In all nephrotic animals receiving tartaric acid, uranium or potassium chromate and dying in barbital coma there was a retention of barbital in the blood until death, amounting to from 0.15 mg. to 0.05 mg. per cc. of blood.

In all animals the damage to the kidney function was checked by phenolsulphonphthalein elimination and subsequent histological examination.

Conclusions. In severe experimental nephrosis, dogs and rabbits behave with reference to barbiturates as bilaterally nephrectomized animals. They *never recover from barbital depression, remaining anesthetized until death,* show *retention* of barbital *in the blood,* and also *eliminate* a relatively *small percentage* of the drug in the *urine.* However, they do recover from the sleep produced by barbiturates other than those largely eliminated by the kidney. In less severe cases there is a retarded recovery and decreased elimination of barbital in the urine with no retention of the drug in the blood above normal.

7137 P

Influence of a Certain Fraction of Pancreas Lipids on Carbohydrate Metabolism of Depancreatized Dogs.*

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Ever since the demonstration of the internal secretion of the pancreas there have been reports of substances antagonistic to insulin which increase the severity of diabetes. A blood sugar raising fraction of the pancreas reported by Gibbs, Root and Murlin[1] under the name of *glucagon* was precipitated by reagents which would precipitate the phospholipids and the cerebroside fractions of the pancreas fats. Further attention has been given recently to the separation of this fraction into its components. A fraction insoluble in acetone and ether, containing therefore the cerebrosides and some sphingomyelin has given some striking effects on the D:N ratios and the R.Q.'s of depancreatized dogs, but no consistent effect

* This paper was presented at the joint meeting of the Western New York branch and Section N of the A.A.A.S. at Syracuse, June 18, 1932.

1 Gibbs, C. B. F., Root, E. W., and Murlin, J. R., *Quart. J. Exp. Physiol.* Suppl. vol., 1923, 128.

on blood sugar. While it does not at this time seem to be identical with the fraction called glucagon in the earlier report, the difference in effects may be due to a difference in dosage. For the time being it will be referred to simply as a diabetogenic fat fraction.

Depancreatized dogs were treated with this fraction by subcutaneous injection. The diabetogenic fraction after precipitation with ether was centrifuged out and suspended in alcohol. The alcohol was then poured into physiological saline and boiled until no odor of alcohol could be detected. Dosage was reckoned by the amount of pig pancreas from which the dose was obtained. With 3 dogs the D:N ratio was markedly increased as shown in Table I.

TABLE I.
Summary of Results on Sugar and Nitrogen Excretion.

	Control 24 hours			Experimental 24 hours				% increase exer. of
Dog	D	N	D:N	D	N	D:N	Excess D	dextrose
14	29.56	12.12	2.44	34.74	10.57	3.29	5.19	17
15	10.06	4.08	2.47	15.50	4.41	3.51	5.44	54
17	6.36	5.61	1.13	17.61	6.19	2.85	11.25	177

The values given are for the 24 hours. The dogs were fed exactly the same on the 2 days. The urine was fractioned into three 4-hour periods and on one 12-hour period. The first 4-hour period (8 a. m. to 12 p. m.) was used as control, the diabetogenic fraction given just after the noon catheterization, and the effects on the metabolism studied in the afternoon. The dogs were fed at 8 p. m. The effect on sugar production and excretion was seen sometimes in the first

TABLE II.
The Influence of Diabetogenic Fraction of Pancreas Lipids on Carbohydrate Metabolism of a Depancreatized Dog. Dog 16. Depancreatized 3/3/32. Last Insulin Injection 4/8/32.

Time	D/hr.	N/hr.	D:N	Bl. Sugar	$L.CO_2$/hr.	$L.O_2$/hr.	R.Q.	Cal./hr.
				April 13, 1932				
8 a.m.	2.035	0.558	3.644	255				
9 "	Injected subcutan. 30 cc. diabetogenic fraction of pancreas lipids in 0.85% NaCl, equivalent to approximately 350 gm. pig pancreas.							
10 "					6.01	8.84	.70	41.41
11 "					5.51	8.38	.68	39.28
12 noon	1.42	0.42	3.37	268				
1 p.m.					5.86	9.27	.66	43.44
2 "					5.28	8.05	.67	39.28
3 "					4.72	7.40	.66	34.69
						Average	.68	39.62
4 "	1.25	0.47	2.63	255				
8 "	0.71	0.34	2.06	253				
				April 14, 1932				
8 a.m.	2.97	0.83	3.60	250				

experimental 4-hr. period, sometimes more plainly in the second, and always in the night period following feeding at 8 p. m.

The one dog which did not respond with a marked increase of D:N ratio, for the 24 hours, nevertheless showed a large increase of sugar excretion in the night period following injection at noon. This dog gave typical respiratory quotients for complete pancreatic diabetes on 2 different days and, as shown in Table II, exhibited a delayed depression of the R.Q., beginning 3 hours after administration of the fat fraction. The R.Q. was depressed as much or more than this in one other depancreatized dog and in a castrated but otherwise normal dog. Since depancreatized dogs exhibit very little ketosis, the depression of the R.Q. below the diabetic level is suggestive of gluconeogenesis from fatty acids.[2] We have not found any report in the literature of a similar effect from a similar lipid fraction.

7138 C

Dinitrophenol Hyperglycemia. I. Its Independence of Asphyxia.*

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The fact that 1-2-4 dinitrophenol, which greatly accelerates the oxidative metabolism of tissues, increases blood sugar concentration has been reported by Magne, Mayer and Plantefol[1] and confirmed by Hall, Field, Sahyun, Cutting and Tainter.[2] Since the large increase in oxygen usage of the tissues provoked by the drug might readily outstrip the ability of the respiratory and circulatory mechanisms to deliver oxygen and so lead to general bodily asphyxia, it seemed possible that the hyperglycemia might be of such asphyxial origin. The experiments described herein were designed to test this hypothesis.

Cats, anesthetized with pentobarbital, received doses of 15 or

[2] Hawley, Estelle E., Johnson, Carroll, and Murlin, J. R., *J. Nutrition*, 1933, **6**, 523.

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[1] Magne, H., Mayer, A., and Plantefol, L., *Ann. Physiol. Physicochim. biol.*, 1932, **8**, 1.

[2] Hall, V. E., Field, J., Sahyun, M., Cutting, W. C., and Tainter, M. L., *Am. J. Physiol.*, 1933, **106**, 432.