Portal dialysis: Five experiments (under amytal), gave 2 dialysates undoubtedly positive, latent period within first hour, 2 probably positive and 1 negative.

Caval dialysis: One experiment, under amytal, gave dialysate positive.

These results prove that a gastric secretory excitant occurs in the blood, and that it is more readily recovered from the portal and inferior caval blood than from the systemic circulation, in which it is probably less concentrated. It is possible that the excitant may be found in the blood before meals. Note the one carotid dialysate obtained before feeding which gave a slightly positive result. These experiments suggest that the excitant is increased after meals.

[1] Ivy, A. C., and Farrel, J. I., *Am. J. Physiol.*, 1925, lxxiv, 639.

[2] Lim, R. K. S., Loo, C. T., and Liu, A. C., *Chinese J. Physiol.*, 1927, i, (in press).

[3] Necheles, H., *Klin. Woch.*, 1923, ii, 27; *Chinese J. Physiol.*, 1927, i, (in press).

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Production of Hyperglycemia in Rabbits by Subcutaneous Injections of Magnesium Salts.

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The presence of magnesium in chlorophyll, and the important role of the latter in the metabolism of carbohydrates in plants, led us to study the effect of magnesium salts on blood sugar in rabbits. Five normal rabbits, starved for 24 hours, were injected subcutaneously with a 5 per cent solution of $MgCl_2$ or $MgSO_4$, using 5 cc. of this solution per kilo of body weight. During the experiment the rabbits were kept quiet in a tray, without restraint, and there was no struggle when the 2 cc. of blood were taken from the marginal ear vein. Such moderate bleeding does not affect essentially the blood sugar level in rabbits, as shown in a previous paper[1].

The results of the experiments are presented in the following table:

Rabbit No.	Magnesium salt solution (5 per cent)	Time of injection	Time when blood was taken	Glucose in blood	Hemoglobin (in per cent of normal human values)
				mg. per 100 cc.	
1	$MgCl_2$	9:40 a. m.	9:35 a. m.	121	
			11:40 a. m.	200	
2	$MgCl_2$	9:50 a. m.	9:45 a. m.	118	
			11:50 a. m.	250	
3	$MgCl_2$	9:35 a. m.	9:30 a. m.	125	
			10:00 a. m.	143	
			11:00 a. m.	182	
			12 noon	222	
4	$MgSO_4$	9:30 a. m.	9:25 a. m.	117	42
			10:00 a. m.	133	40
			11:00 a. m.	154	44
			12 noon	182	43
5	$MgSO_4$	10:00 a. m.	9:30 a. m.	125	
			12 noon	222	
			3:00 p. m.	143	
			9:30 a. m. (the following day)	105	

This table shows that subcutaneous injection of magnesium salts produces a marked hyperglycemia, with a maximum in from 2 to 2 1/2 hours. In 5 1/2 hours the blood sugar is not far from its initial level, and in 24 hours it has reached a normal value. Controls, on the hemoglobin content, show that the hyperglycemia is not due to a change in blood concentration. Similarity in the results obtained by subcutaneous injections of $MgCl_2$ and $MgSO_4$ is a sufficient proof that the hyperglycemic effect is due to the magnesium ion.

Our data throw light on the understanding of the therapeutic value of magnesium salts, given *per os* or intravenously, especially in the treatment of nephritic uremia and eclampsia. According to Blackfan[2], the treatment of uremia (occurring in the course of acute glomerular nephritis) by intravenous injections of 1 per cent magnesium sulphate, has given most favorable results. Lazard, Irwin and Vruwink[3], report that intravenous administration of magnesium sulphate in sufficient dosage will prevent the development of convulsions in eclampsia, and will control them after their onset. Titus, Hoffman and Givens[4], and also Titus and Givens[5], say that the intravenous injection of glucose is a valuable therapeutic measure in toxemia of pregnancy, and that, theoretically, there

is no reason why it should not be of value in the various toxemias not related to pregnancy.

[1] Horvath, A. A., *J. Biol. Chem.*, (in press).

[2] Blackfan, K. D., *Bull. Johns Hopkins Hosp.*, 1926, xxxix, 69.

[3] Lazard, E. M., Irwin, J. C., and Vruwink, J., *Am. J. Obst. and Gyn.*, 1926 xii, 104.

[4] Titus, P., Hoffmann, G., and Givens, M., *J. Am. Med. Assn.*, 1920, lxxiv, 777.

[5] Titus, P., and Givens, M., *J. Am. Med. Assn.*, 1922, lxxviii, 92.

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Trichomoniasis in Kittens.

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Da Cunha and Muniz[1] in South America have reported the finding in a cat of a flagellate to which they have given the name, *Trichomonas felis.* Brumpt[2] later records Trichomonas in two kittens in Paris. One of these kittens had been previously infected experimentally with human feces containing *Endamoeba dysenteriae,* and it therefore is possible that this Trichomonas had been derived from the human source. Brumpt did not state definitely whether opportunity of laboratory transmission was afforded the second kitten. He transferred these flagellates to other kittens, and accepted the name *Trichomonas felis* for them, instead of considering the possibility that the infections might have been derived from other animals, supporting his claim primarily on the basis of the negative transmission results of Hogue[3], who had been unsuccessful in establishing *Trichomonas hominis* from man in kittens.

During the spring and summer of 1926, while engaged in a study of experimental amoebiasis in kittens, the writer found 9 kittens, purchased from the streets of Peking, which showed natural infections of Trichomonas. This Trichomonas, as studied in saline and iodine-eosin smears, did not differ morphologically from the Trichomonas described by Brumpt, from kittens, nor from the common *Trichomonas hominis,* from man. The flagellar count of the majority of the specimens was four. The infected kittens showed a diarrhea consisting of a yellowish-brown stool, and in two instances

* Contribution No. 83.