Regional Blood Flow in Dahl-Iwai Salt Sensitive Rats and the Effects of Dietary L-arginine Supplementation. Hong He¹, Yoshihide Fujisawa², Atsufumi Tomohiro¹, Tomoyuki Kita¹, Yasuharu Aki¹, Shoji Kimura¹, Toshiaki Tamaki¹ and Youichi Abe¹
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The pattern of regional hemodynamic changes accompanying hypertension varies greatly with the form of hypertension. In hypertensive Dahl salt-sensitive (DS) rats, an increased renal vascular resistance has been reported, but the vascular resistance in the other organs has not yet been determined. It was also reported that L-arginine administration increased production of nitric oxide and prevented salt-sensitive hypertension in DS rats. The purpose of the present study is to determine 1) whether different organs undergo similar increases in vascular resistance in DS rats, and 2) the effects of chronic oral L-arginine supplementation on the regional hemodynamics in DS rats.

Male 5 week old Dahl-Iwai salt-sensitive rats and salt-resistant rats (Eisai Co., Tokyo) were maintained initially on tap water for 1 week. The diet was then changed to 8% NaCl chow for 4 weeks, and salt-sensitive rats were divided into two groups. One group(S) was continued on tap water maintenance. The other group(S/Arg) received tap water containing L-arginine at a concentration of 1.5%. Salt-resistant (R) rats were maintained on 8% NaCl chow. At the end of week 4, the urine for 24 hour was collected for the measurement of urinary protein and albumin excretion. After 4 weeks of treatment, organ blood flow and cardiac output were measured with microspheres in the conscious condition.

Mean blood pressure in S rats, S/Arg rats and R rats was 159 ± 5 , 138 ± 3 and 111 ± 4 mmHg, respectively. There were significant differences among groups. Cardiac output in S/Arg rats was higher than that in the other two groups. As a result, total peripheral resistance (TPR) in S/Arg rats was not different from that in R rats. Urinary excretion of protein and albumin in S/Arg rats were significantly suppressed when compared to S rats. Concerning the regional hemodynamics, the flow rate of the kidney was lower in S rats than in R rats, but there were no differences between S and R rats in the flow rates of the brain, heart, lung, liver, spleen, intestines, skeletal muscle and skin. Thus, the abnormality of renal blood flow was solely observed in S rats. The flow rate of the kidney in S/Arg rats was maintained at a higher level as compared to that of S rats. Thus, the supplementation of L-arginine normalized the abnormality of renal hemodynamics accompanying salt-induced hypertension.

Organ blood flow measurements indicate that all organ vascular beds contribute to increased TPR in S rats. However, reduced renal blood flow in S rats suggests a disproportionately larger increase in renal resistance in comparison to the relatively uniform increased resistance among the other organs. The supplementation of L-arginine prevented the increases of organ vascular resistance, especially the increase of renal vascular resistance. Thus, it is suggested that the disturbance in the production of nitric oxide may induce salt-sensitive hypertension and the abnormality of renal hemodynamics in the S rat.