# Dietary Sodium in the Development of Renal Hypertension[1] (34205)

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Sodium retention has been shown to aggravate renoprival hypertension (1); it is essential for the development of DCA hypertension (2); and balance studies have shown that it plays a part in the early stages of the development of hypertension after the application of the Goldblatt clamp to the renal artery (3).

The object of the present study was to investigate the role that sodium retention might play in the maintenance of hypertension over a longer period of time. For this purpose, the evolution of hypertension was compared in rats maintained on a normal sodium intake with those on a virtually sodium-free diet.

*Material and Methods.* One hundred young Wistar rats, of approximately 200 g, were used in this experiment. Blood pressure was recorded from the tail by the microphonic technique (4) twice weekly before and after the application of the silver clip to one renal artery, with contralateral nephrectomy. After the operation half the animals were placed on a synthetic diet, low in sodium (50 mg/kg)[2] (5); while the other half were fed the same diet to which 0.6% sodium chloride was added. Distilled water was permitted *ad libitum*. After 8 weeks on the respective diets, the sodium intake of the two diets was switched for a further period of 5 weeks.

In addition, two groups of control animals were followed concurrently. They were subjected to a sham operation in which a unilateral nephrectomy was performed and a clip was placed on the opposite renal artery and then immediately removed. Twenty-six of these received the low sodium and 22, the higher sodium intake. The diet of the control groups was not switched at 8 weeks to permit evaluation of growth and blood pressure trends in these animals over the entire 14-week period.

*Results.* Hypertension developed over a period of 4–6 weeks in both groups of experimental animals as is illustrated (Fig. 1); but those on the low sodium diet maintained a lower blood pressure throughout the initial period of 8 weeks, at the end of which the mean systolic blood pressure of 158 (SD 28) mm Hg was significantly lower than those with a normal sodium intake, 176 (SD 26) mm Hg, $p<0.05$. When the diets of the experimental groups were changed, the difference between their blood pressures disappeared. This was partly attributable to a rise in blood pressure in those formerly on a low sodium intake and a slight fall in those who had the normal sodium intake.

In the control groups, the systolic pressures were virtually identical for those on a low or normal sodium intake. The weight curves were also identical but, in spite of this, the weight gain in the experimental group on the normal sodium intake was lower than it should have been. This difference reached 47 gm at 8 weeks. With the restoration of a normal sodium intake, the weight in this group promptly rose to the normal level; on the other hand, the experimental group which had been on a normal sodium intake ceased to gain weight as soon as they were placed on the low sodium diet.

*Conclusions.* Renal hypertension in rats appears to be moderated but not prevented by sodium restriction. This finding is analagous to that obtained in short-term balance studies in the dog (3). Thus indicating that this type of hypertension can be maintained in the face of a very low sodium diet. It has been suggested that sodium restriction does

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[2] General Biochemicals, Cleveland, Ohio.

