67) Centrally Mediated Hemodynamic Effects of Angiotensin-II Infused into the Vertebral Artery in Spontaneous Hypertensive Rat (SHR). Toshiyuki Okazaki, Nagao Kajiwara, Masayuki Koshino, Takashi Sasaki, Minoru Kubota, Katsuichiro Sakai, Hirobumi Ueda, Haruka Kanmatsuse, Fumio Sugiura, Kazushige Tsujikawa, Yuichi Nagano, Mitsuaki Higurashi and Michinobu Hatano. Department of Cardiology, Surugadai Nihon University Hospital, Tokyo 101.

There are many reports that the central nervous system plays an important role in the pathogenesis of hypertension in SHR and angiotensin-II is known as a potent direct constriction of peripheral arterioles. The present study was designed to determine the effects of angiotensin-II infusion in the vertebral artery and to investigate whether the sympathetic nervous system is concerned with pathogenesis of hypertension in SHR or not.

MATERIALS AND METHODS;

Fourteen- to twenty-week old male SHR (200-300 gm in weight) and age matched Wistar Rat (WR) were anesthetized with light-ether, alpha-chloralose and pancronium. Under controlled ventilation, the chest was opened and small dose of angiotensin-II (1-5 ng/100 gm) or saralasin (55 ng/100 gm) was infused through catheter which was inserted into the right vertebral artery. An electromagnetic flow probe was placed around the thoracic aorta for measuring cardiac output (CO). A catheter was inserted into the femoral artery and then advanced to the central aorta for measuring blood pressure (BP). Left ventricular-dp/dt (LV-dp/dt) was recorded through the catheter inserted into the left ventricular cavity by direct puncturing. BP, CO, LV-dp/dt, total peripheral resistance (TPR) and heart rate (HR) were measured before and after the intravertebral infusion of angiotensin-II. Both SHR and WR were divided into three groups; group A (angiotensin-II infusion into vertebral artery), group B (angiotensin-II into vertebral artery after intravertebral saralasin) and group C (angiotensin-II into vertebral artery after intravenous saralasin). Student's t-test was used for the statistical analysis.

RESULTS;

In group A, BP, TPR and LV-dp/dt increased higher in SHR than those of WR, but CO and HR responses were similar in SHR and in WR. In group B, BP response was abolished in both SHR and WR. In group C, BP increased in both SHR and WR, but the response was slightly lower as compared with that of group A.

CONCLUSION;

Abnormalities of catecholamine metabolism in the brain stem of SHR have reported and it has been shown that the pressor responses may be possibly related to the central nervous system when angiotensin-II was infused via the vertebral artery. The results are as follows; 1) central administration of angiotensin-II caused acceleration of sympathetic tones, and 2) These effects were abolished by saralasin administration via the vertebral artery, but not by saralasin via the peripheral vein. Angiotensin-II infusion into the vertebral artery elicited an increase in central sympathetic discharge. Therefore, we suggest that the region of central nervous system which supplied by the vertebral artery may play an important role in the pathogenesis of hypertension in SHR.