TABLE I. Melanophoric Indices Observed in Isolated Tadpole Tails; 35 Min. of Immersion (Avg of 2 Readings per Tail).

Conc., units/ml	Standard	Unknown
.08	3.75	3.50
	4.00	3.25
	4.00	4.00
	4.00	2.75
	4.00	3.50
	$S_H = 19.75$	$U_H = 17.00$
.018	3.50	2.00
	3.25	2.00
	2.75	1.75
	3.00	2.75
	3.00	1.50
	$S_M = 15.50$	$U_M = 10.00$
.004	2.25	1.50
	2.25	1.00
	2.00	1.00
	1.50	1.75
	1.00	1.00
	$S_L = 9.00$	$U_L = 6.25$

TABLE II.
Analysis of Variance (Melanophoric Indices).

Variation due to:	df	ms
Samples	1	4.03*
Slope	1	23.11†
Parallelism	1	—
Curvature	2	.26
Error	24	.16

* Highly significant source of variation.
† Very highly significant source of variation.

veloped, using USP corticotropin as the Reference Standard. All ACTH preparations tested showed this activity. Corticotropin B, the most highly purified preparation available, showed the highest degree of melanophore-expanding activity and was also the most potent in causing adrenal ascorbic acid depletion.

TABLE III. Melanophoric Potencies of 5 Corticotropin Preparations (Units/mg).

Sample	Melanophoric potency	Ascorbic acid depletion potency
I	9 ± 5	5
II	58 ± 12	160
III	58 ± 12	60
IV	21 ± 5	60
V	10 ± 3	5
VI*	269 ± 48	163 ± 141

* Sample of Corticotropin-B.

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Received July 31, 1953. P.S.E.B.M., 1953, v84.

Effect of Adrenal Cortical Extract and Steroids upon Glucose Tolerance of Eviscerated Rats. (20536)

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The data of this study support the conclusions of an earlier report(1) that adrenal cortical extract inhibits the tolerance of the eviscerated rat for intravenously administered glucose. Cortisone and hydrocortisone are less effective.

Methods. General details have been published(1,2). Male rats were eviscerated at 250 ± 2 g weight. Each rat received by continuous intravenous injection 20 cc per 24 hours of a solution containing glucose, 4 units of regular insulin, 10,000 units of penicillin

TABLE I. Effect of Adrenal Cortical Hormones upon Level of Blood Glucose in Eviscerated Rat. Averages and standard errors.

Exp.	Duration, hr	Cortical hormone	Dose, 24 hr	Glucose load, mg/100/hr	No. rats	Blood glucose, mg %
1	24	Controls	0	40	12	91 ± 9.8
	24	ACE	20 cc	40	12	141 ± 16.4*
	24	Controls	0	44	12	86 ± 12.9
	24	ACE	20 "	44	12	168 ± 29.0*
	24	Controls	0	48	12	164 ± 20.1
	24	ACE	20 "	48	12	251 ± 30.1*
2	24	Controls	0	44	52	110 ± 5.8
	24	Cortisone	4 mg	44	52	127 ± 8.0
	24	Hydrocortisone	4 "	44	52	128 ± 8.2
3	48	Controls	0	44	35	187 ± 15.6
	48	Cortisone	4 "	44	36	191 ± 15.0
	48	Hydrocortisone	4 "	44	35	223 ± 16.1
4	24	Controls	0	44	24	105 ± 8.1
	24	ACE	20 cc	44	24	174 ± 17.2*
	24	Hydrocortisone	2 mg	44	24	109 ± 4.8

* The difference between this avg and that of the corresponding control group meets the usual requirement for statistical significance.

and 5 mg of streptomycin with and without adrenal cortical extract (ACE) or non-esterified steroids. The ACE was derived from mixed hog and beef adrenal glands and represented 20 glycogen units(3) per rat per 24 hours which is the activity equivalent of 2 mg of hydrocortisone. Temperature was constant at 26.5 ± 0.5°C. The glucose load is expressed as mg of glucose per 100 g of rat per hour (mg/100/hr). The level of blood glucose was determined(4) on jugular vein blood at the end of the experiment.

Experiments and results. The data are in Table I. Comparisons of rats with and without adrenal cortical hormones were made simultaneously in each experimental group. The administration of ACE was associated with a significant elevation of the level of blood glucose above that of the controls (Exp. 1 and 4); each of the 2 steroids failed to cause a significant elevation in the level of blood glucose under these conditions (Exp. 2, 3, and 4).

