The Perfusion Pressure in the Tail Artery of SHRS and M-SHRSP.
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There are many reports on functional changes in isolated vascular smooth muscles of spontaneously hypertensive rat (SHR). Increase of peripheral resistance in SHR is explained by structural abnormality and functional abnormality such as an enhancement of receptor mediated contraction in resistance vessels. In tail artery of SHR, enhanced response to noradrenaline (NA) has been shown using helical strip and perfusing whole tail arteries, but it is still controversial. In the present study, perfusing pressure at different flow rate and responses to contractile agents were studied using the tail of stroke-prone SHR (SHRSP), malignant SHRSP (M-SHRSP) and were compared with those of WKY.

MATERIALS AND METHODS: Male M-SHRSP of 12 weeks, SHRSP of 12 and 20 weeks of age and age matched WKY were used. Blood pressure was measured by the tail-pulse pickup method without anesthesia. Tail was dissected under anesthesia with ether and tail artery was cannulated with polyethylene tube. The preparations were perfused with Tyrode's solution by a constant flow roller pump. The perfusion pressure was monitored by a pressure transducer. The concentration-response curve for NA-induced pressure elevation was obtained by increasing NA concentration in perfusing solution. The endothelium-dependent pressure reduction was observed by applying 10^{-6}M acetylcholine (Ach) to the preparation during the increase in pressure by 10^{-6}M NA.

RESULTS: The systolic blood pressure of animals at the age of 12 weeks was 269 ± 1.9, 226 ± 1.6 and 132 ± 1.1 mmHg in M-SHRSP, SHRSP and WKY respectively. At the age of 20 weeks, it was 256 ± 1.3, 135 ± 1.2 mmHg respectively in SHRSP and WKY. The blood pressure of M-SHRSP and SHRSP was significantly higher than that of WKY. When flow rate was increased from 0.3 to 6 ml/min, the perfusion pressure increased flow rate dependently. The increase was greater in the tail of hypertensive rats than that of WKY at both age. There was no remarkable difference in the response to K 80 mM between hypertensive rats and WKY. NA induced an increase in perfusion pressure concentration dependently. At 12 weeks of age, there was no difference in maximal response between preparations from hypertensive rats and WKY. However, sensitivity to lower concentration of NA was higher in tail of hypertensive rats resulting in leftward shift of concentration-response curve. When Ach was applied, pressure increased by NA was reduced in tail from all strain of rats, probably through endothelium dependent mechanism. Ach-induced pressure decrease was smaller in the tail from hypertensive rats than that from WKY. The pressure reduction induced by acetylcholine decrease with age in SHRSP.

CONCLUSION: Increase in perfusion pressure in M-SHRSP and SHRSP may indicate structural change in tail vascular bed. Increase in the sensitivity to NA in hypertensive rats at 12 weeks indicated facilitation in α-receptor mediated response in the animals. The pressure reduction induced by acetylcholine was impaired in hypertensive rats. These results suggest that increased perfusion pressure, greater sensitivity to NA and impairment of endothelium-dependent pressure reduction play important roles in the development and maintenance of hypertension.