It is reported that Ca influx in the aorta from SHRSP was accelerated and the excitability of sympathetic beta receptor was lowered, while it is not clear whether these changes are secondary to hypertension or due to the hereditary factor. The authors investigated the accelerated Ca mobilization and the lowered excitability of sympathetic beta receptor in the isolated portal vein from SHRSP, from the standpoint of influences of some spasmolytics on the responses to the smooth muscle contractile agents.

Materials and Methods: One year old SHRSP and WKY were used. The isolated portal veins from SHRSP and WKY were mounted in an organ bath containing Locke solution maintained at 30°C. The change in tonus by the drugs was recorded isometrically. The concentrations of the agonists used in the present study were approximately ED 80.

Results and Discussion: Aminophylline and papaverine inhibited the responses to the agonists, K⁺, Ba²⁺, ACh and noradrenaline in a dose-dependent manner. The inhibition was larger in WKY than in SHRSP. It is well known that these spasmolytics produce the relaxation by increasing cAMP which resulted from the inhibition of phosphodiesterase. Thus, these facts suggest that in the portal vein from SHRSP the activity of phosphodiesterase is accelerated and/or the sensitivity to cAMP is lowered. A sympathetic beta stimulant, fenoterol inhibited the responses to the agonists in a dose-dependent manner. The inhibition was larger in WKY than in SHRSP. It is well known that sympathetic beta stimulant increases cAMP by activating adenyl cyclase, producing the relaxation. The weaker inhibition in SHRSP suggests that the activity of adenyl cyclase in SHRSP is lower than in WKY. Dibutryryl cAMP inhibited the responses to the agonists in a dose-dependent manner. The inhibition was larger in WKY than in SHRSP. This result suggests that the sensitivity to cAMP in SHRSP is lower than in WKY. The weaker inhibition observed in SHRSP compared with WKY may be due to the decreased sensitivity to cAMP. However, the thoughts that the decreased phosphodiesterase activity may participate in the decreased inhibition observed in SHRSP might not be denied. Diltiazem inhibited the responses to the agonists in a dose-dependent manner. The inhibition was larger in SHRSP than in WKY. It is well known that Ca antagonist selectively inhibits Ca influx, and thus this result suggests that Ca influx is accelerated more strongly in SHRSP than in WKY.

The results obtained here agree with the previous report in the aorta. The change in reactivity of the blood vessel in SHRSP may not be secondary to the high blood pressure, but may be due to the hereditary factor.