Cortical artery pressure in normotensive and hypertensive aneurysm patients

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Cortical artery pressure (CAP) and systemic pressure (SP) were measured in eight normotensive and six hypertensive patients with anterior circulation aneurysms. In the hypertensive patients significant gradients developed between CAP and SP as these pressures were lowered. The relationship between CAP and SP was expressed by the best-fit equation \( \text{CAP} = 1.02 \times \text{SP} - 9.27 \) in the normotensive patients and by \( \text{CAP} = 1.54 \times \text{SP} - 65.60 \) in the hypertensive patients. In the latter, the cycle of decreasing and increasing pressures formed a hysteresis loop suggesting prolonged cortical vasoconstriction despite recovery of systemic pressure. Selective pressure measurements in the distal (D) and proximal (P) segments of the cortical arteries were also obtained. The D/P ratio describes the relative contribution of the collateral circulation to cortical artery pressure. In normotensive patients, the D/P ratio was maintained down to an SP of 48 mm Hg. In hypertensive patients this ratio decreased with lowered SP, and a critical closing pressure of 40 mm Hg was predicted for the distal circulation. These studies describe the limited capacity of the cortical circulation to maintain perfusion pressure in hypertensive patients. These responses should be considered when assessing the risks associated with such procedures as carotid ligation or hypotensive anesthesia.

KEY WORDS: cortical artery pressure • cerebral aneurysm • aneurysm surgery • internal carotid artery • ligation • hypertension • hypotension • extracranial-intracranial bypass

Despite advances in our understanding of cerebral hemodynamics, certain fundamental relationships between arterial pressures in the extracranial and intracranial segments of the circulation are still unclear. Cerebral perfusion pressure (CPP) is usually defined as the difference between mean systemic arterial pressure and intracranial pressure. The relationship between mean systemic arterial pressure and cortical artery pressure (CAP) has not been well described, and therefore the validity of the definition for CPP as it applies to cerebral regions of interest is uncertain. This is important during carotid ligation or induced hypotension during aneurysm surgery, which may require drastic alterations of systemic pressure (SP). A substantial number of patients who undergo these procedures are hypertensive and the responses of CAP to changes in SP in these patients may be different from those in normotensive patients.

An opportunity for direct study of CAP was provided during craniotomy for aneurysm. In patients who had microsurgical clipping of a cerebral aneurysm, changes in the CAP's were compared to pharmacologically induced changes in SP. In patients with inoperable aneurysms, the pressure changes in the cortical arteries were compared to local changes in SP induced mechanically by internal carotid artery (ICA) ligation. These measurements provide a better understanding of CPP in the normal and hypertensive cerebral circulation.

Clinical Material and Methods

Clinical Material

Fourteen patients were included in this study. Pertinent clinical information regarding these patients is given in Table 1. Eight patients were normotensive; systolic blood pressure ranged between 105 and 145 mm Hg, and diastolic blood pressure ranged between 66 and 90 mm Hg. Six patients were hypertensive and were treated with antihypertensive drugs for 4 to 9 years before admission. Systolic blood pressure in these patients ranged between 140 and 180 mm Hg, and diastolic blood pressure ranged between 80 and 110 mm Hg. The age and sex distribution of these patients is given in Table 1.

Three patients had a hemispheric neurological deficit. Other clinical syndromes led to hospital admission in 11 patients. In five patients, oculomotor palsies were the presenting complaint, and one patient had de-
TABLE 1
Clinical data in patients undergoing intraoperative cortical artery pressure measurements

