15) Hemodynamics on the intraventricular administration of norepinephrine in SHR.
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Central nervous system (CNS) has been shown to play an important role in the pathogenesis of hypertension in SHR. We have been demonstrating the existence of the adrenergic pressor mechanism of CNS in SHR, after administration of clonidine into vertebral artery (Jpn Circul J 47: 970 1983). We have investigated the pressor adrenergic mechanism using intra-cerebroventricular (ICV) infusion of norepinephrine (NE) in SHR.

Materials and Methods:
Eleven to fourteen WKS old male SHRs were anesthetized with urethane and alpha-chloralose. A catheter was introduced into the carotid artery and fixed subcutaneously for the measurement of blood pressure (BP) and heart rate. A microcatheter was inserted into the cerebroventricular to administrate NE 1.0μg, phenoxybenzamine (POB) 25μg or tolazoline 10-20μg with saline 1-2μl.

Results:
ICV administration of 1μg of NE increased BP by 5.4% and bradycardia appeared. While this BP increase was inhibited by 3.5% and heart rate was not changed after the pretreatment of POB. The same dose of ICV administration of NE further increased BP by 7.9% and heart rate after the pretreatment of tolazoline, significantly.

Discussion and Conclusions:
There are some reports concerning the localization of pressor and depressor areas in the CNS (In Central Nervous System Mechanisms in Hypertension; 61-72, 1981). We reported the existence of the pressor noradrenergic receptors in the CNS (IXth World Congress of Cardiol, Abst II: 339). In SHR, BP was increased significantly when subpressor dose of NE was given into vertebral artery, while BP was decreased when the same dose of NE was infused into the carotid artery. These results suggest that the noradrenergic receptors are differently distributed among the peripheral vessels and CNS, and there are differences in the distribution of noradrenergic receptors in CNS between the areas perfused through vertebral artery and those through carotid artery in SHR. Biphasic response was shown after administration of clonidine into the vertebral artery (Jpn Circul J 47: 970 1983) the initial pressor effect was inhibited by α1-blocker and the following depressor are by α2-blocker, demonstration the existence of the pressor α-adrenergic receptors of CNS in SHR. The purpose of the present study was to clarify further the existence of pressor noradrenergic receptors of CNS in SHR. Elevation of BP caused by the ICV injection of NE was inhibited by the pretreatment of POB, and accelerated by the pretreatment of tolazoline, possibly indicating that elevation of BP was induced by α1-adrenergic receptors and the depressor effect was through α2-adrenergic receptors in CNS. Elevation of BP induced by NE was not inhibited even after the pretreatment of POB in some SHRs, also suggesting the possible participation of β-adrenergic receptors in CNS.