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Acute Effects of Captopril and Enalapril on Blood Pressure, and Urinary Excretions of Kinins and Electrolytes in Stroke-Prone Spontaneously Hypertensive Rats

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〔IUPHAR 9th International Congress of Pharmacology (July, 1984, London, England) で発表〕

The mechanism of antihypertensive action of the converting enzyme inhibitor (CEI) was investigated in 12-week-old stroke-prone spontaneously hypertensive (SHRSP) rats, with particular reference to the role of the renal kallikrein-kinin (KK) system. Captopril (30 mg/kg) and enalapril(10 mg/kg)administered orally for 7 days produced an almost the same degree of significant reduction in blood pressure. No significant changes in urinary excretion of kinins were observed throughout the treatment with the CEI. Urine volume, urinary excretions of sodium and potassium were not changed by these CEI. Similar results were observed in the SHR. Thus, the antihypertensive action of these CEI could be mainly due to the decrease in angiotensin II and potentiation of the renal KK system does not seem to play any significant role. Enalapril is about 3 times as potent as captopril in antihypertensive action in SHRSP rats.

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Roles of Vasopressin and Renin-Angiotensin System in the Physostigmine-induced Hypertension in Spontaneously Hypertensive Rats

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The roles of vasopressin (AVP) and renin-angiotensin (RA) system in the pressor effect of physostigmine were examined in conscious unrestraint spontaneously hypertensive (SHR) and Wistar Kyoto (WKY) rats. The pressor response began within 3 min after physostigmine (100 μg/kg, ia) administration and attained the maximun