44) Effects of Dietary Chloride on Spontaneously Hypertensive Rat—Comparison of NaCl and NaHCO₃. Masae Tanaka, Sadaharu Kato, Chiho Inagaki, Mariko Hosaka, Akihiro Ohno, Tomoko Kanamaru, Nagako Nakanishi, Nobuhiro Sugino. Department of Medicine, Kidney Center, Tokyo Women's Medical College, Tokyo 162.

It has been reported that dietary chloride contributes to produce hypertension in some animal model of salt-sensitive hypertension. The present study examined the effect of chloride on producing hypertension of SHR by two sources of sodium loading test: NaCl and NaHCO₃. Fifteen male SHR were maintained on a regular diet (0.41g/100g Na content). At 6 weeks of age the rats were divided into two groups and received one of the following solution ad libitum : a chloride solution (0.9%NaCl solution), or a bicarbonate solution (1.28%NaHCO₃ solution). Both solutions were made equimolar in sodium. During the 11-week study, both groups were fed a low sodium diet (6.45mg/100g Na content). In addition, body weights were recorded, and systolic blood pressures of unanesthetized rats were measured by the tail cuff method once or twice a week. The rats were housed in individual metabolic cages so that water intake and urine volume could be measured every other week. Arterial blood samples were obtained for the measurement of PH, Pco₂, HCO₃, and plasma electrolyte every three weeks.

From week 6 until the end of the experiment, the blood pressure of the two groups were not different from each other. The increase in water intake and urine volume was more striking in NaCl group than in NaHCO₃ group. From 9 to 11 weeks of age, sodium intakes and its excretions of NaCl group were two or three times as much as those of NaHCO₃ group. Chloride excretions of NaHCO₃ group were very low, whereas potassium excretions and serum sodium and chloride were not different from each other. Metabolic alkalosis was not observed in NaHCO₃ group by arterial blood gas analysis.

It has been reported that dietary chloride contributes to produce hypertension in salt-dependent model: DOCA salt hypertension rat and Dahl salt-sensitive rat. However, it has not been reported that chloride contributes to produce hypertension in SHR that is not the apparent model of salt-sensitive. The present study did not show the differences of blood pressure between NaCl and NaHCO₃ groups although sodium intake and output of NaCl group increased two or three times as much as that of NaHCO₃ group. Dietary chloride had very little effect on producing hypertension of SHR and it has less importance to hypertension of SHR than that of salt-sensitive model. These results suggest that sodium-salt has complex effects on the pathogenesis of hypertension.