dogs the mean value is the same for all 3 stimuli. In the third the difference was not significant.

Comment. The specific gravity of pancreatic juice has been used in this laboratory as a measure of the nitrogen output(3) and, by inference, the protein output of the pancreas(4). The latter, in turn, may be taken as an index of the output of enzymes(5-7). Our results indicate that canine gastric juice secreted in response to either histamine or insulin contains no substance other than HCl which modifies either the volume or the nitrogen content of pancreatic juice. Probably the slight stimulating effect of some commercial preparations of substances normally found in gastric juice is due to contaminants.

Conclusion. Canine gastric juice contains no stimulus for the pancreas other than HCl, which can be revealed by our method of study.

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Hypoproteinemia as Protection in Mercuric Chloride Poisoning. (19244)

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Certain experimental states apparently protect animals from $HgCl_2$ toxicity. A hemoglobinuric dog survived 4 mg $HgCl_2$/kg(1), albuminuric rats survived toxic doses of organic mercurials(2), and hypoproteinemic dogs survived 3 mg $HgCl_2$/kg(3). A possible mechanism for the latter action is indicated by the present experiments.

Experimental. Two normal dogs fed a 6% protein 10:3:3 crackermeal:sugar:lard diet supplying 75 Cal./kg/day were plasmapheresed daily by replacement of 25% estimated blood volume by an equal volume of a 50% dog erythrocyte suspension in 0.9% NaCl, until plasma protein totaled less than 4.4 g %. With 5 untreated normal dogs as controls, all dogs were given 3 mg $HgCl_2$/kg intravenously, except one hypoproteinemic dog who received 4 mg/kg. Daily urine Hg^{++} content(4) and clinical signs were noted. To urine samples having less than 0.001 mg Hg^{++}/ml, the lower limit of 3% accuracy for this method, .002 mg $HgCl_2$ was added per ml. Table I shows probable error limits of each determination. Surviving (hypoproteinemic) dogs were then put on a stock diet of 1/4 pound raw horse meat daily plus Friskie dog chow *ad lib.*, and 3 additional doses of 3 mg/kg of $HgCl_2$ were given 1, 2, and 6½ months after the initial dose. Except for omission of urinalysis after the fourth dose, procedure was the same each time.

Results. The 4 control dogs whose plasma protein level exceeded 6.1 g % died 2½ to 4½ days after the $HgCl_2$ dose. The fifth control dog had 5.8 g % plasma protein and died in 16½ days. Each had anorexia, weakness, emesis, bloody diarrhea, ulcerative stomatitis, initial diuresis, albumin- and hemoglobinuria and, in the 12-hour period before death, hyperventilation with rates up to 240/min., weak and rapid pulse and tetany of facial, neck and shoulder muscles. Dehydration, fatty liver, noncrepitant lungs, hemorrhagic intestinal mucosa, and necrosis and edema of renal cortices were seen at autopsy. No control dog excreted as much as half the $HgCl_2$.

TABLE 1. Cumulative % Excretion of Mercuric Chloride.

Days after dose	Control dogs					Initially hypoproteinemic dogs					
						First dose		Second dose		Third dose	
	1 (6.5)*	2 (6.1)	3 (7.4)	4 (6.3)	5 (5.8)	1 (4.4)	2 (4.2)	1 (7.7)	2 (5.7)	1 (6.9)	2 (6)
1	11.4	26	38.1	45.8	36.5	47.5	42.7	48.8	39.6	60.2	49.7
2	12.7	28.7	38.4 (5)†	46.1 (21)	37.6 (8)	65.6	55.9	54.4	47	66.7 (4)	55
3	12.9 (7)	28.9 (5)	38.7 (10)	46.3 (13)	38.1 (9)			57	50.4	70.2	59.1
4		29.3 (8)	38.9 (6)		38.3 (17)	78.7	62.3	57.6 (6)	52.5 (5)	72.5 (5)	61.4
5		30 (7)			38.4 (30)	89.1	65.9	58.8	53.6 (4)	73.9	63.7
6					38.6 (14)	93.4	68.7	59.4 (7)	54.3 (5)		
7					38.7 (21)	94.6	70.2	60.2 (6)	54.8 (14)	74.3 (44)	63.8 (37)

* Figures in parentheses indicate g % total plasma protein at time of $HgCl_2$ inj.
† " " probable error of determination of Hg^{++} excreted when probable error is over 3% in that 24 hr period, assuming probable error of original method to be no more than 3%.

