Cerebral Blood Flow and Metabolism in Normotensive and Hypertensive Patients With Transient Neurologic Deficits

Kenichiro Fujii, MD, Seizo Sadoshima, MD, Yasushi Okada, MD, Hiroshi Yao, MD, Yasuo Kuwabara, MD, Yuichi Ichiya, MD, and Masatoshi Fujishima, MD

We used positron emission tomography to examine retrospectively the effects of blood pressure on regional cerebral blood flow and oxygen metabolism in seven normotensive and eight hypertensive patients with a history of transient neurologic deficits. In the hypertensive patients, a decrease in regional cerebral blood flow was closely related to blood pressure; these changes were most pronounced in the supratentorial structures, especially the striatum and thalamus. In contrast, the regional cerebral metabolic rate for oxygen was less related to blood pressure. Consequently, the regional oxygen extraction fraction was increased in the hypertensive patients, while regional cerebral blood volume and the regional cerebral blood flow:volume ratio were unchanged. Multivariate regression analysis confirmed that hypertension was an independent factor affecting regional cerebral blood flow. The analysis also disclosed that age, sex, hematocrit, smoking, and PaCO₂ affected regional cerebral blood flow. These findings suggest that the hemodynamic reserve in hypertensive individuals is reduced, which may predispose them to cerebral ischemia and perhaps stroke, even during small decreases in cerebral perfusion pressure. (Stroke 1990;21:283–290)

Cerebral blood flow (CBF) in hypertensive persons is maintained at the same level as in normotensive individuals until significant arteriosclerosis and hypertensive vascular disease develop.1,2 Using the xenon-133 inhalation method, Rodriguez et al3 recently demonstrated a significant inverse correlation between mean arterial blood pressure (MABP) and mean hemispheric CBF. Structural adaptation of the vascular wall to long-standing hypertension, such as hypertrophy of smooth muscle and proliferation of connective tissue,4,5 may play an important role in reducing CBF by encroaching on the arterial lumen. However, the effects of blood pressure on regional cerebral blood flow (rCBF), especially in deep structures such as the striatum and thalamus, or on regional cerebral oxygen metabolism are not yet known.

Persons with histories of transient neurologic deficits are considered to have more advanced vascular changes than asymptomatic individuals and are thought to be susceptible to subsequent cerebral ischemia. In such persons, thorough evaluations of cerebral hemodynamic reserve are needed.

We designed our study to explore retrospectively how blood pressure affects rCBF and cerebral oxygen metabolism measured by positron emission tomography (PET) in patients with transient neurologic deficits. Although the study design has some limitations, several interesting relations between blood pressure and rCBF and cerebral oxygen metabolism were disclosed.

Subjects and Methods

From July 1984 to September 1987, we used PET to examine 96 patients admitted to the Second Department of Internal Medicine at Kyushu University Hospital in Fukuoka City, Japan. Among these 96 patients, we retrospectively studied 17 who were neurologically normal on admission but had histories of transient neurologic symptoms. No patient had evidence of head trauma, brain tumor, epilepsy, or valvular heart disease. Two patients with atrial fibrillation were excluded from the study because thromboembolism was considered to be responsible for their neurologic events. Table 1 shows the transient neurologic abnormalities in the remaining 15 patients. Transient central nervous system disorders were considered to be responsible for all neurologic
Table 1. Clinical and Neuroradiologic Findings in Seven Normotensive and Eight Hypertensive Patients With Histories of Transient Neurologic Symptoms