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Clinical Syndrome</th>
<th>Site of Aneurysm</th>
<th>Hypertension</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>48, M</td>
<td>rt III, IV, &amp; V nerve palsies</td>
<td>ICA</td>
<td>+</td>
<td>ICA ligation, bypass</td>
</tr>
<tr>
<td>2</td>
<td>57, M</td>
<td>decreased vision, headache</td>
<td>ACoA</td>
<td>-</td>
<td>clip</td>
</tr>
<tr>
<td>3</td>
<td>48, M</td>
<td>rt III &amp; VI nerve palsies</td>
<td>ICA</td>
<td>+</td>
<td>ICA ligation, bypass</td>
</tr>
<tr>
<td>4</td>
<td>60, M</td>
<td>headache, III &amp; VI nerve palsies</td>
<td>ICA</td>
<td>-</td>
<td>ICA ligation, bypass</td>
</tr>
<tr>
<td>5</td>
<td>55, F</td>
<td>headache</td>
<td>ICA</td>
<td>-</td>
<td>clip</td>
</tr>
<tr>
<td>6</td>
<td>56, M</td>
<td>rt III nerve palsy</td>
<td>PCoA</td>
<td>+</td>
<td>rt common carotid ligation</td>
</tr>
<tr>
<td>7</td>
<td>60, F</td>
<td>rt hemiparesis</td>
<td>ACoA</td>
<td>-</td>
<td>clip</td>
</tr>
<tr>
<td>8</td>
<td>57, F</td>
<td>headache</td>
<td>PCoA</td>
<td>-</td>
<td>ICA ligation, bypass</td>
</tr>
<tr>
<td>9</td>
<td>56, M</td>
<td>rt hemiparesis</td>
<td>ACoA</td>
<td>+</td>
<td>clip</td>
</tr>
<tr>
<td>10</td>
<td>62, F</td>
<td>dysphasia, rt hemiparesis</td>
<td>MCA-ICA</td>
<td>+</td>
<td>clip</td>
</tr>
<tr>
<td>11</td>
<td>53, M</td>
<td>headache, dysphasia</td>
<td>ICA</td>
<td>-</td>
<td>ICA ligation, bypass</td>
</tr>
<tr>
<td>12</td>
<td>32, F</td>
<td>headache</td>
<td>ophtal</td>
<td>+</td>
<td>ICA ligation, bypass</td>
</tr>
<tr>
<td>13</td>
<td>56, F</td>
<td>rt III, V, &amp; VI nerve palsies</td>
<td>ICA</td>
<td>-</td>
<td>ICA ligation, bypass</td>
</tr>
<tr>
<td>14</td>
<td>62, F</td>
<td>headache</td>
<td>ICA</td>
<td>-</td>
<td>ICA ligation, bypass</td>
</tr>
</tbody>
</table>

* Abbreviations: ICA = internal carotid artery; ACoA = anterior communicating artery; PCoA = posterior communicating artery; MCA = middle cerebral artery; + = present; - = absent.

Results

Systemic Pressures

Radial Artery Pressure. Under anesthesia, the mean blood pressure (MBP) in the 14 patients studied ranged between 66 and 116 mm Hg. The MBP in the hypertensive patients was 94 ± 13 mm Hg and in the normotensive patients 78 ± 8 mm Hg. The difference in the intraoperative resting MBP in the two groups was significant (p < 0.05).
Cortical artery pressure related to hypertension

**Internal Carotid Artery Pressure.** Blood pressure was measured in the ICA in nine patients. Five of these patients were normotensive and four were hypertensive. There was no significant difference between MBP measured in the radial and carotid arteries. Temporary occlusion of the ICA resulted in an average fall of 48% and 55% of MBP in the ICA in the normotensive and hypertensive patients, respectively. The difference was not significant.

**Cortical Artery Pressures**

**Relation to Cortical Artery Diameter.** Cortical artery blood pressure was measured from 10 arteries on the surface of the temporal lobe and four arteries on the surface of the parietal lobe. The outer diameter of these arteries varied between 0.6 and 1.6 mm. There was no significant relationship between the CAP's or the CAP/SP ratios and the arterial diameters of the arteries studied.

**Relation of Systemic Pressure.** In normotensive patients, the decrease in CAP was proportionate to that in SP, as expressed by the best-fit equation, \( \text{CAP} = 1.02 \text{ SP} - 9.27 \). In the hypertensive patients, gradients between SP and CAP developed and became more significant as pressures were lowered. This relationship was expressed by the best-fit equation, \( \text{CAP} = 1.54 \text{ SP} - 64.40 \). The 95% confidence bands for both groups of patients are given in Fig. 1, and the regression coefficients are significantly different \((p < 0.005)\). To be certain that these differences were unrelated to intrinsic differences in SP in normotensive and hypertensive patients, the relationship of the CAP/SP ratio to SP was also examined (Fig. 2). The regression for the CAP/SP ratio compared with SP in the hypertensive and normotensive patients was significantly different \((p < 0.01)\).