The 2 hypoproteinemic dogs showed essentially no toxicity. Stomatitis, bloody diarrhea and fever were seen only within the 4 days after the second and third doses. In each case, each dog excreted more than half the Hg^{++} within 3 days. The fourth dose, half a year after the initial hypoproteinemia, caused the characteristic response to the poison, terminating in death within 5 days.

Discussion. Hypoproteinemia is characterized by a relatively large and prolonged albumin deficit, albumin restoration lagging well behind the osmotically less active globulin. The lowered colloidal osmotic pressure allows increased extracellular volume(5) and therefore a greater dilution of the poison. Probably more important, mercury is partially bound to plasma albumin and the per cent bound is less when the albumin:mercury ratio is low (6). "Free Hg^{++}" is greatest with low plasma albumin; this is assumed to be the basis for the increased excretion rate over controls in our experiments. Since return of albumin to normal electrophoretic pattern takes over 3 times as long as the return of total albumin concentration, and requires many weeks when protein intake is only moderate(7), protection from Hg^{++} toxicity would last even longer if Hg^{++} is bound primarily to a slowly-returning fraction. So far there is no independent evidence for the last possibility, but the effects of total albumin level may be crucial. This may explain the finding that hypoproteinemic dog 1 was protected from the second dose, with a plasma protein level of 7.7 g %, and was killed by the fourth dose 5½ months later, with a plasma protein level of 6.7 g %. The possibility exists that changes in the pattern of renal and other intracellular proteins on protein depletion may alter the susceptibility of the cells to the toxic effects. Since there are no data on this point, further speculation does not seem profitable.

Summary. Five normal and 2 hypoproteinemic dogs were given similar intravenous $HgCl_2$ injections. Each normal dog died with characteristic mercury poisoning signs; the hypoproteinemic dogs survived this and 2 more such doses, time interval between doses being 1 month, but died from a fourth dose

given 6½ months after the initial hypoproteinemia, at which time their plasma protein levels were 6.7 and 5.8 g %. Control dogs excreted less than half the mercury; the hypoproteinemic dogs excreted over half of it within the first 3 days. It is suggested that the resistance to mercury toxicity seen in the hypoproteinemic animals may be due to increased extracellular volume and, more important, a relatively small binding of Hg++ by protein, permitting more rapid excretion and diminished cell susceptibility.

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Effect of Intravenous Injections of Desoxycorticosterone Glucoside upon Blood Glucose of Adrenalectomized Dogs.* (19245)

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It is well established that the oil soluble preparation of DCA, given intramuscularly in small doses, exerts little, if any, effect upon the intermediary metabolism of carbohydrate. On the other hand, those adrenal steroids oxygenated at C-11 are highly efficacious in this respect(1-5). However, within recent years several reports have appeared indicating that DCA, administered in large doses, induces glycosuria in the force-fed, partially depancreatized rat(6) and "restores glycogen production to normal from carbohydrate or protein in liver and muscle of adrenalectomized animals which are kept alive for some time" (7,8). Harrison and Harrison(9) concluded from their experiments that "the injection of adequate amounts of desoxycorticosterone acetate into fasted adrenalectomized rats does prevent the drop in concentration of blood sugar found in untreated adrenalectomized rats." Other investigators have reported some positive effects of large amounts of DCA on gluconeogenesis and blood sugar level of adrenalectomized rats(10) and the glucose requirements of adrenalectomized-eviscerated rats(11). In the course of a study on the efficiency of the water soluble glucoside of desoxycorticosterone‡ (DCG), in reviving adrenalectomized dogs from insufficiency, massive doses of this steroid were administered as a single injection by vein. Since the amounts employed were far larger than anything reported in the literature, and since fasted adrenalectomized animals receiving DCG, during revival from severe insufficiency, were only occasionally observed to exhibit sharp increases in blood glucose, it was considered worth while to study in more detail the possible correlation between DCG and blood glucose levels.

The present report is based upon a study of 16 adrenalectomized dogs and one intact animal. The type of dog employed, amount of DCG administered and other details of the

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