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>Transient neurologic deficits</th>
<th>Duration (yr)</th>
<th>Known highest (mm Hg)</th>
<th>Hypertension</th>
<th>Blood pressure (mm Hg)</th>
<th>Electrocardiography</th>
<th>Antihypertensive treatment on admission</th>
<th>Neuroradiologic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotensive</td>
<td></td>
<td></td>
<td></td>
<td>On admission</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/66/M</td>
<td>R numbness</td>
<td></td>
<td></td>
<td>158/70</td>
<td>99</td>
<td>158/84</td>
<td>109</td>
<td>I</td>
</tr>
<tr>
<td>2/61/F</td>
<td>Amnesia</td>
<td></td>
<td></td>
<td>112/74</td>
<td>47</td>
<td>112/74</td>
<td>87</td>
<td>I</td>
</tr>
<tr>
<td>3/59/F</td>
<td>Amnesia</td>
<td></td>
<td></td>
<td>132/84</td>
<td>100</td>
<td>134/88</td>
<td>103</td>
<td>I</td>
</tr>
<tr>
<td>4/58/M</td>
<td>L numbness</td>
<td></td>
<td></td>
<td>138/86</td>
<td>103</td>
<td>114/70</td>
<td>85</td>
<td>0</td>
</tr>
<tr>
<td>5/62/F</td>
<td>Amnesia</td>
<td></td>
<td></td>
<td>112/76</td>
<td>88</td>
<td>130/76</td>
<td>94</td>
<td>0</td>
</tr>
<tr>
<td>6/53/M</td>
<td>Amnesia</td>
<td></td>
<td></td>
<td>104/70</td>
<td>81</td>
<td>104/70</td>
<td>81</td>
<td>0</td>
</tr>
<tr>
<td>7/52/M</td>
<td>Paraparesis</td>
<td></td>
<td></td>
<td>130/84</td>
<td>99</td>
<td>110/70</td>
<td>83</td>
<td>I</td>
</tr>
</tbody>
</table>

Mean±SD 59±5

Hypertensive

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>Transient neurologic deficits</th>
<th>Duration (yr)</th>
<th>Known highest (mm Hg)</th>
<th>Hypertension</th>
<th>Blood pressure (mm Hg)</th>
<th>Electrocardiography</th>
<th>Antihypertensive treatment on admission</th>
<th>Neuroradiologic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>8/64/M</td>
<td>Paraparesis</td>
<td>0.5</td>
<td>200/110</td>
<td></td>
<td>150/84</td>
<td>106</td>
<td>136/86</td>
<td>103</td>
</tr>
<tr>
<td>9/66/M</td>
<td>Amnesia Dyscalculia</td>
<td>0.8</td>
<td>200/110</td>
<td></td>
<td>190/84</td>
<td>119</td>
<td>174/88</td>
<td>117</td>
</tr>
<tr>
<td>10/71/M</td>
<td>L hemiparesis</td>
<td>8</td>
<td>Unknown</td>
<td></td>
<td>190/100</td>
<td>130</td>
<td>140/88</td>
<td>105</td>
</tr>
<tr>
<td>11/70/F</td>
<td>Dizziness, dysarthria</td>
<td>5</td>
<td>190/100</td>
<td></td>
<td>156/86</td>
<td>109</td>
<td>142/84</td>
<td>103</td>
</tr>
<tr>
<td>12/60/F</td>
<td>Paraparesis</td>
<td>11</td>
<td>Unknown</td>
<td></td>
<td>142/70</td>
<td>94</td>
<td>138/76</td>
<td>97</td>
</tr>
<tr>
<td>13/53/M</td>
<td>Amnesia</td>
<td>6</td>
<td>160/110</td>
<td></td>
<td>128/88</td>
<td>101</td>
<td>134/84</td>
<td>101</td>
</tr>
<tr>
<td>14/61/M</td>
<td>Amaurosis fugax</td>
<td>16</td>
<td>180/110</td>
<td></td>
<td>134/88</td>
<td>103</td>
<td>126/74</td>
<td>91</td>
</tr>
<tr>
<td>15/65/M</td>
<td>Dizziness, gait disturbance</td>
<td>6</td>
<td>180/90</td>
<td></td>
<td>170/90</td>
<td>117</td>
<td>130/80</td>
<td>97</td>
</tr>
</tbody>
</table>

Mean±SD 64±6

<table>
<thead>
<tr>
<th>Ocular fundi</th>
<th>LVH Mean scoring points</th>
<th>Brain CT (low-density area)</th>
<th>Angiography</th>
</tr>
</thead>
</table>

** Notes: **
- Pt, patient; S, systolic blood pressure; D, diastolic blood pressure; MABP, mean arterial blood pressure; PET, positron emission tomography; CT, computed tomography; M, male; F, female; R, right; L, left; +, presence; —, absence of abnormal findings; Ca, calcium antagonist; β-B, β blocker; D, diuretics; AC, angiotensin-converting enzyme inhibitor; B, bilateral; ICA, internal carotid artery; VA, vertebral artery.
- *p<0.01, 0.001, respectively, different from normotensive.
was 1-3 months in all patients. rCBF and regional cerebral metabolic rate for oxygen (rCMRO₂) was calculated as rCMRO₂ = rCBF x rOEF x arterial oxygen content. Both rOEF and rCMRO₂ were corrected using a single inhalation of C₁₅O gas. We used a HEADTOME-III device (Shimadzu Inc., Kyoto, Japan and Akita Noken, Akita, Japan) with planar and axial resolutions of 8.2 mm full-width at half-maximum and corrected for attenuation by means of a transmission scan using an external germanium-68-gallium-68 ring source in each patient. During the PET study, each patient was placed in the supine position with the eyes open and the ears unplugged in a dimly lit room. One femoral artery was cannulated to sample blood for radioactive concentration and blood gases. Arterial blood gases were analyzed at the start and end of each scan.

