Summary. (1) In dogs prepared under thiopental and then immobilized with C_{10}, the injection of *d*-amphetamine causes a progressive increase in the frequency of the EEG, with a corresponding decrease in amplitude. These effects are maximal within the first hour. (2) When unanesthetized dogs are gently roused from sleep and then fully alerted, there is a rise in the frequency of the EEG, accompanied by a decrease in amplitude. (3) When *d*-amphetamine is injected into unanesthetized dogs, there is an increase in motor activity, the peak effect being reached within the first hour. There is some evidence for an increase in the frequency of the EEG. (4) These findings suggest that the effects of *d*-amphetamine on the EEG of the dog are parallel to its effect on behavior.

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Hormonal Influences on Liver Lipid Partition, Carbohydrate and Electrolyte Metabolism in the Rat. (20228)

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Among recent evidence indicating there exists an association between potassium and carbohydrate metabolism are the effects of insulin, epinephrine, and the infusion of large quantities of glucose upon the potassium level of plasma or serum. It was reported from this laboratory that following epinephrine and insulin injection in intact rats the plasma potassium level was lowered; this effect on potassium was also found after epinephrine in adrenalectomized or adrenal-demedullated rats but not after insulin(1-3). It was also reported that insulin and epinephrine induced divergent changes in the potassium content of liver and skeletal muscle(4-5). Further elucidation of the mechanism of action of insulin and epinephrine on the potassium content of plasma and tissues was desirable. Since it is increasingly evident that lipid and carbohydrate metabolism are interdependent phenomena, it seemed important to determine the effects of certain induced endocrine conditions upon liver lipid partition. In contrast to the present knowledge of the effects of hormones and their regulatory roles in carbohydrate and protein metabolism, information concerning hormonal influences upon lipid metabolism is limited. Experimental and clinical observations indicate that the endocrine secretions of the pancreas, thyroid, and adrenal cortex affect serum lipid partition(6-10). Evidence has been reviewed(11) which indicates that adrenal cortical homones either directly or through effects upon carbohydrate and protein metabolism have a critical influence upon lipid metabolism. Levin and Farber(12) reported increased total liver fat in the mouse following certain types of stress, but not after others. These investigators concluded that the stress per se did not initiate the "mobilization" of fat to the liver since it was prevented if the increased caloric requirements were met by the administration of glucose. However, Brownell, Hartman and Liu(13) reported experiments in the dog which indicated that there was a distinct hormone secreted by the

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adrenal cortex which influenced the deposition of liver lipids in test mice.

The interrelationships of insulin, epinephrine, and adrenocortical hormones in the regulation of various phases of carbohydrate metabolism are well known. Since the existence of an intimate relationship between carbohydrate and lipid metabolism is increasingly evident(14,15), experiments on the effects of insulin, epinephrine, intravenous glucose administration, and the influence of certain endocrine deficient conditions upon liver lipid partition were performed. At the same time determinations were also made of the liver and plasma water and electrolytes, liver glycogen, and plasma glucose content.

Materials and methods. Male Wistar rats weighing 250-300 g were used. A laboratory chow biscuit and tap water was given *ad libitum* and greens twice weekly. A 1% NaCl solution replaced the tap water for those groups of rats from which the adrenal glands were removed. The animal types used in the experiments reported here were: Intact: Adrenalectomized (5-6 days); and Adrenalectomized-Alloxan injected. In each of these categories there were control and treated groups as described in the experimental sections. *Adrenalectomy* was done by the dorsal-lumbar approach 5-6 days before the predicted time for obtaining tissues from these animals. In order to have groups of rats which were deficient in both adrenal gland secretions and also insulin, advantage was taken of the property of alloxan for selective destruction of pancreatic islet cells. Rats 2-3 days post-adrenalectomy were injected with Alloxan monohydrate (Eastman No. 1722) directly into an exposed saphenous vein at a dose level of 40 mg/kg body weight approximately 65 hours before sacrificing the animals. Epinephrine, insulin, and glucose were injected 60 minutes before tissue samples were to be removed from the designated groups, as follows: Epinephrine (Adrenaline tablets; Parke, Davis & Co.; prepared fresh for use in isotonic saline solution) was injected subcutaneously at a dose level of 0.04 mg/100 g body weight (approx. 0.2 ml); Insulin, (Iletin, regular; Lilly) was injected subcutaneously at a dose level of 0.5 Unit/rat (0.6 ml); the glucose (208 mg as an 8% solution freshly prepared in Sorenson's sodium phosphate buffer 15/M, pH 7.4) was injected slowly into an exposed saphenous vein. All surgical procedures, injections of agents, and the removal of tissues for analyses were done with the rats anesthetized with EVIPAL (n-methylcyclo-hexenyl - methyl barbituric acid). All animals in these experiments were fasted a minimum of 16 hours (overnight). At the designated time of the experiment, cardiac blood was obtained, immediately centrifuged, and aliquots of plasma taken for the determination of sodium, potassium, glucose and water content. The abdomen and chest were opened, the heart incised and allowed to bleed. A specimen of liver for glycogen determination was taken, gently blotted on filter paper, and put in a tared flask charged with 30% KOH. Another specimen of liver (approx. 1 g) was taken for the determinations of water and electrolytes content. The remainder of the liver was dissected free of adherent tissues and taken for the determination of liver lipids (total and fractions). All tissue samples were weighed on a chainomatic balance immediately after removal from *situ*.