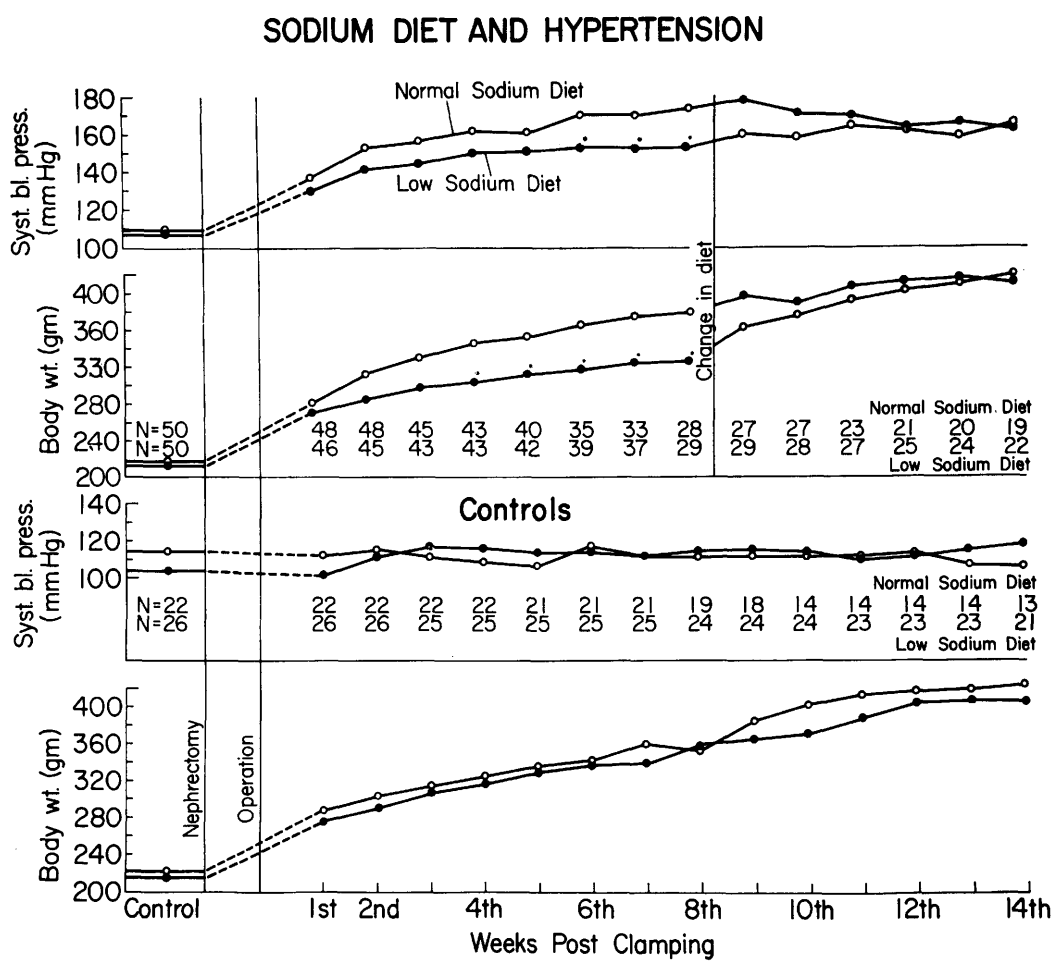


FIG. 1. Evolution of hypertension in animals fed on diets differing in sodium content. Systolic blood pressure and weight gain in rats on a normal (○) or a low sodium intake (●). All animals were subjected to nephrectomy; (upper), a clip was applied to the opposite renal artery; and (lower), a sham operation on that side; *N* indicates the numbers of animals in the study during each week of observation; *Indicates a "significant difference" $p<0.05$ between rats on the sodium deficient diet as compared to those on a normal diet.

not affect the level of blood pressure in hypertensive rats (6, 7); although if a low sodium diet was administered before clipping the renal artery, the subsequent level of blood pressure was lower (7). The satisfactory survival rate and weight gain in the control animals on a low sodium diet in the present study attest to its adequacy in this respect. The clipped rats on a low sodium diet failed to gain weight normally. This unexpected finding is without explanation, but it may be related to the polydypsia which is known to develop after the application of a clip to the renal artery (8). In conclusion, severe sodium restriction did not prevent development of experimental renal hypertension even though free access to sodium aggravated the condition.

*Summary.* The evolution of experimental renal hypertension was studied in rats on a sodium deficient diet (50 mg/kg) as compared to those on the same diet with 0.6% sodium added and also with sham operated controls on the two diets. On the low sodium diet, hypertension emerged more gradually and at 8 weeks, the blood pressure was sig-

nificantly lower than those with the normal intake. The mean difference, however, was only 18 mm Hg. The experimental animals on the sodium-deficient diet weighed less than those on the normal diet. In control animals without a clip on the renal artery, there was no difference, according to diet, in weight gain or indeed in blood pressure.

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