Five slices (20, 35, 50, 65, and 80 mm above the orbitomeatal line) were obtained and imaged in each scan. Regions of interest 18x14 mm were set on the orbitomeatal line) were obtained and imaged in each slice. Parameters were chosen for the multiple regression model by the stepwise forward selection method. We used PaCO₂ in our analysis.

Data are given as mean±SD. We compared average rCBF, rOEF, rCMRO₂, rCBV, and the rCBF:rCBV ratio in each region of interest between the normotensive and hypertensive groups using Student’s small-sample t test and Welch’s t test for unequal variances. The relations between MAP on admission or at the PET study and rCBF, rOEF, rCMRO₂, and rCBV in each region of interest were evaluated by linear regression analysis. To elucidate the clinical parameters that could influence rCBF, we performed multivariate regression analysis. Parameters were chosen for the multiple regression model by the stepwise forward selection method. We used PaCO₂ in our analysis.
TABLE 2. Blood Chemistries, Risk Factors for Stroke, and Blood Gas Parameters in Seven Normotensive and Eight Hypertensive Patients With Transient Neurologic Symptoms

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>Normotensive (n=7)</th>
<th>Hypertensive (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (cigarettes/day)</td>
<td>1-2</td>
<td>3-6</td>
</tr>
<tr>
<td>Drinking &gt;34 g alcohol/day</td>
<td>4-6</td>
<td>8-10</td>
</tr>
<tr>
<td>Arterial acid-base parameters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>40.2±2.2</td>
<td>40.0±4.1</td>
</tr>
<tr>
<td>PaO₂ (mm Hg)</td>
<td>75.5±7.2</td>
<td>78.4±11.9</td>
</tr>
<tr>
<td>pH</td>
<td>7.42±0.02</td>
<td>7.42±0.03</td>
</tr>
<tr>
<td>Hemoglobin concentration (g/dl)</td>
<td>13.7±1.2</td>
<td>13.2±1.2</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>42.2±4.8</td>
<td>40.3±3.4</td>
</tr>
</tbody>
</table>

Values are mean±SD, except for risk factors, which are number of patients.

because it is known to be the most powerful cerebrovascular regulator.

Results

Table 1 summarizes the clinical and neuroradiologic findings in the seven normotensive and the eight hypertensive patients. Age and sex ratio did not differ between the groups. MABP on admission was significantly higher in the hypertensive than in the normotensive group (Table 1). Three of the eight hypertensive patients (10, 12, and 14) had received antihypertensive treatment, and after admission the treatment was continued in patient 10 but was stopped in patients 12 and 14. Blood pressure in the normotensive patients changed very little after admission, while that in the hypertensive patients tended to decrease; the difference in MABP between the groups became nonsignificant by the time of the PET study. Mean scoring points for the normotensive and hypertensive groups averaged 0.32 and 1.44, respectively. Electroencephalograms showed no focal or diffuse abnormalities. Brain CT revealed very small low-density areas in one normotensive and three hypertensive patients. Angiography disclosed stenosis of the major arteries or diffuse arteriosclerosis in three normotensive and four hypertensive patients. The degree of stenosis of all these vessels was <50% of the lumina. The incidences of abnormal findings on CT and angiography did not differ significantly between the groups.

Table 2 depicts the group means for serum chemistries and the incidence of smoking and drinking habits on admission. The average values for arterial blood gases, arterial pH, hemoglobin, and hematocrit measured during the PET study are also listed in Table 2; all were within normal ranges. None of these values differed significantly between the groups.

Table 3 depicts the group means for rCBF, rOEF, rCMRO₂, rCBV, and the rCBF/rCBV ratio at 10 brain regions in the two groups. The values of the

rCBF, regional cerebral blood flow; rOEF, regional oxygen extraction fraction; rCMRO₂, regional cerebral metabolic rate for oxygen; rCBV, regional cerebral blood volume; n, number of regions of interest; N, normotensive; H, hypertensive. Values are mean±SD for n regions of interest, except for brainstem, which are mean±SD for number of patients.