The procedures for the determinations of plasma and tissue water and electrolytes were the same as previously described(2). Sodium and potassium were determined with the aid of a duo-optical designed internal lithium standard flame photometer; plasma glucose by the photometric method of Kingsley and Reinhold(16); liver glycogen by the method of Good, Kramer and Somgyi(17) and glucose equivalents after acid hydrolysis by iodometric titration method of Somogyi(18). The liver samples taken for lipid analyses were macerated in a mortar with sand and taken up in 95% ethyl alcohol. Extraction of the liver lipids was accomplished by refluxing 3 times with 95% alcohol and thrice with ether followed by distillation under reduced pressure of the combined extracts. The residue was taken up in petroleum ether (30-60 degrees); washed 3 times with an equal volume of water; then the petroleum ether extract was dried over anhydrous sodium sulfate and made up to a convenient volume. Suitable aliquots

TABLE I. Liver and Plasma Water and Electrolytes, Liver Glycogen, and Plasma Glucose Content in Intact, Adrenalectomized, and Adrenalectomized-Alloxan Groups of Rats.

	Liver constituents (per kg wet tissue)				Plasma constituents			
						K	Na	
Groups	Water, g	K, m.eq.	Na, m.eq.	Glycogen, mg %	Water, %	(m.eq./kg plasma water)		Glucose, mg %
Intact (12)	695±2	96.8±1.7	30.3± .7	20± 3	92.8±.1	5.36±.12	161.0±1.1	72±28
Adrenx (8)	*716±2*	94.0±1.2	28.8±1.2	25±12	93.7±.8	5.78±.19	159.4±2.0	*45± 2*
Adrenx-alloxan (8)	*707±2*	94.1±1.3	*34.8± .7*	*79±32*	92.8±.2	*7.69±.56*	165.4±3.0	75±15

Figures in parentheses are No. of animals in each group. All values are mean ± S.E.
Italicized values in table are significantly different from controls; "P" = <0.05.

of the petroleum ether were taken for the determination of the lipid partition as follows: Total lipids calculated from weight of residue after distillation of petroleum ether and drying to constant weight; lipid phosphorous by a modification of the Youngsburgs' method and the method of Fiske and SubbaRow(19); phospholipids (derived from the lipid phosphorous values x 26); total and ester cholesterol by the method of Schoenheimer and Sperry(20); neutral fat content was derived by calculation of difference.

Results. An established procedure in experiments to elucidate the effects of endocrine products is to study the effects of ablation of the secretory glands upon the biochemical constituents or physiological processes under investigation. Since phenomena observed following the removal of one type of endocrine gland may be the result of either the induced specific glandular deficiency or the unopposed action of the homonal products of another gland(s), it is often desirable in studies of intermediary metabolism to use animals with double glandular ablations. In this experiment the liver lipid partition, electrolytes and other constituents of liver and plasma were determined in intact, adrenalectomized, and adrenalectomized-alloxanized groups of rats.

