12) Structural Vascular Alteration in Relation to Cardiac Hypertrophy in SHR. Yukio Yamori[°], Masahiro Kihara^{°*}, Shigeharu Fujishin^{*}, Yasuo Nara^{°*} and Akira Ooshima[°]. Japan Stroke Prevention Center^{*}, Department of Pathology[°], Shimane Medical University, Izumo 693.

INTRODUCTION

Structural vascular changes are not only secondary phenomena in hypertension but regarded as pathogenic process for stabilizing or accelerating hypertension. However, the direct estimation of structural vascular changes is not available especially with noninvasive technique. Our previous studies in SHR have indicated that there seems to be a close relationship between cardiac hypertrophy and structural changes of coronary arteries (YAMORI et al.: AM J CARDIOL 44:964, 1979). Since cardiac hypertrophy is now clinically detectable in the early stage, it may be a useful indicator for structural vascular changes. Therefore, this experiment was designed to study structural changes of the aorta, coronary and intratesticular arteries in hypertension in relation to the heart weight.

MATERIALS AND METHODS

Thirty male stroke-prone spontaneously hypertensive rats (SHRSP) and 30 male Wistar-Kyoto rats (WKY) were divided into 3 groups, young (5 SHRSP: 42 ± 0 days, 6 WKY: 41 ± 0 days), adult (6 SHRSP: 143 ± 18 days, 5 WKY: 133 ± 1 days) and old (6 SHRSP: 336 ± 15 days, 6 WKY: 315 ± 1 days). All rats were sacrificed by the intravenous injection of KCl solution (1 mEq/ml) to arrest the heart at the similar status. Hearts and testes were extirpated to measure their absolute weights and to calculate organ to body weight ratios. After fixation with 10% formalin, hearts and testes were transversely cut at the middle of the long axis for making histological sections under well controlled conditions. Then in H-E stained sections the transectional areas of coronary and testicular arteries and their lumens were measured microscopically by a quantitative picture analysis system, MOP-AM03 (KONTRON), and the ratio of luminal area to transectional area of the vessel was calculated as an index of arterial wall thickening. Similarly in 12 SHRSP, 13 SHRSR and 12 WKY all over 4 months of age, the weight of cleanly dissected right and left ventricles were weighed and the aorta from the orifice of a left subcravian artery to the level of a right renal artery was neatly dissected to measure the weight and the length, and the weight of the unit length was calculated. Data were analyzed by Student's t-test.

RESULTS AND DISCUSSIONS

Coronary arteries showed a wall thickening especially in the media, which was microscopically observed in adult and old SHRSP, but no arterionecrosis was noted even in old SHRSP. On the other hand, the wall thickening with arterionecrosis of intratesticular arteries was frequently found in old SHRSP but the grade was not so marked in adult SHRSP. Significant inverse correlations between the heart weight (Y:g) and the ratio of luminal area to transectional area (X) were observed both in the coronary ($Y = -7.61X + 2.76$, $r = -0.807$, $p < 0.001$) and intratesticular arteries ($Y = -6.35X + 2.58$, $r = -0.696$, $p < 0.01$), respectively.

Further, a highly significant correlation between ventricular weights (Y:g) and the unit weight of the aorta (X:mg/mm) indicating arterial wall thickening was proved in 3 groups of adult rats ($Y = 4.44X + 0.75$, $r = 0.640$, $p < 0.001$). Ventricular and aortic weights were significantly greater in SHRSP than in WKY and those in SHRSR showed values between SHRSP and WKY as shown in Table. Therefore, our present study indicates that cardiac hypertrophy is regarded as a good index of arterial wall thickening in hypertension.

Strain (No. of rats)	Organ Weight (Mean $\pm$ SE)	
	Ventricles (g)	Aorta (mg/mm)
SHRSP (12)	$1.573 \pm 0.056^{**}$	$16.4 \pm 0.4^{**}$
SHRSR (13)	$1.391 \pm 0.045^*$	14.7 ± 1.2
WKY (12)	1.221 ± 0.063	12.6 ± 0.4

*, **: Significant differences from WKY ($p < 0.05$, $p < 0.001$)