The return of baseline SP in the normotensive patients was associated with increases in CAP's corresponding to the best-fit equation, \( \text{CAP} = 1.033 \text{ SP} - 7.10 \). In the hypertensive patients, CAP values were significantly lower during blood pressure recovery than during induced hypotension (Fig. 3). This hysteresis effect produced a relative decrease in CAP of 32 mm Hg at a SP of 80 mm Hg. The return to baseline SP in the hypertensive patients was described by the best-fit equation CAP = 0.81 SP - 26.26. The 95% confidence bands for both normotensive and hypertensive patients are given in Fig. 4. The regression coefficients were significantly different \((p < 0.005)\). The CAP/SP ratios in the hypertensive patients also changed significantly compared to the ratio in the normotensive patients as SP returned to normal (see Fig. 2).

**Distal to Proximal Cortical Artery Pressure Ratios.** Before SP was manipulated, the ratio of distal to proximal pressure (D/P) in the normotensive patients varied between 0.88 and 0.97 with a mean D/P ratio of 0.93. The D/P ratio in the hypertensive patients varied between 0.78 and 0.91, with a mean D/P ratio of 0.86. The mean ratios were not significantly different \((p < 0.01)\).
Relation of The D/P Ratio to Changing Systemic Pressure. In the normotensive patients, the D/P ratio remained constant as SP was lowered to 48 mm Hg. Below this level, the D/P ratio dropped in relation to SP. The relationship of D/P and SP was expressed by the best-fit line whose equation was D/P = 0.011 SP + 0.28. The 95% confidence bands for D/P are given in Fig. 5. Extrapolation of the data to lower SP’s predicted that the D/P ratio will remain above 0.35 at very low SP’s. In the hypertensive patients, the D/P ratio decreased in relation to the earliest decreases in SP. The relationship of D/P to SP was expressed by the best-fit line whose equation was D/P = 0.014 SP - 51. Extrapolation of this slope predicted that distal CAP will approach zero as SP approached 40 mm Hg (see Fig. 5). The regression coefficients for the normotensive and hypertensive patients were significantly different (p < 0.001).

Discussion

The safety of carotid ligation and induced hypotension is directly related to the adequacy of CPP and its relationship to cerebrovascular resistance. There are few data, however, that describe the relationship between blood pressure measured systemically or in the extracranial carotid arteries and the blood pressure in the intracranial arteries. Previously published studies of CAP do not provide sufficient data to define its relationship to systemic artery pressures. Bloor, et al., measured pressures simultaneously in the ICA and in the larger intracranial arteries at the base of the brain in four patients. One patient had an anterior cerebral artery aneurysm, two had arteriovenous anomalies, and one had an anomaly of the external carotid artery. In one case, the pressure drop in the MCA was greater than, and in one case, less than, the pressure drop in the ICA after ICA ligation. Pressures in the surface cortical arteries were not measured. Bakay and Sweet, measured the pressures in larger intracranial arteries and in the cortical arteries of 11 patients after resection of brain tumors both before and after common carotid artery ligation. The percentage fall in systolic pressure during common carotid artery ligation was the same in the anterior and middle cerebral artery as in the ICA. However, pressures in the smaller temporal cortical branches were 86% and in the frontal cortical branches were 82% of the pressure in the common carotid artery. Despite these lower resting pressures the percentage fall in CAP was similar to the percentage fall in the ICA pressure after common carotid artery ligation.

More systematic studies have been carried out in animal experiments, however, the relative drop in CAP varies considerably in the different reported studies. Symon, et al., found that in dogs the pressure in 0.35 to 0.55-mm arteries varied between 80% and 90% of the femoral artery pressure. In the macaque monkey, CAP varied between 80% and 85% of femoral arterial pressures. Kanzow and Dieckhoff found in cats that MCA pressure was 80% and CAP was only 60% to 70% of aortic pressure. Shapiro, et al., studied a large range of pial arterioles and arteries. The pial aortic pressure ratios indicated that pressure in the larger pial arteries was 66% and pressure in the smaller arteries was 56% of aortic pressure. The rete mirabile found in cats, however, may influence the relationship between the pial arterial pressures and SP’s in a unique way. Shulman found a more significant difference in 0.4- to 0.7-mm arteries in adult mongrel dogs. At a mean aortic blood pressure of 137 ± 10 mm Hg the mean CAP in these arteries was only 63 ± 26 mm Hg. It is difficult to extrapolate the results of these measurements to the cerebral circulation in man. The data from our study indicated that at normal blood pressures the surface arteries with diameters in the range of 0.6 to 1.6 mm acted primarily as a conducting system. Furthermore, the CAP/SP ratio in the hypertensive
Cortical artery pressure related to hypertension

patients was similar to that in the normotensive patients. This suggests that chronic hypertension does not impose additional anatomic gradients proximal to the cortical circulation.