Exp. I. The mean values of water and electrolytes content of liver and plasma together with the glycogen and glucose content of the respective tissues are presented in Table I. The means in each gland-deficient animal group were tested for statistically significant difference from the intact group. In the adrenalectomized group the liver water content was greater, and the plasma glucose level was significantly lower than the respective means of the intact group. These results are similar to those generally found in this type of animal maintained on a saline solution for drinking purposes. In the adrenalectomized-alloxanized group the means of liver water, sodium, glycogen, and plasma potassium content were found to be significantly greater than the respectives mean values in the intact group. The normo-glycemic level in these animals in association with an increased liver glycogen are noteworthy. Although no definite conclusions regarding the metabolic status in these animals can be made, the data of this group (examined against the results of the other two groups) suggests that the level of carbohydrate utilization was decreased, or that there was an increased formation of glycogen from intermediates of fat metabolism in the absence of insulin(21). The increased plasma K level probably reflects an inefficient utilization of glucose in this group.

In Fig. 1 are presented graphically the mean values of the liver lipid partition in intact, adrenalectomized, and adrenalectomized-alloxanized groups of rats. The means of the latter two groups were tested in turn for statistically significant difference from the respective mean values of the intact group. The influence of the altered hormonal conditions upon liver lipids composition is evident. The total lipid content in the two types of endocrine deficient rats was significantly lower than in the intact rats. Also, the mean total lipid content in the adrenalectomized group was significantly lower than that of the group with the dual glandular ablation. The phospholipid fractions were significantly lower, and the neutral fat fractions were significantly greater in the two

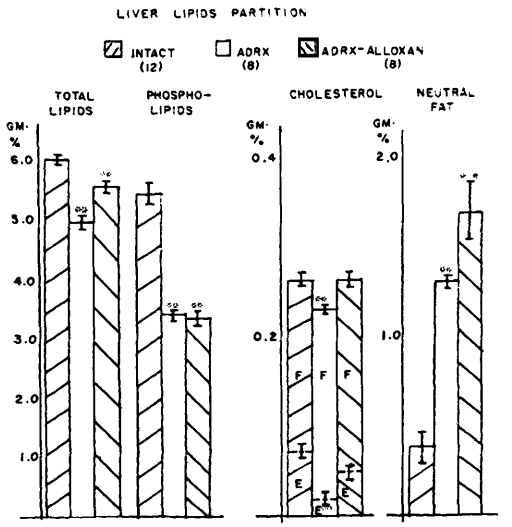

FIG. 1. Starred lipid values are significantly different from controls: *"P"= <0.05; **"P"= <0.01.

groups with endocrine deficiencies than the respective means found in the intact group. However, the neutral fat fraction in the adrenalectomized-alloxanized group was greater than that of the adrenalectomized group. The liver cholesterol fractions (total and ester) of the adrenalectomized animals were lower than the controls. It is noteworthy that the pattern of changes in the liver lipid partition in both the adrenalectomized and the dual-glandular deficient groups were the same except for the cholesterol fractions. These data suggest that the differences in liver lipid partition in the two endocrine deficient groups may be attributed essentially to the imposed condition of adrenal insufficiency and not to the consequence of an unopposed action of insulin. However, that the latter hormone did exert an influence upon liver lipid composition in the adrenalectomized group is indicated by the differences in mean values for total lipids, cholesterol, and neutral fat fractions in these two types of animals.

Exp. II. In this experiment observations were made on the liver lipid partition, electrolytes, and other constituents of liver and plasma in groups of intact rats not treated, and 60 minutes after epinephrine or insulin injection. It had previously been demonstrated that both of these hormones induced a significant decrease in the plasma potassium content of intact rats but divergent effects were found in the potassium content of plasma and other tissues in adrenalectomized and adrenal-enucleated rats following epinephrine and insulin injection(1,3,5). Since the mechanism of action of epinephrine and insulin on tissue potassium content is unsettled, it was decided to observe the concurrent effects of these two hormones upon liver lipid partition, glycogen, electrolytes, and plasma constituents. The 60 minute period after treatment was chosen because of the established evidence that maximum changes in glycemic level occur at this time following the injection of epinephrine or insulin. The mean-results of liver and plasma water, electrolytes, liver glycogen, and plasma glucose 60 minutes after the injection of each of these hormones in intact rats were compared with the respective mean levels in the untreated rats for statistically significant difference by Fisher's "t" test. The data are presented in Table II. The results are in agreement with data previously reported from this laboratory after similar treatment of intact rats; *viz*, a significant fall in plasma potassium, increased plasma sodium content, and the hyper-and hypo-glycemic levels consequent to epinephrine and insulin treatment, respectively. Inspection of these data shows that none of the other constituents of liver and plasma in the treated groups were significantly different from the respective mean values of the untreated intact group.

