

Table 2. Effects of Ang II, Ang (3-8), and Ang (1-7) Intracerebroventricular Infusion on Blood Pressure and Renal Function in WKYs

		Ang II	Ang (3-8)	Ang (1-7)
MABP (mm Hg)	Before	127 ± 4	124 ± 3	124 ± 4
	After	141 ± 5*	127 ± 3	127 ± 2
GFR (mL/min)	Before	1.9 ± 0.2	1.4 ± 0.1	1.5 ± 0.1
	After	1.9 ± 0.3	1.4 ± 0.1	1.6 ± 0.2
V̇ (uL/min)	Before	6.4 ± 0.4	6.8 ± 1.1	6.7 ± 1.0
	After	7.7 ± 0.7*	6.8 ± 0.8	7.0 ± 0.8
U _{Na} V̇ (uEq/min)	Before	0.20 ± 0.03	0.21 ± 0.05	0.28 ± 0.08
	After	0.26 ± 0.06	0.10 ± 0.02*	0.09 ± 0.03*
FE _{Na} (uEq/min)	Before	0.07 ± 0.01	0.11 ± 0.03	0.17 ± 0.05
	After	0.09 ± 0.02	0.05 ± 0.01*	0.07 ± 0.02*
U _K V̇ (uEq/min)	Before	1.11 ± 0.10	1.06 ± 0.15	1.12 ± 0.15
	After	1.60 ± 0.17*	1.03 ± 0.12	1.31 ± 0.17*
FE _K (uEq/min)	Before	15.2 ± 2.4	19.6 ± 2.6	20.2 ± 3.3
	After	22.8 ± 4.9*	19.1 ± 3.0	20.8 ± 3.0
T ^C _{H₂O} (uL/min)	Before	31.3 ± 1.8	35.6 ± 3.3	31.2 ± 3.3
	After	42.5 ± 3.9*	40.0 ± 4.3*	35.8 ± 4.1*

Results were recorded before and after 20 min intracerebroventricular infusion of 16.7 pmol Ang II, Ang (3-8), or Ang (1-7) per minute to the various groups. MABP = mean arterial pressure; GFR = glomerular filtration rate; V̇ = urine flow; U_{Na}V̇ = absolute sodium excretion rate; U_KV̇ = absolute potassium excretion rate; FE_{Na} = fractional sodium excretion rate; FE_K = fractional potassium excretion rate; T^C_{H₂O} = free water reabsorption rate. Values are means ± SE., *p < 0.05 as compared with control (before).

fusional doses of Ang II, (3-8), and (1-7). Central infusion of Ang II to SHR_s significantly increased MABP and augmented the renal excretions in urine flow, absolute and fractional potassium excretion rates, and free water reabsorption rates. Neither Ang (3-8) nor (1-7) administration changed MABP, but these angiotensins decreased absolute and fractional sodium excretion rates and increased free water reabsorption rates.

The averaged wet weights of left and right kidneys were 1.39 ± 0.20 and 1.42 ± 0.20 g in WKY_s and 1.39 ± 0.19 and 1.40 ± 0.18 g in SHR_s, respectively. There were no significant differences in kidney weights between the 2 rat groups. Vehicle control using saline injection or infusion did not change MABP, drinking, or renal excretions intravenously or intraventricularly.

DISCUSSION

Spontaneously hypertensive rats (SHR_s) demonstrate numerous abnormalities in the regulation of blood pressure and are widely used to mimic essential hypertension in humans.²² In this study, SHR_s did pos-

sess higher blood pressure than the control WKY_s (Table 1). Intravenous injection of angiotensin II increased MABP in both SHR_s and WKY_s. Based on previous assumption that alterations in angiotensin II receptor number, reactivity, and responsibility may be the basis of the role of angiotensin II in increased vascular reactivity of this hypertensive model,^{23,24} it was predicted that the pressor response of angiotensin II in SHR_s should be higher than that in WKY_s. However, in this experiment no difference was observed in MABP change by neither intravenous nor intracerebroventricular injection of angiotensin II between WKY_s and SHR_s (Figs. 1 and 2). Furthermore, no differences in water intake caused by central administration of angiotensin II between these 2 rat groups were found (Fig. 3). It is possible that there is genetic diversity in WKY_s, which may lead to variable pressor responses to angiotensin II.

Most of the brain regions are separated from the blood-borne renin-angiotensin system (RAS) by the blood-brain barrier (BBB). It is well known that Ang II can not diffuse across BBB. Several studies have shown that peripheral and central administrations of