TABLE I. Assay of Anti-lymphosarcoma Sera by Electrophoretic Method and Neutralization Test.

Antiserum No.	Neutralization test: Serum dilution 1/2	1/4	1/8	1/16	1/32	1/64	Electrophoretic titer
Normal*	19/30	16/30	17/30	26/30	30/30		<50
4	2/6†	0/6	2/6	6/6	5/6	—	50
6	0/6	0/6	0/6	3/6	6/6	—	100
5b	0/6	0/6	0/6	0/6	4/6	—	200, 210
10	0/6	0/6	0/6	3/6	4/6	4/6	250
9	0/6	1/4	0/6	0/6	1/4	4/6	335, 335

* Summary of assay of 5 normal rabbit sera.

† $\frac{\text{No. of tumor takes}}{\text{No. of sites inoculated}}$.

be estimated from accurate determinations of the mobility of a few different dilutions in the neighborhood of the endpoint.

The available antisera which had been prepared against whole cells or whole cell homogenates were assayed. Table I summarizes the promising correlation between the "electrophoretic titer" and animal protection as demonstrated by the neutralization test(5).

It is obvious from the results of the two completely independent assays of antiserum No. 5b and antiserum No. 9 as well as from consideration of the titration curve that the electrophoretic titration is considerably more precise than our original criterion of acceptability.

Summary. 1. An electrophoretic technic is described which permits the assay of antisera for their power to combine with the cell surface, as distinguished from any reaction subsequent to combination. 2. This technic has been applied to cells of the Gardner lymphosarcoma 6-C3H-ED. 3. Preliminary studies with regard to the correlation of the electrophoretic titer and animal protection are promising.

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Peripheral Assimilation of Fructose in Man.* (20762)

THEODORE B. VAN ITALLIE, MARY C. MORGAN, RICHARD T. CATHCART,† GILLES G. LEDUC, AND LOUIS B. DOTTI.

From the Department of Medicine, St. Luke's Hospital, New York City.

Uptake of fructose by hepatic cells has been demonstrated by a number of investigators(1-3). On the other hand, the ability of extrahepatic tissue, and specifically of muscle, to utilize fructose as such has been

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† Present address: Jefferson Medical College, Philadelphia, Pa.

questioned(4). Other evidence suggests that fructose is utilized directly by muscle(5-7). To permit further study of the ability of peripheral tissues to assimilate fructose, successive peripheral arteriovenous fructose differences were measured in 11 human subjects during intravenous administration of fructose solutions.

Methods. Eleven adult patients without known metabolic disorders served as subjects. All subjects were studied while they were at rest and in the postabsorptive state. In 9 subjects venous blood samples were obtained with a minimum of stasis from an antecubital vein, and almost simultaneously, capillary (arterial(8)) blood was collected by direct pipetting from a finger tip of the same extremity after cutaneous puncture. In 2 subjects arterial blood samples were obtained by means of a Cournand needle placed in a femoral artery, and venous blood was obtained through a cardiac-type catheter. The catheter was inserted in the basilic vein and manipulated under fluoroscopy so that its tip was in the distal portion of the subclavian vein. Roe's method(9) was used to measure fructose in whole blood and was found to be accurate within ± 1.0 mg %. A Bowman constant infusion pump was used to deliver fructose into an arm vein at a constant controlled rate. First, 200 to 350 ml of 10% fructose in water solution was administered rapidly as a priming dose, and then constant fructose infusion was begun. The fructose always was injected at a rate of 0.58 g per minute. Blood samples were obtained at 15 ± 3 minute intervals (periods I-IV) for one hour beginning 30 to 100 minutes after start of fructose administration. One subject (E.G.) was given 875 ml of 10% fructose intravenously over an 80-minute period at 1.1 g per minute, and arterial and venous samples were obtained at 20-minute intervals during and after the infusion.