In Fig. 2 are presented the mean-results of the liver lipid determinations in the groups 60 minutes after epinephrine or insulin injection compared with the respective mean values of the untreated controls. Inspection of the data in Fig 2 shows that following the administration of either hormone the level of the total lipid in the liver was not different from the controls. However, the partition of liver lipids (phospholipids, cholesterol, and neutral fat fractions) were significantly changed in the groups injected with epinephrine or insulin. The greatly increased neutral fat fraction in the two treated groups is particularly noteworthy since this change evi-

TABLE II. Liver and Plasma Water and Electrolytes, Liver Glycogen, and Plasma Glucose Content in Groups of Intact Rats 60 Minutes after Epinephrine and after Insulin.

	Liver constituents (per kg wet tissue)				Plasma constituents			
Groups	Water, g	K, m.eq.	Na, m.eq.	Glycogen, mg %	Water, %	K (m.eq./kg plasma water)	Na (m.eq./kg plasma water)	Glucose, mg %
Not treat. (12)	695±2	96.8±1.7	30.3± .7	20± 3	92.8±.1	5.36±.12	161.0±1.1	72±28
Epinephrine (8)	697±2	97.3±1.3	28.7±1.0	14± 1	92.6±.1	*4.32±.19*	*166.7± .9*	*230±17*
Insulin (6)	698±3	95.2±2.0	30.3±1.6	14± 1	92.8±.2	*4.47±.23*	*166.9± .7*	*44± 5*

Figures in parentheses are No. of animals in each group. All values are mean ± S.E.
Italicized values in table are significantly different from controls; "P" = <0.05.
Epinephrine inj. subcut. at dose level of 0.04 mg/100 g body wt.
Insulin (regular) inj. subcut. at dose of 0.5 unit/rat.

dently was sufficiently great to account for the apparent unaltered total lipid content although the phospholipid fraction was significantly lower in both groups than in the untreated group.

Exp. III. In the 2 experiments presented above the effects of insulin and epinephrine injection, and the influence of certain deficient endocrine conditions upon liver lipid partition as well as other constituents of liver and plasma were determined. Since the hormones injected and the induced endocrine conditions in the groups of the preceding experiments are known to stimulate or adversely affect carbohydrate metabolism, it was decided to observe the effect of glucose administration on the liver lipid partition and the other constituents of liver and of plasma in comparable groups of animal preparations. In this experiment the liver and blood were obtained for the several biochemical determinations 60 minutes after the intravenous injection of 208 mg of glucose in intact, adrenalectomized, and adrenalectomized-alloxinized groups of rats. The mean-results found in these groups compared with the respective means found in similar groups of untreated rats are presented in Table III and Fig. 3. Inspection of the data in Table 3 shows that in the adrenalectomized group the plasma glucose level was significantly greater after the glucose infusion, and in the adrenalectomized-alloxanized group there was a marked hyperglycemia 60 minutes after glucose compared to the mean plasma glucose levels in the respective untreated groups. The liver glycogen content after glucose infusion in the intact and adrenalectomized groups was significantly greater than in their untreated controls; but the glycogen content was not altered in the group with dual-glandular deficiencies after the glucose infusion. The association of the marked hyperglycemia and the unchanged glycogen content in the latter group conform with the expectancy of such results in these animals in view of the classical defects known to exist in carbohydrate assimilation and utilization in the absence of both adrenocortical hormones and insulin. The only significant changes in plasma potassium and sodium concentration observed in these experiments was found in this group. The plasma changes may possibly be the resultant of the influences of a definite diuresis (consequent to the hyperglycemia) which was observed in these animals at the time they were sacrificed. The only

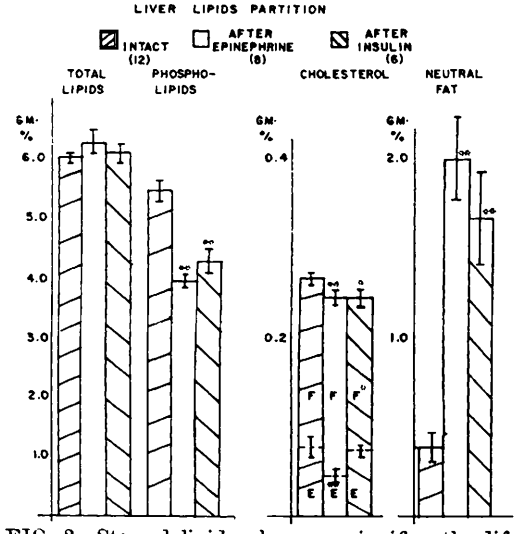

FIG. 2. Starred lipid values are significantly different from controls: *"P" = <0.05; **"P" = <0.01.

