Although the morphological difference of the cerebral vasculature between NTR and SHR is minimal, vasodilatory response of cerebral arteries to the lowering of MAP seems insufficient in SHR, resulting in the lower limit of autoregulation to be shifted to 95 mm Hg of MAP, which is apparently higher than that in NTR.

The lower limit of autoregulation is also altered by anesthetic or changes in $PaCO_2$[9,10]. Hypercapnia may totally impair the autoregulation or raise the lower limit upwards, whereas hypocapnia may have an adverse effect on the autoregulation.

In our previous studies, bilateral carotid artery occlusion caused an extremely high mortality[11], a marked increase in anaerobic glycolytic metabolites of the brain[12], and diffuse-extensive cerebral infarcts in SHR[8], while a lower mortality, a minimal increase in metabolites and small-circumcribed infarcts were observed in NTR. These biochemical and histological changes following bilateral carotid occlusion in SHR seem attributed to the hemodynamic difference rather than the morphological difference of the cerebral arteries between two groups. It is likely that cerebral perfusion pressure following carotid occlusion might fall below the lower limit of autoregulation in SHR, but not in NTR.

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Carotid Back Pressure Following Bilateral Carotid Occlusion in Normotensive and Spontaneously Hypertensive Rats

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Summary. In SHR, CBP fell markedly and remained below 50 mm Hg after carotid occlusion despite SBP being elevated, while in NTR changes in CBP were related with changes in SBP. Vascular resistance of the brain in hypertensive rats is discussed.

Bilateral carotid artery occlusion causes a diffuse and extensive cerebral infarction in spontaneously hypertensive rats (SHR)[1], resulting in a great increase in lactate and lactate-pyruvate ratio[2], and a high mortality[3], whereas in normotensive rats (NTR) it causes a minimal metabolic and histological change of the brain. These observations suggest that cerebral perfusion pressure following bilateral carotid occlusion might fall by a greater extent beyond the lower limit of cerebral autoregulation in SHR than in NTR, with consequently severe ischemic changes of the brain. In the present study, carotid back pressure as an index of cerebral perfusion pressure was measured before and after carotid occlusion in NTR and SHR.

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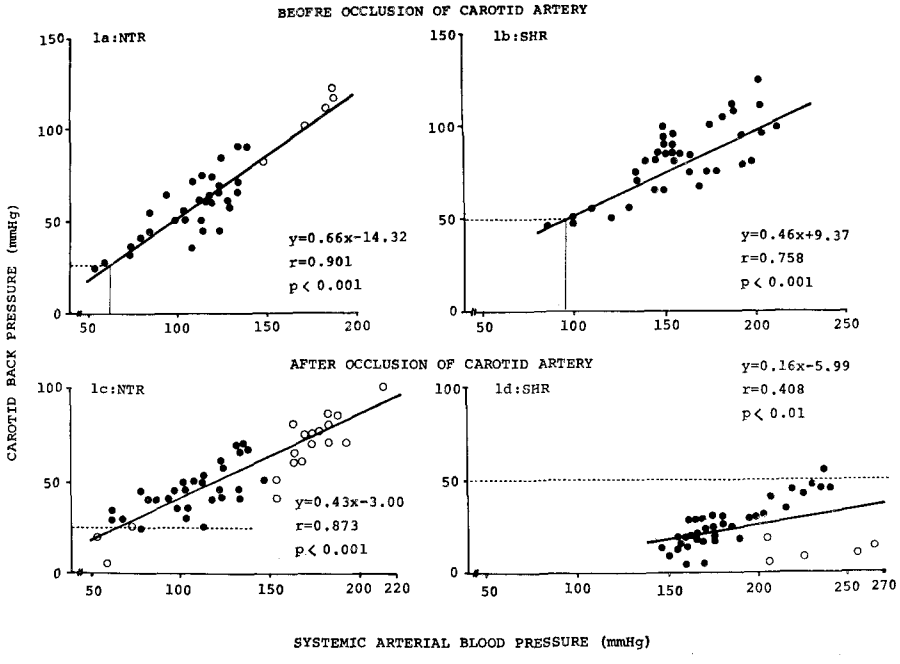

Fig. 1. Correlation between carotid back pressure and systemic blood pressure before and after bilateral carotid artery occlusion in normotensive rats (NTR) and spontaneously hypertensive rats (SHR). Open circle indicates systemic blood pressure being changed by drugs or bleeding. A dotted line designates a critical carotid back pressure level calculated from the lower systemic blood pressure limit of cerebral autoregulation (a solid line).

Methods. 7 NTR and 7 SHR rats, weighing 300 to 420 g, were anesthetized with i.p. amobarbital of 10 mg per 100 g of body weight. 1 femoral artery was cannulated for blood pressure recording with an electromanometer and blood sampling. Bilateral carotid arteries were dissected free from the vagosympathetic trunk in the neck. 1 carotid artery was cannulated upwards to the brain with PE 50 tubing, connected with a pressure transducer for recording of carotid back pressure. The contralateral carotid artery was prepared for clipping or ligating during the experiment.

