it more tasteful, but defeated the purpose of low-volume intake.

Two (B.B. and H.G.) of the 6 patients had severe diarrhea and mucous discharge per rectum with no abdominal cramps during administration of "high liquid glucose." In both diarrhea was uncontrollable despite large doses of constipating agents and the HLG had to be discontinued in B.B. in 3 days, in H.G. after one week. One patient had a mild diarrhea which was controlled with Kaopectate, and the other 3 patients did not experience any gastrointestinal symptoms. The development of diarrhea may be due to the increased osmolality within the gastrointestinal tract.

Comment. From this preliminary study, it appears that an increased ingestion of carbohydrate which roughly doubles the basal intake of daily calories results in a decreased rate of urea production in patients with renal failure. The fact that the anephric patient receiving HLG had negligible fecal nitrogen would indicate that this less severe uremia occurs because of a decrease in protein catabolism. Since the protein intake of all of these patients except B.B. was the same when they were not taking the HLG as when they were, the increased carbohydrate intake must act by reducing protein catabolism. This is of benefit to the patient not only because of the protein-sparing, but also because it reduces the number of dialyses needed. This lessens the risks to the patient and improves the financial and personnel problems associated with the care of these patients.

The increase in caloric intake can be obtained without substantial increase in fluid intake, an ideal situation for a patient with acute or chronic renal failure. Another advantage of HLG for the patient with renal failure is the negligible electrolyte content and the extremely low microbial colony counts of the drink(9). These particular attributes were not evaluated in this study. A troublesome feature of the drink is the diarrhea which may accompany its use.

Conclusion. "High liquid glucose" drink, if tolerated by a patient in renal failure, is an excellent agent for increasing carbohydrate and caloric intake. This decreases the rate of rise of BUN by decreasing protein catabolism and thus reduces the incidence of hemodialysis.

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1. Bennett, T. I., Dodds, E. C., Lancet, 1924, v208, 429.

2. Borst, J. G. G., ibid., 1948, v254, 824.

3. Merrill, J. P., Treatment of Renal Failure: Therapeutic Principles on the Management of Acute and Chronic Uremia, Grune & Stratton, New York, 1955, p238.

4. Gjrup, S., Act. Med. Scand., 1958, v161, 233.

5. Wolthius, F. H., ibid., Suppl., 1961, v373, 1.

6. Blagg, C. R., Parsons, F. M., Young, G. A., Lancet, 1962, v1, 608.

7. Thaysen, J. H., Anabolic Steroids and Treatment of Renal Failure. Symposium on Protein Metabolism: Influence of Growth Hormone, Anabolic Steroids and Nutrition in Health and Disease, Leyden, June 25-29, 1962, Springer, Berlin, 1962, p450.

8. Parsons, F. M., Fore, H., Lancet, 1962, v2, 386.

9. Allen, R. J. C., personal communication.

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