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Effects of Sodium Intake on Renal Cortical Blood Flow Distribution (39238)

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Two separate nephron populations in the mammalian kidney have been described by Trueta *et al.* (1) and others (2–4). That the different anatomical structures of outer and inner cortical nephrons may be important in the regulation of sodium excretion was suggested by Horster and Thurau (2) in their micropuncture study. They demonstrated that superficial nephron glomerular filtration rate (GFR) increased and juxtamedullary nephron GFR decreased on a high sodium diet. Theoretically, such a redistribution of nephron GFR could be concomitantly regulated by alterations of intrarenal blood flow distribution. In this study, we have evaluated renal cortical blood flow distribution in chronic sodium-loaded and sodium-deprived rats.

Methods. High sodium group. Eight Sprague–Dawley rats weighing 70–100 g were fed Purina rat chow and given 1% sodium chloride solution in place of drinking water for a period of 4–5 weeks.

Low sodium group. Eight Sprague–Dawley rats weighing 150 g were fed sodium-deprived rat chow (Nutritional Biochemicals) and were given distilled water to drink for a period of 4–5 weeks.

A group of rats weighing approximately 200 g, fed with Purina rat chow and drinking tap water, served as controls.

Twenty-four-hour sodium excretion was measured in high, low, and normal salt-intake rats using metabolic cages. Renal blood flow (RBF) was measured by the microsphere method (5). All animals were lightly anesthetized with ether and were cannulated with polyethylene tubing No. 10 through the femoral artery for blood collection and through the carotid artery into the left ventricle for injection of microspheres. After awakening from anesthesia, the animals were placed in restraining cages and allowed to recover for 45–60 min prior to microsphere injection. Two separate measurements of RBF were taken 1 hr apart using two different nuclides (^{85}Sr- and ^{141}Ce-labeled microspheres 15 ± 5-μm diam). RBF was calculated as:

$$\text{RBF} = \frac{\text{total kidney cpm}}{\text{femoral blood cpm}} \times \text{femoral blood flow rate (millilters per minute).}$$

A coronal section of the midportion of the kidney (0.3–0.4 cm thick) was sliced into sections less than 1-mm thick on a Stadie–Rigg's microtome. The cortex was separated from the medulla with a scalpel and the outer two-thirds of the cortex was cut and weighed. Superficial cortical blood flow (OC-RBF) was determined by counting this portion. Absolute blood flow rate per gram of superficial cortex was calculated as:

$$\text{OC-RBF} = \frac{\text{cortical tissue cpm}}{\text{total kidney cpm} \times \text{cortical tissue wt}} \times \text{RBF}.$$

The details of this procedure have been previously described (5). The values of RBF determined by ^{85}Sr and ^{141}Ce were averaged for each rat, and data were expressed as means ± SEM. Statisical analysis was performed using the Student's t test.

Results and discussion. The 24-hr sodium excretion rate of rats fed a normal sodium diet was 2.04 mEq (Table I). This value was significantly higher than that of low sodium diet animals (0.06 mEq/24 hr, $P < 0.001$), and lower than that of high

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TABLE I. RENAL CORTICAL BLOOD FLOW OF RATS ON NORMAL, HIGH, AND LOW SODIUM INTAKES.[a]

Experiment	RBF/min/100 g body weight (ml)	RBF/min/g kidney weight (ml)	OC-RBF/min/g kidney weight (ml)	Na excretion (mEq/24 hr)	Serum Na (mEq/liter)	Body weight (g)
Normal salt intake + water (N = 9)	4.97 ± 0.33	6.36 ± 0.38	11.56 ± 0.88	2.04 ± 0.31	146.1 ± 0.92	229 ± 8.02
Normal salt intake + 1% saline (N = 8)	4.80 ± 0.37	6.20 ± 0.45	11.03 ± 1.14	13.31 ± 1.10[b]	143.9 ± 0.99	310.5 ± 15.9
Salt deprived + water (N = 8)	4.80 ± 0.18	5.57 ± 0.37	10.91 ± 0.57	0.06 ± 0.002[b]	145.8 ± 0.96	261.8 ± 9.6

[a] All values = mean ± SEM. RBF, renal blood flow; OC, outer cortex; N, number of animals.
[b] Significantly different from normal salt intake + water animals ($P < 0.001$).

sodium diet animals (13.3 mEq/24 hr, $P < 0.001$). Serum sodium concentrations, however, were not different among these groups.

RBF of control rats on a normal sodium diet was 6.36 ml/min/g of kidney or 4.97 ml/min/100 g of body weight. This is identical to that found by Arendshorst *et al.* (6) using an electromagnetic flow probe. RBF values of rats on either a low sodium or high sodium diet (Table I) were not statistically different from control. Since microspheres are trapped in the glomerular capillary beds, the RBF measurement actually determines renal cortical blood flow. The superficial cortical blood flow (OC-RBF) in low sodium or high sodium rats was not different from that of normal sodium diet rats. Although juxtamedullary cortical blood flows were not measured, neither total renal cortical blood flow nor superficial cortical blood flow was different among the three groups of animals. Thus, juxtamedullary cortical blood flow also must be similar.

Goodyer and Jaeger (7) suggest that alterations in RBF and filtration distribution patterns within the kidney might account for changes in sodium excretion, even though total GFR remains unchanged. Horster and Thurau (2) found that in rats fed a high sodium diet the superficial nephron GFR was almost twice as much as in rats fed a low sodium diet, whereas whole kidney GFR was unchanged. Further, during acute volume expansion in the rat, GFR tends to increase more in superficial nephrons than in deep nephrons (8, 9), although some investigators find no evidence of such filtration redistribution (10). Hollenberg *et al.* (11) demonstrated intrarenal redistribution of blood flow in humans on different sodium intakes by using the xenon washout method. They found significantly higher fractional outer cortical and lower fractional inner cortical flows in persons on a high sodium diet compared to those on a normal sodium intake. Exactly the opposite redistribution of cortical flow occurred in individuals on a low sodium diet. Kinney *et al.* (12) using the xenon washout technique in the rat, showed that chronically sodium-deprived rats had a higher percentage of outer cortical flow than did normal sodium rats. However, since outer cortical blood flow distribution varied so much among control rats, the authors concluded that outer cortical flow and distribution were probably similar in both groups.

In the present study, we were not able to confirm redistribution of intracortical blood flow in rats fed a high or low sodium diet. Baines (13) has shown that redistribution of nephron GFR and increased superficial nephron filtration occur in young rats, but not in older rats. It is possible that our failure to demonstrate renal cortical blood flow redistribution may be related to the maturity of the animals, as they all weighed more than 200 g.

Summary. Renal cortical blood flow and superficial cortical blood flow were measured in chronic sodium-loaded, sodium-deprived, and normal rats. Neither total renal cortical blood flow or distribution of cortical blood flow was different among the three groups of animals. Alterations in the amount of sodium excreted, therefore, are

not related to alterations of renal cortical blood flow distribution.

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