Discussion. ACE can affect the glucose tolerance of the eviscerated rat given insulin but cortisone and hydrocortisone are either ineffective or relatively weak in this respect. In the non-eviscerated rat the diabetogenicity and the glycogenic potency of 4 mg per day of either steroid is much greater than that of 20 cc of ACE. Does the effect of ACE upon the glucose tolerance of the eviscerated rat represent some physiological function of the cortical hormones? Is this effect due to the presence in ACE of some hormonal principle other than either cortisone or hydrocortisone, or is it due to some non-hormonal contaminant? The results of this study may relate to the observation(5) that adrenal cortical extract modifies the hexokinase reaction, whereas cortisone fails to do so. It has been considered possible that these steroids undergo some bio-conversion in the liver before they become physiologically active, yet, each of these steroids, as well as ACE, has a positive effect upon the ability of the adrenalectomized-eviscerated rat to work(6) and cortisone will raise the level of plasma amino acids in such animals(7). The biological responsiveness of the eviscerated rat to adrenal cortical hormones is, in our experience, sluggish and feeble as compared to that of non-eviscerated rats. The role of liver in adrenal cortical physiology is not clear.

Summary. Eviscerated rats were each given continuous intravenous injections of 20 cc per 24 hours of solutions containing glucose, insulin, and antibiotics with and without adrenal cortical hormones. Adrenal cortical extract caused a significant elevation of blood

glucose above the average value for control animals but cortisone and hydrocortisone were relatively ineffective.

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Received July 31, 1953. P.S.E.B.M., 1953, v84.

Plasma Radioactive Iron Turnover in Acute Viral Hepatitis. (20537)

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The literature contains many references to the hypersideremia of acute hepatitis(1), and in the past year additional papers have appeared(2-6). Histochemical studies of liver biopsy sections have revealed an increased deposition of "stainable-iron" in the liver in acute hepatitis(7-9). None of the studies have to date been concerned with an attempt to locate the responsible metabolic defect. Previously proposed theories attempting to explain the disturbance have been either contrary to accepted theories of iron metabolism, or have lacked any strong evidence in their behalf(1). The aberration in iron metabolism may result from a disturbance in the function of: (a) deposition (storage) of iron in the liver, (b) utilization of iron (red cell production), (c) excretion, or (d) absorption of iron.

In this study, the turnover of intravenous radioactive iron^{59} has been made in 8 patients with acute viral hepatitis. Similar studies in 5 normals, one patient with refractory anemia, and one patient with hemolytic anemia have been included for comparison.

Methods. The studies were carried out in a manner similar to the procedure described by Huff(10,11), except that the iron was made up in 2% sodium citrate. Between one to 6 μc and 5 to 30 μg of iron was used. The procedure was carried out in the morning after a 12-hour fast. Serum iron samples were taken at the time of injection and analyzed for non-radioactive iron(12). Samples of plasma and

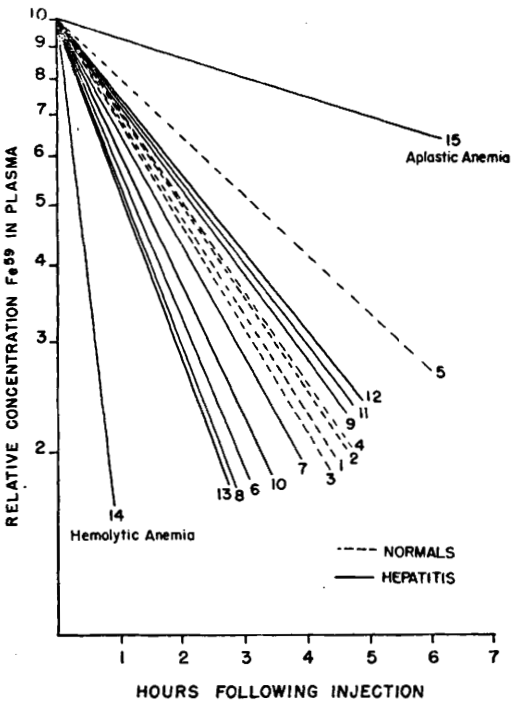

FIG. 1. Disappearance radioactive iron^{59} from plasma following intravenous injection.

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