9710

A Study of the Factors Concerned in Renal Hypertension.*

L. N. KATZ, M. MENDLOWITZ† AND M. FRIEDMAN. (In collaboration with W. Weinstein and S. Rodbard.)

From the Cardiovascular Department, Michael Reese Hospital, Chicago, Ill.

Since the fundamental work of Goldblatt and his associates,[1] persistent hypertension in dogs by renal ischemia has been produced by other investigators.[2-5] However its etiology and exact mechanism are still unknown.

We have been particularly interested in the effects of clamping one of the renal arteries. Various observers[1, 5, 6] have noted that following the clamping of one renal artery, one of 3 results may be expected; *viz.*, (a) a permanent rise, (b) a temporary rise or (c) no alteration in blood pressure. This report is based on observations in 6 dogs in whom unilateral renal artery clamping by the Goldblatt technique[1] was carried out. Five of these 6 dogs showed a rise in blood pressure following this procedure. In 2 of these 5 dogs, the blood pressure returned to normal within 3 and 8 weeks, respectively; the blood pressure in the other 3 fell somewhat but did not return to the preoperative level. The blood pressures were uniformly determined in the afternoon on trained animals. A direct arterial puncture was made under local anesthesia (2% procain subcutaneously) with an ordinary hypodermic needle. The pressure was recorded with a calibrated Hamilton manometer on bromide paper.[7] From 30 to 101 days following the unilateral clamping, the normal kidney was removed from each of the 6 dogs, leaving only the kidney with its renal artery previously clamped. In 5 of the 6 dogs, a marked rise in both systolic and diastolic pressures occurred following the removal of the normal kidney. The sixth

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1 Goldblatt, H., Lynch, J., Hanzal, R. F., and Summerville, W. W., *J. Exp. Med.*, 1934, **59**, 347.

2 Collins, D., *Am. J. Physiol.*, 1936, **116**, 619.

3 Elaut, L., *Compt. rend. Soc. de Biol.*, 1936, **122**, 126.

4 Page, T. H., and Sweet, J. E., PROC. SOC. EXP. BIOL. AND MED., 1936, **34**, 260.

5 Blalock, A., and Levy, S. E., *Ann. Surg.*, 1937, **106**, 829.

6 Harrison, T. R., Blalock, A., and Mason, M. F., PROC. SOC. EXP. BIOL. AND MED., 1936, **35**, 122.

7 Hamilton, W. F., Brewer, J., and Brothman, I., *Am. J. Physiol.*, 1934, **107**, 427.

dog, which died in uremia and had never shown any rise in blood pressure, was found at necropsy to have had the renal artery completely occluded by the Goldblatt clamp. Three of the other animals also died in uremia, while 2 are still living. In the 3 latter dogs dying in uremia, the pressure rise following removal of the normal kidney averaged 38 mm. Hg. for the systolic and 33 mm. Hg. for the diastolic pressure. The other 2 dogs which are still living (one has now survived for 90 days following the nephrectomy) showed an even greater rise in pressure following removal of the normal kidney, *viz.*, an average of 62 mm. Hg. for the systolic and 32 mm. Hg. for the diastolic pressures.

Fig. 1 is a chart of one of the latter dogs. It will be observed that there is a marked rise in blood pressure following the removal of the normal kidney greater than the rise previously caused by partially occluding the renal artery of the other kidney. The blood non-protein nitrogen in this dog was always relatively low and at no time was there evidence of uremia. Blalock and Levy[5] and Goldblatt[8] also have observed a rise in blood pressure following the removal of the normal kidney in a dog whose other kidney had been rendered ischemic previously.

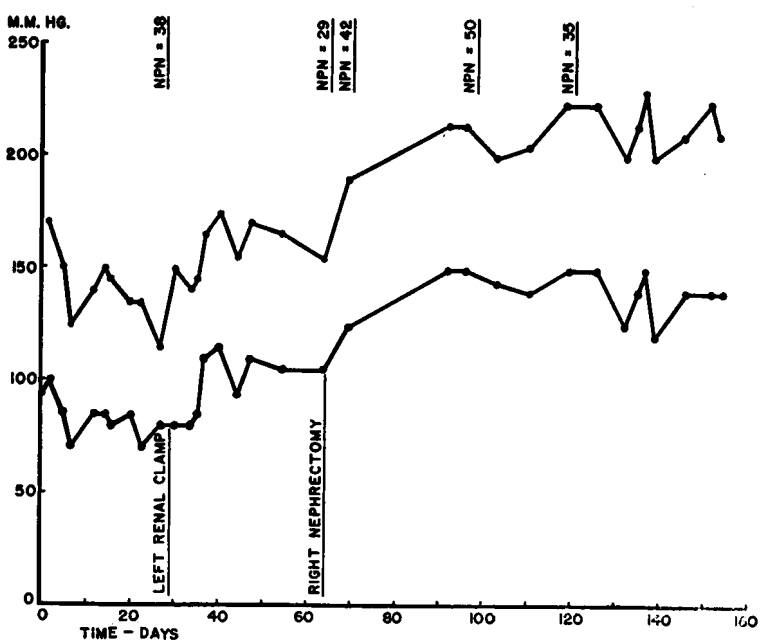

FIG. 1.
NPN is blood non-protein nitrogen in mg. %

8 Goldblatt, H., personal communication.

These experiments clearly indicate that the temporary hypertension produced by ischemia of a single kidney may reappear or be further increased and protracted when the remaining normal kidney is removed. Experiments such as ours are difficult to reconcile with a neurogenic origin of renal hypertension but fit better with the current view of a humoral origin. It is noteworthy that removal of a normal kidney not only permits the development of hypertension, but sometimes of uremia as well. Hypertension of renal origin depends, our results suggest, on the ratio of ischemic to normal kidney tissue rather than on the amount of ischemic renal tissue alone. Decreasing the former will tend to have an effect similar to increasing the latter.

Summary. Six dogs had ischemia produced in one kidney by a Goldblatt clamp applied to one renal artery, and after a varying time interval, the normal kidney was removed. This latter procedure caused a noticeable rise in blood pressure in 5 out of the 6 animals, and 4 of the animals died in uremia.

9711 P

Treatment of Hepatomegaly in Juvenile Diabetes Mellitus with a Pancreatic Extract.

HAROLD G. GRAYZEL AND LEO S. RADWIN. (Introduced by B. Kramer.)

From the Department of Pediatrics and the Pediatric Research Laboratories of the Jewish Hospital of Brooklyn.

Hepatic enlargement is not a common complication of diabetes mellitus. Its incidence is much higher during the first 2 decades of life than later.[1] Excluding cirrhosis of the liver, the most frequent pathological finding in cases of this type is a fatty infiltration and degeneration of the liver.

As a rule the condition occurs in severe and uncontrolled diabetics.[1, 2] In most cases, adequate management of the diabetes with diet and insulin, and more recently with protamine-zinc-insulin[1, 2] results in hepatic recession. However, there are instances where careful dietary management coupled with adequate insulin therapy

1 Hanssen, P., *J. A. M. A.*, 1936, **106**, 914.

2 White, P., *Diabetes in Childhood and Adolescence*, Philadelphia, Lea and Febiger, 1932, p. 169.