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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, $d(\text{CH}_2)_5\text{Tyr}(\text{Me})\text{AVP}$, 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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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 unrestrained 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 suppressed 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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