TABLE III. Effect on Liver and Plasma-Water and Electrolytes, Liver Glycogen, and Plasma Glucose Content 60 Minutes after Intravenous Glucose Administration in Intact, Adrenalectomized, and Adrenalectomized-Alloxan Groups of Rats.

	Liver constituents (per kg wet tissue)				Plasma constituents			
Groups	Water, g	K, m.eq.	Na, m.eq.	Glycogen, mg %	Water, %	K (m.eq./kg plasma water)	Na (m.eq./kg plasma water)	Glucose, mg %
Intact:								
Not treated (12)	695±2	96.8±1.7	30.3± .7	20± 3	92.8±.1	5.36±.12	161.0±1.1	72±28
Glucose, i.v. (12)	694±2	97.3±1.6	28.3± .6	*347±45*	*93.2±.1*	5.26±.18	165.8± .7	*111± 5*
Adrenx:								
Not treated (8)	716±2	94.0±1.2	28.8± .6	25±12	93.7±.8	5.78±.19	159.4±2.0	45± 2
Glucose, i.v. (8)	*728±3*	95.5±3.3	29.8±1.0	*110±29*	94.4±.2	5.27±.24	157.2±2.3	*109±12*
Adrenx-alloxan:								
Not treated (8)	707±2	94.1±1.3	34.8± .7	79±32	92.8±.2	7.69±.56	165.4±3.0	75±15
Glucose, i.v. (6)	*723±3*	93.3±1.9	*28.7±1.1*	113±21	*93.7±.1*	*5.52±.16*	*151.5±1.1*	*500—*

Figures in parentheses are No. of animals in each group. All values are mean ± S.E.
Italicized values in table are significantly different from controls: "P" = <0.05.
Glucose inj. intrav. (208 mg) 60 min. before tissues taken for analyses.

other notable changes in tissues constituents shown in the data of Table III were the small but statistically significant increased plasma water in the intact group and those with duo-endocrine glands deficiencies, and the increased liver water content in the latter and the adrenalectomized groups.

The liver lipid partition 60 minutes after intravenous glucose administration in intact, adrenalectomized, and adrenalectomized-alloxanized groups of rats compared with the respective mean values in their untreated controls are presented in Fig. 3. Total lipid content was unchanged in intact rats; but the phospholipids, total and ester cholesterol fractions were significantly decreased, and the neutral fat fraction was significantly greater after the glucose infusion. In the adrenalectomized group the only significant changes were increase in total liver lipids and in the neutral fat fraction. In the adrenalectomized-alloxanized group liver total lipids content and the neutral fat fraction were significantly lower, but the phospholipid fraction was significantly greater after glucose infusion than the respective mean values of their untreated controls.

Comment and summary. The experiments presented here represent an attempt to obtain data on the comparative influences of short-term hormonal overdosage or endocrine insufficiencies upon electrolyte, carbohydrate, and lipid metabolism followed concurrently in the animal. The major purpose of these studies was to secure information on the metabolic patterns of the two latter foodstuffs which would enable an evaluation of the endocrine influences upon potassium content and distribution in tissues. It is generally agreed that hormones modify the rates of metabolic reactions. It was our intent in Exp. I (Table I and Fig. 1) to obtain data concerning a number of constituents of liver and plasma in intact, adrenalectomized, and adrenalectomized-alloxanized which would allow a comparative evaluation of the metabolic status of the latter two types of animals with that of the intact rat. The two endocrine-deficient kinds of animals which were used in this comparative study were considered desirable for our purposes since it is well known that certain adrenocortical hormones and insulin have similar resultant influences upon certain phases of carbohydrate metabolism (glycogen formation) and are dissimilar in their resultant influences on other phases of carbohydrate metabolism (glucose oxidation). There is also increasing evidence that adrenocortical hormones and insulin are antagonistic in their resultant influences upon the mobilization of fatty acids to the liver and the mobilization of depot fat. It is apparent therefore that caution must be used in relating the level of tissue constituents to the effects of a specific hormone on intermediary metabolism or the absence of a gland in that the observed effects may be due to "unrestrained" influences