*Data for seven normotensive and eight hypertensive patients.

†‡p<0.05, 0.01, respectively, different from normotensive.
latter four measures for two patients (one normoten-
vive and one hypertensive) were excluded because of
inappropriate head position during the PET study.
rcBF and rCMRO2 in the hypertensive group were
lower than in the normotensive group, and the dif-
fERENCE was significant in the frontal cortex for rCBF
and in the striatum and thalamus for both rCBF and
rCMRO2. In contrast, rOEF in the hypertensive
group was significantly higher than in the normoten-
vise group in all regions except the brainstem. rCBV
in the cerebral cortices and cerebellum were virtually
the same in the two groups. The rCBF:rCBV ratio
was slightly lower in the hypertensive than in the
normotensive group, but the values in both groups
were higher than the 5.5–6.0 thought to be the lower
limit of autoregulation.9

Table 4 shows the correlation coefficients (rs) for
rCBF, rOEF, rCMRO2, rCBV, and the rCBF:rCBV
ratio versus MABP on admission for the two groups
combined. Regions of interest for both hemispheres
were considered. Among the supratentorial struc-
tures, highly significant inverse correlations were
present between rCBF and MABP for all except the
frontal cortex. Similarly, rOEF was significantly
correlated with MABP in all supratentorial structures.
On the other hand, significant but weaker inverse
correlations were observed between rCMRO2 and
MABP in the temporal and parietal cortices, the
thalamus, and the white matter. rCBV was signifi-
cantly (inversely) correlated with MABP only in the
white matter. The rCBF:rCBV ratio tended to
decrease with rising MABP, but the correlations
were not significant in any region. Among the infra-
tentorial structures, there were no significant corre-
lations between MABP and any of these five mea-
sures. The relations between MABP and rCBF,
rOEF, and rCMRO2 are shown in Figure 2.

The relation between rCBF and MABP on the day
of the PET study also revealed a highly significant
inverse correlation in the thalamus (r = -0.498,
p < 0.01) but not in the other regions.

Table 5 depicts the standardized multiple regression
coefficients for various clinical parameters ver-
sus rCBF. Age, male sex, and hematocrit were neg-
atively correlated with rCBF in the cerebral cortices
and white matter. Smoking was negatively correlated
with rCBF in the cerebral cortices and the thalamus.
MABP was also negatively and strongly correlated
with rCBF in the parietal and occipital cortices, the
striatum, and the thalamus. The correlation between
total cholesterol, triglyceride, or fasting blood sugar
concentrations and rCBF was relatively weak and
differed from region to region. On the other hand,
alcohol drinking was positively correlated with mean
cortical rCBF and with rCBF in the occipital cortex.
There was also a positive correlation between Paco2
and rCBF in the cortices, the striatum, the white
matter, and the brainstem.

Discussion
There are several limitations to interpreting our
results. The design of our study has its major disad-
vantage in the retrospective selection of patients and
their matching between groups. Multivariate regres-
sion analysis was necessary because we could not
exclude hypertensive patients with other risk factors
for stroke from the study due to the overall limited
number of patients. In addition, lack of magnetic
resonance imaging restricted our information about
underlying tissue changes such as white matter dis-
case. Finally, our patients had a history of transient
neurologic deficits so our results cannot necessarily
be generalized to asymptomatic hypertensive per-
sons. Even considering these limitations, our study
has disclosed several interesting relations between
blood pressure and regional cerebral circulation and
metabolism.

rCBF was decreased in the hypertensive patients,
and the decrease was closely related to MABP. In
contrast, rCMRO2 was less closely related to MABP.
Consequently, the brain in hypertensive individuals
extracts more oxygen from the blood (increased
rOEF) to maintain rCMRO2, while rCBV and the
rCBF:rCBV ratio were unchanged. In addition,
interesting differences among brain regions were
disclosed. The relations of MABP with brain regions
and rCMRO2 were pronounced in the supratentorial struc-

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**Table 4. Correlation Coefficients for rCBF, rOEF, rCMRO2, rCBV, and rCBF:rCBV Ratio vs. Mean Arterial Blood Pressure on Admission in 13 Patients With Transient Neurologic Symptoms by Brain Region**

