held true also for the lobules, although there appeared to be no increase in their actual number. There was evidence of active secretion as indicated by the presence of colostrum corpuscles and what appears to be milk.

[1] Herrmann, E., *Monatsschr. f. Geburtsh. u. Gynak.*, 1915, xli. 1.
[2] Fellner, O., *Arch. f. Gynak.*, 1913, c, 641.

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Effect of Bilateral Nephrectomy Upon the Acid-Base Equilibrium of Dogs.*

W. W. SWINGLE, W. F. WENNER AND P. STANLEY.

From the Zoological Laboratroy, State University of Iowa.

For several years the senior writer and his students have been studying the effect of bilateral adrenal extirpation in cats and dogs and reached the conclusion that one of the train of causes resulting in death from adrenal ablation is acid intoxication.[1, 2, 3, 4] As a result of our experiments the hypothesis was advanced that the adrenal cortex secretes a hormone which in some manner assists in maintaining the normal functioning of the kidney. We were interested in the fact that the type of acidosis which appears during adrenal insufficiency is similar to that occurring in uremia. As a further means of testing the idea whether or not the kidney is involved in adrenal insufficiency the present writers undertook to make a careful comparison of the symptoms and blood findings occurring in adrenal insufficiency with those which follow kidney extirpation.

Large, well nourished dogs were employed for the kidney work —the average weight being 18-20 kilos. The right kidney was extirpated and after a 7 to 10 day interval the left kidney was removed. Animals so operated generally remain normal for several days before untoward symptoms develop. The unilaterally nephrectomized dogs were bled for CO_2 capacity, CO_2 content, pH, phosphorus, sulfur, chlorides, sugar and urea. Later when symptoms of renal insufficiency appeared the animals were bled at various times.

The first symptoms noted were anorexia and lassitude, the animals appearing normal otherwise. Later they vomited considerably, refused all food and had to be fed daily 200-400 cc. of milk by stomach tube. Weakness of the hind limbs appeared, drowsiness

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and coma. Our animals survived the double operation for varying periods (3-6 days). It is probable that the survival period would have been greater if the animals had not been bled repeatedly.

The results show that acid intoxication is not a prominent symptom of renal insufficiency in dogs—in fact our data indicate that marked acid intoxication does not appear in nephrectomized dogs even near the terminal stages. There is usually a slight drop in the CO_2 capacity and content, a negligible change in pH, marked increase in inorganic sulfate and phosphate and urea, some increase in blood sugar and a marked drop in chloride. We are not prepared to say at this time, whether or not the quantity of chloride lost in the vomitus is sufficient to offset the marked increase in inorganic phosphate and sulfate, thereby maintaining an approximately normal acid-base equilibrium.

There are some striking resemblances and also marked differences between the blood picture of adrenalectomized and nephrectomized dogs. Adrenalectomized animals invariably develop a marked uncompensated acidosis with low CO_2 capacity, CO_2 content, pH, and sugar. The blood sugar of such animals falls despite forced feeding, in the later stages of adrenal insufficiency. The amount of urea, non protein nitrogen, inorganic phosphate and sulfate in the blood are greately increased in both nephrectomized and adrenalectomized animals. All dogs survived the second operation approximately the same length of time, *e. g.,* 4-6 days. It will be noted at once that the chief difference between nephrectomized and adrenalectomized dogs are: Adrenalectomized dogs with intact kidneys show marked acid intoxication and fall in blood sugar, nephrectomized dogs, on the other hand, show but slight evidence of acidosis and their blood sugar rises and remains higher than normal up until the time of death. Moreover, phosphate and sulfate values are considerably higher in the nephrectomized dogs showing symptoms than in adrenalectomized animals—but despite this increase acid intoxication does not appear as a marked symptom.

It would seem not improbable, therefore, that the acidosis of adrenal insufficiency and probably also of uremia is extrarenal in origin and probably only partially dependent upon kidney injury because, as we have indicated, complete kidney removal does not produce such symptoms or at most, but very slight symptoms.

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[1] Swingle, W. W., and Eisenman, A., *Am. J. Physiol.*, 1927, lxxix.

[2] Swingle, W. W., and Wenner, W. F., PROC. SOC. EXP. BIOL. AND MED., 1927, xxv, 169.

[3] Swingle, W. W., *Am. Naturalist*, 1927, lxi, 132.

[4] Yonkman, F., PROC. SOC. EXP. BIOL. AND MED., 1927, xxiv, 786.