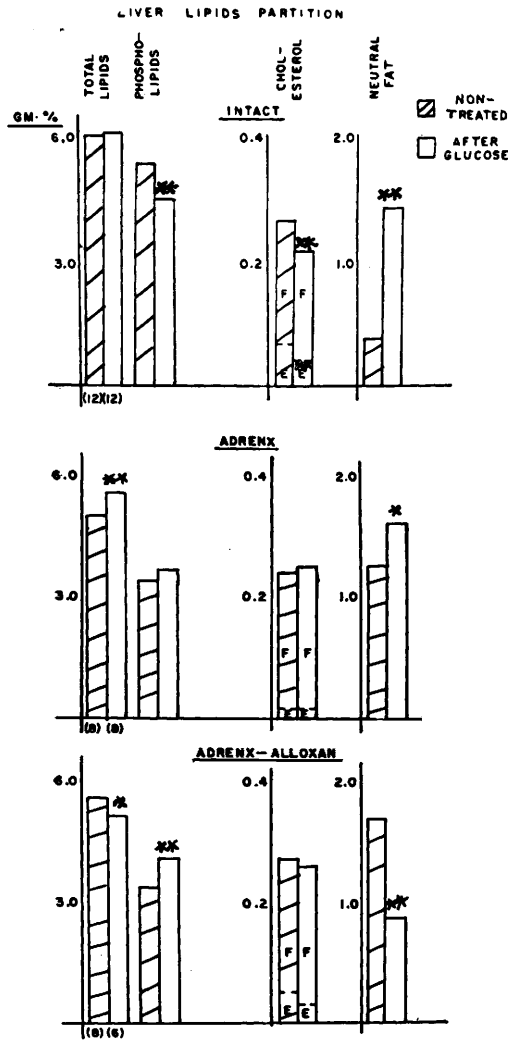

FIG. 3. Starred lipid values are significantly different from controls: * "P" = <0.05; ** "P" = <0.01.

of an antagonistic hormone(s). Examination of the data in Experiment I within this context suggests that in the adrenalectomized rat carbohydrate oxidation, and lipogenesis or neutral fat mobilization was greater than in the intact rat. As stated above, this might be interpreted as the resultant influence of lack of adrenocortical hormones or the unopposed activity of insulin. Parallel inspection of the data of the adrenalectomized-alloxanized group suggests that glucose oxidation in the adrenalectomized group was proceeding under the unopposed action of insulin since in the former group the evidence suggests an amelioration of this metabolic status during the condition of dual-glandular insufficiencies (adrenal and insulin). However, it is equally evident that the status of the metabolism of fat as evidenced by the levels of neutral fat and phospholipid in the liver was not altered in the absence of insulin and adrenal hormones from that found in the absence of adrenal gland secretions alone. Thus the changes in the levels of the various lipids in the two groups from those found in the controls of Experiment I must be due mainly to the absence of the hormonal influences of the adrenal gland. A more detailed interpretation of the liver lipid changes must necessarily be tentative in view of the uncertainties regarding the metabolic interrelationships of the lipid fractions in the liver. It has been reported that in the depancreatized dog there is a marked increase in the turnover of liver phospholipids when exogenous insulin is withdrawn(22). Since the liver phospholipids probably are not involved in fat transport(23), the increased turnover suggested that some of the liver phospholipids participate in fat catabolism. In the present experiments the decrease in liver phospholipids observed in the animals with glandular deficiencies suggests an elevated level of fat metabolism. The increase in neutral fat could be due either to mobilization of fat from depots or an increased rate of synthesis in response to the elevated level of fat metabolism.

