Objective: We investigated the cardiovascular effects of ICV administered GRF in conscious rats, which has never been reported, referring to the involvement of adrenergic mechanisms.

Design and Method: Male SHR and wistar rats (WR) aged 12 weeks were used. Lateral ventricular cannulae was implanted under pentobarbital anesthesia. Seven days after cannulation, mean arterial pressure (MAP) was monitored through a right femoral artery cannulation. GRF was injected into the ventricle in a volume of 2.0μl.

Results: 1) The ICV GRF (0.1, 0.3, 2.0 nmol/kg) induced biphasic change of MAP in SHR. In the first phase, maximum pressor response was observed [7±1, 13±2, 21±2 mmHg(mean±SE, n=6)] 2-3 min after injection. In the second phase, maximum depressor response was observed [-11±2, -26±2, -46±3 mmHg] 12-13 min after injection, and lasted up to 21-22 min. In WR, there were no changes in MAP. 2) Pretreatment with ICV phentolamine 200μg/kg attenuated the GRF induced pressor response [23±2 to 8±2 mmHg, p<0.01], but did not affect the depressor response. 3) Pretreatment with ICV bunazosin 50 μg/kg had no effect in both responses. 4) Pretreatment with ICV yohimbine 20 μg/kg attenuated the GRF induced pressor response [23±2 to 9±2 mmHg, p<0.01], but did not affect the depressor response.

Conclusion: 1) It is indicated central GRF plays an important role in the maintenance of hyper-tension in SHR, since ICV GRF caused a significant depressor effect in SHR, but not in WR. 2) The depressor effect induced by ICV GRF was larger than that induced by any other peptide hormones, reported previously. 3) ICV GRF induced biphasic change of MAP in SHR. The pressor response was mediated through central α 2-adrenoceptors. The depressor response was not mediated through central α-adrenoceptors, other mechanisms considered to be attributable.