During blood pressure changes produced by carotid ligation or hypotensive anesthesia, a more complex relationship between the CAP and SP was anticipated. Stromberg and Fox found that when systemic blood pressure was reduced to 50 mm Hg there was a substantial loss in pressure head (21%) across the pial arterial network, but the loss was almost twice as great across the "upstream" arteries (39%). The data obtained from our patients indicated the relative rates of decline of CAP during hypotension and predicted the critical closing pressure for cortical arteries in normotensive and hypertensive patients. In the normotensive patients the regression equation predicted that CAP would remain near SP levels through the blood pressure range commonly employed during aneurysm surgery. In the hypertensive patients, the slope relating CAP and SP was steeper over the entire range of SP's. Below 72 mm Hg, the 95% confidence bands diverged from those of the normotensive patients. The sharper slope in the hypertensive patients was consistent with the development of increased resistance between the systemic and cortical artery measurement points.

The pressure gradients induced by changes in SP may be related to the known structural and functional alterations commonly found in hypertension. Roggendorf, et al., found that changes associated with mild hypertension include a thickening of the endothelial cell layer and hypertrophic smooth-muscle cells in the media. In chronic hypertension, lipohyalinosis and fibrinoid necrosis result in an actual decrease of the lumen wall ratio. Many other changes have been found, and may contribute to the increase in vascular resistance characteristic of the hypertensive circulation. These findings may have a bearing on the relationship of blood flow and blood pressure in hypertensive patients. Johansson, et al., and others found that the curve describing autoregulation of blood flow in hypertensive patients was shifted, so that there was a relatively early drop in blood flow as blood pressure was reduced. It was postulated that the mechanism for autoregulation to lowered blood pressure was a relative vasodilation. Our data are consistent with the development of increased resistance in the cortical arterial circulation during induced hypotension. Extrapolation from the CAP-SP slope to lower pressure levels indicates that the critical closing pressure for cortical arteries in hypertensive patients may occur at an SP as high as 40 mm Hg.

During return of SP to normal in the normotensive patients the slopes relating CAP and SP were similar to those obtained during falling SP. In hypertensive patients, however, the CAP's were lower than those achieved when SP was lowered. The wide confidence bands in the data from hypertensive patients are related to the hysteresis effect described. This hysteresis effect may indicate a further propensity for the hypertensive pial network to remain constricted despite restoration of SP.

Failure of the collateral circulation may also be more profound in hypertensive compared to normotensive patients. The ratio of distal and proximal CAP is related to the resistance in the distal circulation, including that in the penetrating arteries and the leptomeningeal collateral circulation. In the normotensive patients this ratio did not change between SP's of 50 to 80 mm Hg. The sharply decreased D/P ratios in the hypertensive patients, however, indicate a more marked increase in the resistance elements in the smaller-caliber distal arteries. At decreasing levels of perfusion, extrapolation of the slopes indicated that the distal pressure drops are more significant in hypertensive patients and contribute little to cortical artery back pressure as the critical closing pressure is approached. It is apparent then that, in hypertensive patients, both the larger extraparenchymal intradural arteries and the smaller distal cortical arteries have significantly higher critical closing pressures compared to normotensive patients. Furthermore, reperfusion after induced hypotension is significantly delayed, both because of increased cerebrovascular resistance and failure of the distal collateral circulation.

The ischemic complications of both carotid ligation and intracranial surgery are greater in hypertensive than in normotensive patients. The responses of the cortical circulation to changes in SP may explain the limited ability of the hypertensive patient to tolerate induced hypotension during carotid ligation or hypotensive anesthesia. When such modalities become necessary in hypertensive patients, it may be prudent to ensure that SP is maintained above 60 mm Hg in the ICA stump or in systemic blood pressure measurements. Specific measures to enhance the contribution of the collateral circulation to CAP in hypertensive patients deserve further study.

References

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