One half ml of arterial blood was obtained before and after carotid occlusion for determination of pH, PCO_2 an PO_2 by IL meter of Model 113. In some NTR and SHR, systemic blood pressure was raised or lowered by i.p. hypertensive agents with noradrenaline, hypotensive drugs with phentolamine, or bleeding the animal.

Results. Before occlusion of carotid artery contralateral to the cannulated one, there was a highly significant correlation between systemic blood pressure (SBP) and carotid back pressure (CBP) in both NTR and SHR as shown in Figure 1a and b, although a slope of the regression equation was greater in NTR than in SHR, implying that change in CBP was 66% of SBP in NTR and 46% in SHR, respectively.

Following occlusion of the contralateral carotid artery, as shown in Figure 1c, there was a highly significant relationship between 2 parameters in NTR despite percentile change in CBP decreasing to 43% of SBP. Figure 2a depicts the actual recording of CBP and SBP in one of NTR. CBP did transiently fall immediately after occlusion, followed by a gradual rise as related with changes in SBP.

In SHR, Figure 2b shows that CBP dropped by a great extent from 100 to 30 mm Hg following carotid occlusion, while SBP rose from 200 to 230 mm Hg. Then CBP rose slightly, but never above 50 mm Hg despite SBP remaining at high level. As shown in Figure 1d, CBP in most SHR fell markedly below 50 mm Hg and did not rise or respond to the further increase in SBP by hypertensive agent administration. Change in CBP was 16% of SBP in these rats.

Discussion. The configuration of the circle of Willis is essentially identical in both NTR and SHR. However, the diameter of the posterior communicating artery and the basilar artery is somewhat smaller in SHR than in NTR, the difference being insignificant[1]. CBP seems reflecting the pressure of collateral circulation through the posterior and the anterior communicating artery before occlusion of the contralateral carotid artery, and through the posterior communicating artery only after occlusion. CBP following occlusion remained above a certain level in NTR, while it remained lower and responded little to the drug-induced further rise in SBP in SHR. These results strongly suggest that vascular resistance of the circle of Willis and the vertebrobasilar system might be higher in SHR than in NTR, resulting in a greater reduction of CBP following occlusion in SHR.

A pressure difference between aorta and intracranial arteries is varied depending on species. There is no reduction of pressure in the major cerebral artery in humans, despite a 17% fall in pial artery[4]. Arterial pressure in a large branch of the middle cerebral artery is lower than aortic pressure by 10% in dogs and 13% in monkeys[5], while basilar arterial pressure in cats falls by 10–30%[6]. DIECKHOFF and KANZOW[7], who measured small parietal pial pressure in cats, have demonstrated that mean blood pressure of pial artery ranging from 70–85 to 30–40 μm in diameter is between 59.5 and 53.3 mm Hg, or 73% and 61% of aortic pressure. It is suggested that a great reduction of pressure could occur in relatively large arteries in small animals, and in a sustained hypertension, a pressure fall might be more profound.

From our previous study[8] showing that the lower blood pressure limit of cerebral autoregulation is 62 mm Hg of SBP in NTR and 95 mm Hg in SHR, the lower CBP limit calculated was approximately 25 mm Hg in NTR and 50 mm Hg in SHR, respectively, as shown in Figure 1. CBP following occlusion was maintained at the level beyond 25 mm Hg in NTR at SBP above 60 mm Hg, while CBP remained below 50 mm Hg in most of SHR despite SBP being higher than 145 mm Hg. From the present study, a marked fall of CBP to below the critical perfusion pressure of cerebral autoregulation in SHR might be a major causative factor of a diffuse and extensive cerebral infarction following bilateral carotid artery occlusion as demonstrated in the previous studies[1–3].

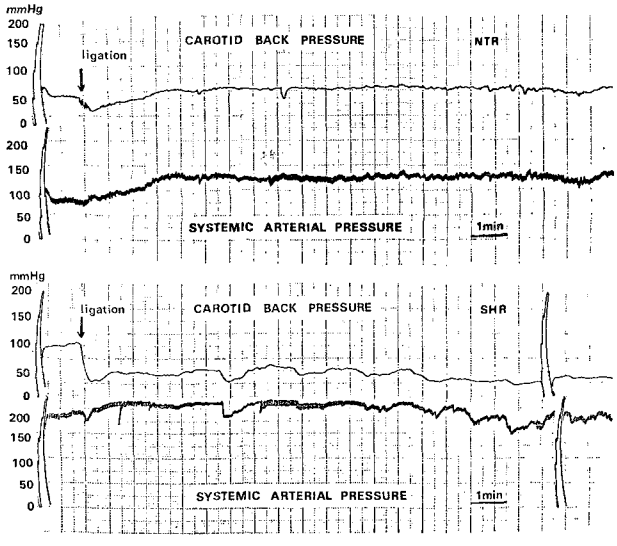

Fig. 2. Changes in carotid back pressure and systemic blood pressure following carotid occlusion in NTR and SHR. In NTR, an immediate but transient fall of carotid back pressure after occlusion is followed by a gradual rise as systemic blood pressure rises, whereas a profound fall of carotid back pressure in SHR is followed by a progressive lowering despite systemic blood pressure remaining at higher level.

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