INTRODUCTION

It was repeatedly reported that high blood pressure causes vasoconstriction of retinal and cerebral arterioles in stroke-prone spontaneously hypertensive rats (SHRSP) and inversely, attenuation of high blood pressure results in regression of such vasoconstriction. And it was also demonstrated that such reversible vasodilatation can not be observed around the age of 7 to 8 months in both retinal and cerebral arterioles, although a degree of the effect of blood pressure attenuation on retinal arterioles does not well correspond to that on cerebral arterioles. And, as one of reasons for such a discrepancy, a difference in originating arteries to retinal and cerebral arteries was suggested based on an experimental evidence, i.e., remarkable differences in atherogenesis between them by Horie et al., 1978. Therefore, in the present study, changes of retinal vessels in SHRSP at the more advanced stage was investigated in correspondence with blood pressure measurement before and after antihypertensive treatment.

MATERIALS AND METHODS

Fourteen SHRSP (6 males and 8 females, F61, A3 strain) were used. At the age of 13 months, an antihypertensive treatment (apresoline, 80 mg/dl) was performed. Blood pressure measurement (tail-pulse pickup method) and optic fundi photography by a fundus camera (RC-II, Kowa Co.) were repeatedly carried out without anesthesia during the 3 periods, i.e., before the antihypertensive treatment, and one week and 2 months after the treatment. Retinal arteriole (A) to venule (V) ratios were obtained by the strictly unformed measurement of enlarged vessels on a screen. Further, external and internal carotid arteries were dividedly traced, under a neurosurgical microscope. And an effect of selective external or internal carotid artery ligation on blood flow in retinal arterioles was alternatively examined by a optic fundi photography.

RESULTS AND DISCUSSION

Blood pressure after the treatment was significantly lower than that before the treatment (p<0.01), and A/V ratios after the treatment were rather greater than those before the treatment in both males and females as shown Table. On the other hand, it was clarified that retinal artery and/or arterioles were derived from, mainly, ipsilateral external carotid artery and partly, ipsilateral internal carotid artery and contralateral carotid artery in contrast to cerebral arterioles from internal carotid arteries.

These results suggest that above-mentioned discrepancy may be partly caused from such fundamental difference in their vasculature. Although further precise mechanisms such as vascular neural control (Horie et al: CLIN EXP HYPERTENSION 2: 1115, 1980) were remained unsolved, it is fact that retinal angiopathy dose not always well correspond to suspected pathological changes of cerebral arterioles such as lenticulostriate arterioles from the clinical point of view.