TABLE II. Effect of Intravenous Infusion of One Liter of 10% Fructose on the Level of Whole Blood Fructose.

Patient Age (yr) Sex Wt (kg)	Rate of infusion (g/kg/hr)	Whole blood fructose levels (mg%) Initial	Middle	Final
1. G.T. 50 M. 64	1	0	41	6.5†
	2	0	102	14 †
2. C.W. 62 M. 64	1	0	61	2.7†
	2	0	105	5.6†
3. H.T. 62 M. 53	1	0	123	8.7†
4. F.M. 64 M. 62	.8	0	37	—
5. S.R. 67 F. 44	1.2	0	49	56 *
6. B.V. 60 F. 61	1.2	0	33	0 ‡
7. J.D. 46 M. 57	.8	0	38	65 *
8. N.A. 17 M. 63	.7	0	41	12 †
9. E.H. 50 F. 79	.7	0	46	11 †
10. H.E. 40 F. 45	1.4	0	76	40 *

* 30 min. post infusion.
† 60 min. post infusion.
‡ 120 min. post infusion.

high degrees of glycosuria which followed the glucose injection (Table I). The amount of sugar lost in the latter case was sometimes as much as 25% of that infused, whereas the maximum loss of fructose was 5.8%. The degree of diuresis which accompanied the glucose infusions was not seen following the fructose infusions. Of interest are the findings in the diabetic patient, No. 9. Also of interest are the findings in the patients tested during the immediate postoperative period, who showed a diminution of glucose tolerance.

Discussion. It seems apparent from the present findings that fructose when given intravenously to the human subject, even at rates as rapid as 2 g/kg/hr. leaves the circulation promptly. It does not appear in the urine and is presumably converted just as promptly into glycogen. These findings suggest that fructose is a much better sugar for intravenous feeding than glucose. Not only is less sugar and water lost in the urine but it is likely that the increases of hepatic glycogen which are undoubtedly obtained, have important practical and theoretical implications. For example, the ability of the liver to overcome harmful agents or stresses is undoubtedly increased thereby.

Summary. In 10 experiments on human subjects comparative intravenous injections of 10% glucose and 10% fructose were carried out. Even at rapid rates of infusion very little fructose appeared in the urine as compared to the amount of glucose. Diuresis was also less pronounced with fructose. Moreover, the fructose infusions were not followed by either hyperglycemia nor by an excessive rise in the blood fructose level, thus indicating a more rapid utilization of fructose and presumably more rapid glycogenesis.

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Role of the Thyroid Gland in Urinary Pentose Excretion in the Rat.* (18356)

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Coover, Feinberg, and Roe(1) found that the excretion of urinary pentose in rats was markedly increased by keeping the animals in

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1. Coover, M. O., Feinberg, L. J., and Roe, J. H., PROC. SOC. EXP. BIOL. AND MED., 1950, v74, 146.

TABLE I. Effect of Thiouracil Feeding on Urinary Excretion of Pentose. Data are in mg per 24 hr as arabinose.

Rat No.	Basal diet daily avg for 3 day period	Basal diet + 2% thiouracil. At room temp. (20°-30°C). Days 1	2	3	4	5	6	7	Basal diet only. Days 8	9
1	2.56	.74	.77	.15	.63	.77	.0	.0	1.41	1.89
2	2.48	.70	.84	.46	1.30	1.24	.97	1.95	.67	2.95
3	1.45	1.61	1.44	.55	.15	.95	.95	1.27	1.48	2.11
4	2.90	1.61	.63	.31	.70	.95	.62	.55	1.61	2.00
5	2.74	2.44	.18	.80	1.42	.15	.64	1.06	2.13	3.00
6	2.69	.23	.15	.08	.60	1.19	.81	.27	1.22	3.00
7	2.53	1.11	.78	.46	.80	.46	.75	.02	1.15	2.00
8	2.93	1.24	2.67	2.46	1.52	.93	.23	.30	.77	3.02
		Basal diet + 2% thiouracil at −1°C								
9	2.06	1.97	1.94	1.99	2.01	died				
10	1.95	.0	.0	died						
11	1.87	.24	died							
12	2.17	.14	died							
13	2.28	.0	died							
14	1.74	.08	.02	.0	died					

a cold room at −1°C and by thyroid feeding. It was suggested that the accelerated excretion of urinary pentose in the cold is due to increased metabolism brought about by greater thyroid activity.

Further evidence of the part played by the thyroid in the excretion of urinary pentose has been obtained by experiments in which thiouracil was fed to rats. The data are shown in Table I. Rats kept at room temperatures and placed on an adequate synthetic diet to which 2% of thiouracil was added showed a highly significant decrease in urinary pentose excretion ($P<0.01$). In some instances the daily urinary pentose excretion was less than the amount determinable by the method used (2) and is recorded as zero excretion. These observations appear to indicate that the thyroid gland has a regulatory influence upon the excretion of urinary pentose.

In the experiment of Table I in which rats were placed in the cold room at −1°C and fed a thiouracil-containing diet, the urinary excretion of pentose was markedly decreased, and the animals survived poorly. Of the 6 rats placed in the cold room, 3 died on the second day, and one died on the third, fourth and fifth days. Thus animals fed thiouracil cannot compensate sufficiently to survive at −1°C. This observation is further evidence for the assumption that the compensatory adjustment of animals to cold occurs through activity of the thyroid gland.

Summary. It has been observed in rats that thyroid feeding markedly increases the urinary excretion of pentose, and thiouracil feeding greatly reduces the output of urinary pentose. Animals on a diet containing thiouracil also survive poorly in the cold. These observations offer strong evidence for the assumption that in the rat the thyroid gland has a dominating role in the production of urinary pentose and that adjustment of animals to cold takes place, at least in part, through activity of the thyroid gland.

2. Roe, J. H., and Rice, E. W., *J. Biol. Chem.*, 1948, v173, 507

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