13) Effects of vanadate on membrane potential and contraction of portal vein from SHRSP and WKY. Keiichi Shimamura, Tomoko Shimada, Kazuo Yamamoto and Satoru Sunano. Research Institute of Hypertension, Kinki University, Osaka, 589.

Portal vein is the only one vessel which has spontaneous activity. Differences in spontaneous contraction and changes in structure and sensitivity to several agonists have been reported in portal vein of SHR but still controversial. Recently, a Na,K-ATPase inhibitor, sodium vanadate (NaVO_3) was found to induce contraction in vascular smooth muscles and postulated to contribute, somehow, to the initiation of hypertension. These contractile effects have been examined mostly in arterial smooth muscle and it has been revealed that NaVO_3 causes a change in Ca mobilization without relation to Na,K-ATPase inhibition. In the present study, spontaneous phasic contraction of portal vein was compared between SHRSP and WKY and effects of NaVO_3 on the contraction were examined.

Six-month old SHRSP and WKY of either sex were used. Each isolated portal vein was cut along its axis into four strips and isometrical tension of longitudinal muscle was measured in modified Tyrode's solution. Membrane potential was measured by means of microelectrode method. In portal vein from both rats, spontaneous phasic contraction was observed but tension and frequency of the contraction were significantly different. Tension of the contraction was approximately 170 and 60 mg and frequency of the contraction was 0.8 and 1.3 cpm in SHRSP and WKY, respectively. Increased tension of the spontaneous contraction in SHRSP was compatible with increase in 140 mM-K induced contraction. When NaVO_3 was applied to the solution, spontaneous contraction force was augmented dose dependently in both rats, the augmentation being greater in WKY portal vein than SHRSP one. Contraction frequency increased dose dependently in WKY but not in SHRSP. Duration of each phasic contraction was increased in the presence of NaVO_3 but no tetanic contraction was observed. Recording of membrane potential revealed that resting membrane potential did not alter significantly but number of action potential in the spike burst which corresponds to each contraction increased after NaVO_3 application. Since effect of NaVO_3 was not affected by phentolamine, the effect was presumed not to be adrenergic nerve mediated one. To confirm Na,K-ATPase inhibitory action of NaVO_3 , effect of ouabain was tested. Ouabain increased contraction frequency markedly and rather decreased spontaneous contraction height. Since Na,K-ATPase inhibition can be expected to depolarize membrane, the effects of depolarization by elevated K were studied. Elevation of K concentration enhanced spontaneous contraction force but increased frequency and induced tetanic contraction. Tetraethylammonium (TEA) and 4-aminopyridine (4-AP) was also tested and these drugs enhanced contraction and prolonged duration of each contraction. 4-AP induced contraction frequency increase in WKY portal vein but not in SHRSP. Mode of increase in contraction by TEA and 4-AP was very similar to that of NaVO_3 and response of SHRSP portal vein to TEA and 4-AP was smaller than WKY as that was in NaVO_3 .

These results indicate 1) NaVO_3 enhanced spontaneous contraction of portal vein through mechanisms other than inhibition of Na,K-ATPase. 2) The mode of the action was similar to those of TEA and 4-AP and was thought to be related to the inhibition of K-conductance. 3) Spontaneous contraction of portal vein from SHRSP was different from WKY and some mechanisms related to K-conductance might contribute to the difference.