<table>
<thead>
<tr>
<th></th>
<th>Cerebral cortices</th>
<th>Deep gray matter</th>
<th>White matter</th>
<th>Infratentorial structures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FrONTAL</td>
<td>TEMPORAL</td>
<td>PARITEL</td>
<td>OCCIPITAL</td>
</tr>
<tr>
<td>rCBF* 30</td>
<td>-0.327</td>
<td>-0.580†</td>
<td>-0.521†</td>
<td>-0.570†</td>
</tr>
<tr>
<td>rOEF 26</td>
<td>0.480†</td>
<td>0.626†</td>
<td>0.559†</td>
<td>0.521†</td>
</tr>
<tr>
<td>rCMRO2 26</td>
<td>-0.206</td>
<td>-0.424‡</td>
<td>-0.422‡</td>
<td>-0.375</td>
</tr>
<tr>
<td>rCBV 26</td>
<td>-0.006</td>
<td>0.165</td>
<td>0.025</td>
<td>0.106</td>
</tr>
<tr>
<td>rCBF:rCBV 26</td>
<td>-0.234</td>
<td>-0.370</td>
<td>-0.342</td>
<td>-0.328</td>
</tr>
</tbody>
</table>

rcBF, regional cerebral blood flow; rOEF, regional oxygen extraction fraction; rCMRO2, regional cerebral metabolic rate for oxygen; rCBV, regional cerebral blood volume; n, number of regions of interest. Data are values of r for n regions of interest, except for brainstem, which are values of r for number of patients.

*Data for 15 patients.
†p<0.01, 0.05, respectively, different from 0.
Hypertension has been shown to be an important risk factor for reduced CBF among neurologically normal volunteers\(^1\) and among patients with transient ischemic attacks.\(^1\) An autopsy study in Hisayama, Japan,\(^8\) has shown that cerebral atherosclerosis occurs 20–30 years earlier in hypertensive than in normotensive patients. In stroke-prone spontan-

**TABLE 5. Standardized Multiple Regression Coefficients for Clinical Parameters vs. Regional Cerebral Blood Flow in 15 Patients With Transient Neurologic Symptoms**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Cerebral cortices</th>
<th>Deep gray matter</th>
<th>White matter</th>
<th>Infratentorial structures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Frontal</td>
<td>Temporal</td>
<td>Occipital</td>
<td>Mean</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>-0.580*</td>
<td>-0.404†</td>
<td>-0.822*</td>
<td>-0.686*</td>
</tr>
<tr>
<td>Male sex (yes)</td>
<td>-0.454‡</td>
<td>-0.614*</td>
<td>-0.621*</td>
<td>-0.680‡</td>
</tr>
<tr>
<td>Mean arterial blood pressure (mm Hg)</td>
<td>-0.362†</td>
<td>-0.615*</td>
<td>-0.807*</td>
<td>-0.669*</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>-0.234†</td>
<td>0.281‡</td>
<td>-0.406‡</td>
<td>0.321†</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>0.267†</td>
<td>0.286‡</td>
<td>-0.420‡</td>
<td>-0.406‡</td>
</tr>
<tr>
<td>Fasting blood sugar (mg/dl)</td>
<td>-0.398†</td>
<td>-0.493*</td>
<td>-0.673*</td>
<td>-0.969*</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>-0.424‡</td>
<td>-0.455‡</td>
<td>-0.393*</td>
<td>-0.327†</td>
</tr>
<tr>
<td>Smoking (yes)</td>
<td>0.810*</td>
<td>0.650*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drinking (yes)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paco(_2) at PET study</td>
<td>0.237†</td>
<td>0.313†</td>
<td>1.183*</td>
<td>0.545†</td>
</tr>
</tbody>
</table>

PET, positron emission tomography. Values are \(r\) for 30 regions, except for brainstem, which are \(r\) for 15 patients. Only significant values are indicated.

\(^*p<0.001, 0.05, 0.01\), respectively, different from 0.
ously hypertensive rats, it was suggested that thickening of the arterial wall at the terminal stage of severe hypertension caused an irreversible and severe decrease in rCBF. These findings suggest that rCBF is markedly influenced by structural vascular changes promoted by hypertension. Our patients had histories of transient signs of neurologic deficits and were considered to have advanced vascular changes. Thus, our hypertensive patients might have lower rCBF than asymptomatic hypertensive individuals.