Results. Throughout the 4 periods, capillary (arterial) and venous fructose levels during constant fructose infusion remained virtually constant in individual subjects. For the 10 subjects who were given fructose at 0.58 g per minute, the average capillary fructose level for period I was 68.9 mg % (range

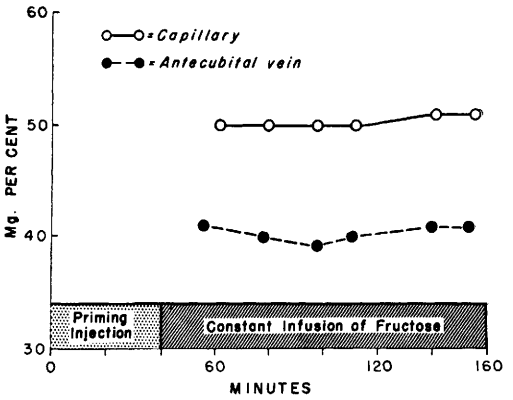

FIG. 1. Maintenance of constant capillary (arterial) fructose level and uniform capillary-venous fructose differences during constant, slow fructose infusion (0.58 g/min.).

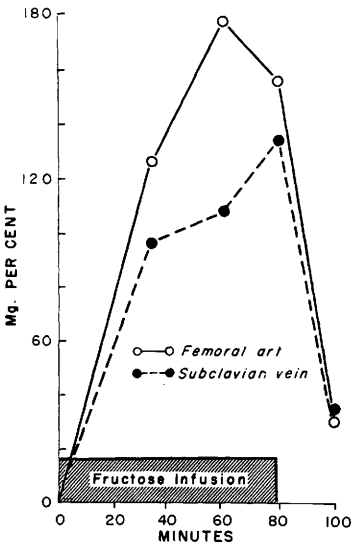

FIG. 2. Changing arterial fructose level and variable fructose arteriovenous differences during and after rapid fructose infusion (1.1 g/min.).

40-100); for period II, 67.6 mg % (range 39-98); for period III, 67.1 mg % (range 39-98); and for period IV, 66.1 mg % (range 37-88).

The average venous fructose level for period I was 57.3 mg % (range 30-78); for period II. 54.2 mg % (range 29-78); for period III, 55.5 mg % (range 26-78); and for period IV, 55.6 mg % (range 26-75).

The average arteriovenous fructose difference for period I was 11.6 ± 5.5 mg %; for period II, 13.4 ± 3.9 mg %; for period III, 11.7 ± 4.0 mg %; and for period IV, 10.6

± 3.1 mg %. A representative arteriovenous fructose curve during constant fructose infusion is shown in Fig. 1.

In patient E.G. (Fig. 2), arterial and venous fructose levels rose rapidly and fell precipitously after termination of the infusion. At one point, when the infusion was three-quarters completed, an arteriovenous fructose difference of 70 mg % was observed. However, at the end of the infusion this difference had decreased to 22 mg %. Twenty minutes after the end of the infusion the arterial fructose level had fallen to 32 mg % and the arteriovenous fructose difference was —4 mg %.

Discussion. In order to distinguish between peripheral arteriovenous differences due to simple diffusion into the interstitial space and those associated with tissue uptake it was found necessary to perform serial determinations of arterial and antecubital or subclavian venous fructose levels in subjects who were receiving fructose by constant slow infusion following administration of a priming dose. In this way it was possible to keep the arterial fructose level constant. An example of the difficulty involved in interpreting arteriovenous differences associated with a rapidly changing arterial fructose level is shown in Fig. 2, where the venous fructose level continues to rise even while the arterial level is falling off slightly.

It was believed that maintenance of appreciable fructose arteriovenous differences in the presence of constant arterial fructose levels could be regarded as good evidence for direct assimilation of the fructose by peripheral cells. Had the fructose arteriovenous differences quickly become negligible, or had the arterial and venous values for fructose shown a consistent trend in the direction of equilibrium(10), the findings could have been interpreted as evidence of simple diffusion rather than assimilation of the sugar. However, the results show that the arteriovenous fructose differences were well maintained in time.

Although these findings indicate that fructose enters peripheral cells, proof of utilization of this hexose by such cells would require technics other than those employed in this study.

Summary. 1. Serial peripheral arteriovenous fructose differences were measured in 10 human subjects receiving a constant intravenous infusion of 10% fructose in water, following an initial priming injection. Maintenance of consistent and appreciable arteriovenous differences under these circumstances occurred in every case, and this finding is interpreted as evidence for direct assimilation of fructose by the peripheral tissues. 2. The problem of distinguishing between diffusion, assimilation and utilization is discussed.

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