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

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

by 6 min. Blood pressure (BP) returned to control level within 30 min. The maximal pressor response was significantly higher in SHR than in WKY rats (40 vs. 25 mmHg). Heart rate in the SHR was increased by physostigmine while it was decreased in the WKY. In the both strains, $d(CH_2)_5Tyr(Me)AVP$, an antagonist of vasopressor action of AVP, did not affect both basal BP and pressor effect of physostigmine. Plasma renin concentration was decreased by physostigmine when the pressor response was maximum, and was comparable to control value at 15 and 30 min. These data indicate that AVP and RA system do not have roles in the pressor effect of physostigmine in SHR and WKY rats.

Cardiovascular Effects of Bunitrolol and Propranolol in Spontaneously Hypertensive Rats at Rest and during Stress

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

Effects of bunitrolol (BNL) (5 mg/kg, po) and propranolol (PPL) (5 mg/kg, po) on mean arterial pressure (MAP) and heart rate (HR) were studied in conscious unrestraint spontaneously hypertensive rats. BNL caused a gradual fall in resting MAP which was significant at 3 and 4 hr, and a significant increase in resting HR for the first 2 hr which returned to the control level by 3 hr. PPL did not produce any significant changes in both resting MAP and HR. Handling stress loaded by lifting the rat by the tail for 30 s induced marked rises in MAP and HR. BNL and PPL caused a significant reduction in hypertensive response to stress only at 4 hr. Tachycardia due to stress was markedly supressed by these drugs throughout the experiment. It takes some time for β-blocker to fully develop an antihypertensive action. The early onset of the decrease in resting MAP after BNL may be due at least in part to the intrinsic sympathomimetic action.

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