Information concerning the effect of epinephrine on lipid metabolism is very meagre. Cori and Cori(24) deduced from their data that fat is the fuel for the increased metabolism due to epinephrine. An increase in liver phospholipids and fatty acids 2-3 hours after subcutaneous injection of epinephrine in rabbits was reported by Pollack(25). There is increasing evidence however that insulin affects several phases of lipid metabolism. A marked reduction in the phospholipids of blood and liver in depancreatized dogs maintained with insulin was reported(26,27). A depression in lipogenesis in the alloxan-diabetic rat(28) and an increase in hepatic lipogenesis in the normal rabbit fed a high carbohydrate diet given insulin injection has been

reported(29). In general the evidence reported in the literature indicates that insulin's influence upon lipid metabolism is related through the effects of this hormone on glucose utilization. The data presented in Experiment II (Fig. 2) show that significant changes in the fractions of liver lipids followed the injection of insulin and epinephrine. However, the present data does not permit a definitive interpretation of a specific effect of either epinephrine or insulin on these lipid fractions because of the evidence recently reported that in the intact animal each of these hormones stimulates the secretion of the other hormone (30-32); the known effects which both of these hormones exert upon carbohydrate utilization and oxidation; and the increasing evidence of an influence of the latter processes upon lipid metabolism. A particularly puzzling aspect of the findings in this experiment is that the shift in the liver lipid pattern from that in the intact untreated animal is essentially the same found in the adrenalectomized, the adrenalectomized-alloxanized rats, and the intact rat which received injections of epinephrine or insulin. It would appear that the same changes in fat metabolism in the liver occurred in all 4 types of experimental animals. Further investigation of the effects of insulin and epinephrine with the purpose of delimiting their specific actions on liver lipid metabolism is presented in the succeeding paper.

The data of Fig 3 show clearly that administration of intravenous glucose affected the level of the liver lipid fractions. Also the results show that the endocrine status of the animals exerted a definite influence on the effects of the glucose administration upon the liver fractions. In the adrenalectomized group the increase in total lipid content was accounted for by the increased neutral fat fraction alone. In the intact and the adrenalectomized-alloxanized groups the changes in the neutral fat and the phospholipid fractions were in opposite directions following the glucose infusions. It is noteworthy that the liver glycogen content of the intact and adrenalectomized rats was significantly increased after the glucose (Table III) and in both these groups the neutral fat content was increased. The evidence presented is suggestive that in the absence of insulin and adrenal gland secretions there was a decrease in the level of fat metabolism following the glucose infusion. Furthermore, the data presented in Table III is suggestive that carbohydrate utilization was apparently proceeding at an extremely slow rate in the adrenalectomized-alloxanized group.

There are several general aspects of the liver lipid data obtained in these experiments which deserve special comment. First, significant changes in the level of the lipid constituents can occur with much greater rapidity (30-60 minutes) than is generally recognized. Thus, it appears that fat metabolism is as responsive to the changing metabolic requirements of the organism as is carbohydrate metabolism. Secondly, the changes in the levels of phospholipid and neutral fat were always in opposite directions. When changes occurred in the cholesterol fractions, they paralleled those for the phospholipids. These interrelationships are probably a reflection of the functions of these constituents in fat metabolism. Neutral fat being the form in which fat is mobilized and transported, and the phospholipid and cholesterol esters constituents involved in some stages of fat catabolism in the liver. Thirdly, it is apparent from the present data that the level of total fat in the liver is not a dependable criterion in studies on the influence of hormone deficiency or excess on fat metabolism in the liver. Some of the shifts in lipid pattern reported here occurred without any change being evident in total lipid values. The total lipid values found in the various experimental groups reported here appeared to be the resultant of opposing changes in the different fractions, and whether the values were the same, greater, or less than the respective controls was due to the quantitative rather than the qualitative nature of the changes in the level of the lipid constituents. Finally, it should be emphasized that the level of the various lipids found in these experiments are all within the so-called normal range for the rat. Thus, the significant changes are not due to grossly abnormal conditions but rather represent "normal" shifts in the pathways of metabolism in response

to changes in the hormonal pattern of the organism.

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Epinephrine and Insulin Effect on Potassium Mobilization: Relationship of Lipid and Carbohydrate Metabolism. (20229)

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The comparative effects of epinephrine and insulin on the potassium content of plasma, skeletal muscle, and liver in adrenalectomized-alloxanized rats given a glucose infusion were reported in a recent paper by Dury(1). The use of that type of animal and the design of the experiments were discussed in that report. The results indicated that the effects of epinephrine and of insulin upon tissue potassium content were of such divergent pattern that it reflected differences in the action of these two hormones on intermediary metabolism. Experiments were designed to obtain more detailed information concerning the constituents of the tissues of adrenalectomized-alloxanized rats given an infusion of a

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