

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

		Ang II	Ang (3-8)	Ang (1-7)
MABP (mm Hg)	Before	156 ± 6	153 ± 5	160 ± 6
	After	171 ± 7**	151 ± 5	161 ± 4
GFR (mL/min)	Before	1.3 ± 0.2	1.2 ± 0.2	1.7 ± 0.3
	After	1.6 ± 0.3*	1.2 ± 0.2	1.9 ± 0.4
V̇ (uL/min)	Before	5.1 ± 0.5	5.5 ± 0.7	4.9 ± 0.5
	After	7.7 ± 1.2*	5.6 ± 0.5	5.2 ± 0.4
U _{Na} V̇ (uEq/min)	Before	0.13 ± 0.02	0.10 ± 0.03	0.11 ± 0.03
	After	0.23 ± 0.11	0.06 ± 0.02*	0.08 ± 0.03*
FENa (uEq/min)	Before	0.11 ± 0.04	0.08 ± 0.03	0.08 ± 0.02
	After	0.23 ± 0.16	0.03 ± 0.01	0.04 ± 0.01*
U _K V̇ (uEq/min)	Before	0.72 ± 0.11	0.76 ± 0.06	0.82 ± 0.11
	After	1.06 ± 0.13*	0.91 ± 0.12*	1.10 ± 0.19*
FE _K (uEq/min)	Before	13.4 ± 3.4	16.0 ± 2.7	15.6 ± 2.9
	After	27.7 ± 10.9*	19.9 ± 2.5*	16.5 ± 3.7
T ^C H ₂ O (uL/min)	Before	19.5 ± 2.9	18.3 ± 1.4	22.2 ± 3.0
	After	33.9 ± 3.9*	26.2 ± 3.5*	29.0 ± 2.7*

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; FENa = fractional sodium excretion rate; FE_K = fractional potassium excretion rate; T^CH₂O = free water reabsorption rate. Values are means ± SE., *p < 0.05 as compared with control (before).

angiotensins would cause differently physiological effects.²⁶ In the present study, intravenous or intracerebroventricular injections of Ang (1-7) or (4-8) caused no significant changes in MABP or water intake in either SHRs or WKYs. Intravenous injection of Ang (3-8) also caused no significant changes in MABP or water intake in either rat group. On the other hand, intracerebroventricular injection of Ang (3-8) caused similar pressor but a lower drinking response than did angiotensin II in both rat groups (Figs. 1, 2, and 3). At least 2 distinct subtypes of the Ang II receptor (AT₁, AT₂) have been demonstrated.²⁷ Most of the functions of Ang II are mediated via AT₁ receptors.²⁸ Tonnaer et al.⁶ believed that the C-terminal part of the octapeptide chain contains the sequence which is necessary for biological activity. The presence of phenylalanine in position 8 is essential for receptor activation.⁶ Hence, the central pressor and dipsogenic effects of Ang (3-8) might also be mediated through the angiotensin II receptors.

The maintenance of sodium and water homeostasis by the kidneys is widely believed to be the primary long-term determinant of systemic arterial pressure.

An abnormal renal function is critical for the initiation, development, and maintenance of hypertension.²⁹ In Table 1, SHRs show lower basal renal function indices than do WKYs. The present data agreed well with others and suggested that the kidney dysfunction might contribute to high blood pressure in SHRs. It is well known that the central administration of Ang II induces elevation of blood pressure and natriuresis.³⁰ In the present study, we observed that intracerebroventricular infusion of Ang II induced diuresis and natriuresis in both WKYs and SHRs (Tables 1 and 2). It has been suggested that Ang II-induced diuresis may be due to redistribution of the renal plasma flow,³¹ and natriuresis may be due to inhibitions of aldosterone secretion and renal nerve activity.^{25,32} The present results also show that the free water reabsorption rate increased with the intracerebroventricular infusion of angiotensin II. This increment is speculated to be associated with the secretion of vasopressin.^{33,34}

It has been assumed that the actions of the renin-angiotensin system in the brain and periphery arise from interactions of Ang II with its receptors.³⁵ However, increasing evidence indicates that the actions of