Interestingly, the correlation between MABP on the day of the PET study and rCBF was weak and nonsignificant except in the thalamus. The blood pressure of hypertensive patients usually falls after hospital admission, although the mechanism is not fully understood. Restriction of sodium intake to 137-171 meq/day compared with the Japanese average daily sodium intake of 222-256 meq/day and bed rest during hospitalization may be part of the reason for the decline in blood pressure. Thus, the lack of a significant relation between rCBF and MABP on the day of the PET study seems to be due to narrowing of the MABP range by a spontaneous decline in blood pressure after hospital admission. The reason why a significant correlation was observed only in the thalamus is unclear.

The above-mentioned reduction in MABP might reduce cerebral perfusion pressure and CBF in hypertensive individuals in whom the autoregulatory curve is shifted to the right, especially patients receiving antihypertensive treatment. In such patients with hemodynamic failure, the cerebral vessels usually dilate and increased rCBV and a decreased rCBF: rCBV ratio are expected in addition to an increased rOEF. However, neither rCBV nor the rCBF: rCBV ratio in our hypertensive group differed from those in the normotensive group. The absence of increased rCBV and a decreased rCBF: rCBV ratio despite increased rOEF and similar MABP during the study in our patients with chronic hypertension rather suggests a baseline narrowing of vessel lumen, probably due to either advanced arteriosclerosis or excess vasoconstriction.

The differences in rCBF and rCMRO2 between our normotensive and hypertensive groups were more pronounced in the deep gray matter, that is, in the striatum and thalamus, than in the cerebral cortices. In the deep gray matter, the reduced rCMRO2 in the hypertensive group seemed not to be effectively compensated for in spite of increased rOEF. The increased rOEF, including measurements in two hypertensive patients with deep-seated small lesions proven by CT, minimizes the possible effects of infarction, where matched hyperperfusion is expected. The absence of increased rCBV and a decreased rCBF: rCBV ratio in the deep gray matter as in the cerebral cortices suggests a minimal contribution of the reduction in MABP after admission in patients with hypertension to the greater reduction in rCBF. Also, it is well known that systemic hypoten-

sion and/or arterial occlusive disease in the neck mainly produce ischemic lesions in arterial border zone territories and not in the deep gray matter. A greater reduction of rCBF within the striatum and thalamus suggests that hypertensive vascular alterations occurred more markedly and selectively with elevated vascular resistance in the deep structures of the brain. The striatum and thalamus are the areas most susceptible to lacunar thrombotic infarction. Since their small branches are separated directly from the main trunks of the middle cerebral artery or the posterior cerebral artery, high intraluminal pressure is conducted directly to the vessel wall, which may lead to intimal thickening, angionecrosis, and the formation of fibrous nodules. In contrast, the relation of MABP with rCBF and rCMRO2 was obscure in the infratentorial structures, especially the cerebellum. Less marked arteriosclerosis in the cerebellum might account for this finding.

After multivariate regression analysis, strong negative correlations between MABP on admission and rCBF were still present in the striatum, the thalamus, and the parietal and occipital cortices. In several supratentorial structures, age, male sex, hematocrit, and smoking habits were negatively correlated with rCBF. These factors are risk factors for stroke, and our results suggest that they could also affect rCBF. In other studies, similar effects of sex and smoking on CBF are also shown. In our study, there were more smokers among the hypertensive than among the normotensive patients. Although this difference was not significant, it might be responsible in part for the greater reduction in rCBF in our hypertensive group. Hematocrit is a major determinant of blood viscosity and plays an important role in regulating CBF. Our results also support this concept. Hyperglycemia and hypercholesterolemia are known risk factors for stroke, but our study did not reveal any consistent relation between these clinical parameters and rCBF, possibly because of the few patients with these abnormalities or the mildness of these abnormalities. Also, in some brain regions we could not exclude the effect of PaCO2, which is a well-known cerebrovascular regulator.

In conclusion, patients with chronic hypertension had lower rCBF and higher rOEF than normotensive patients despite similar perfusion pressures (the rCBF: rCBV ratio). These findings suggest that hypertensive individuals have reduced hemodynamic reserve and are more susceptible to cerebral ischemia and perhaps to stroke during small reductions in cerebral perfusion pressure. One implication of these findings is that care should be taken in such individuals to avoid reductions in cerebral perfusion pressure, especially during antihypertensive treatment.

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References


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