Cerebral Hyperperfusion during Surgical Resection of High-Flow Arteriovenous Malformations

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Cerebral hemodynamics were evaluated in 16 patients with arteriovenous malformations (AVMs) using intraoperative thermogradient or laser Doppler flowmeter. The postexcision/preexcision blood flow ratio was determined. Two of seven patients with large AVMs (>4 cm) developed a postoperative hematoma. Their flow ratios were larger than 1.9. In 4 patients with large AVMs, their blood flow ratios were reduced from an initial ratio exceeding 2.0 to less than 1.5 using carotid or special flow regulation clamps. These patients did not develop any hemorrhagic complication. In 9 patients of small AVMs (<4 cm), the ratio was significantly low and there were no hemorrhagic complications.

KEY WORDS: Intraoperative monitoring; Cerebral blood flow; Arteriovenous malformation; Normal perfusion pressure breakthrough

The aim of this study was to investigate cerebral hemodynamics in arteriovenous malformations (AVMs) in relation to various prognostic factors; angiographic finding of AVMs, hemodynamic changes occurring immediately after AVM excision, and postoperative hemorrhagic complications. According to the intraoperative cortical blood flow measurement, the degree of postexcision hyperperfusion was evaluated. In several patients with high-flow AVMs, clamps were applied on the proximal feeding arteries or on the ipsilateral common carotid artery so as to modulate the excessive blood flow after excision. After flow modulation, changes in the intraoperative hemodynamic parameters and the surgical results were analyzed.

Materials and Methods
Cortical blood flow was measured in 16 patients before and after AVM excision. A thermal blood flow monitor (Biomedical Science Inc., Tokyo, Japan) was used in every case except for cases 12 and 13. In cases 12 and 13, a laser Doppler flowmeter (model PF3, Perimed Co., Jarfalla, Sweden) was used. A clinical characteristics of the patients is summarized in Table 1. There were 14 males and 2 females tested with a mean age of 32.2 ± 14.8, ranging from 13 to 62 years. Blood flow was measured on several parts of the surrounding cortex about 2 cm away from the AVM in each patient. The ratio of postexcision/preexcision cortical blood flow was determined, and the maximum value of the ratio (P-P ratio) was correlate with the size of the AVMs and with the surgical outcome.

If the P-P ratio was greater than 2.0 in some region, special clamps made from silicone tubes of (Silascon, medical grade silicon tubes, Dow Corning Japan Co. Ltd., Tokyo, Japan) and absorbable thread (#8-0 Coated Vicryl, Ethicon Inc. Somerville, NJ) were applied around the most proximal portion of the main feeding arteries as is shown Figure 1 [31]. The feeding arteries were then constricted by tightening the threads until the P-P ratio was reduced below 1.5. Thus, the sudden increase in blood volume that develops after AVM excision is redirected to the contralateral carotid and posterior circulation or to the ipsilateral intact cerebral arterial system. Excision was confirmed to be complete by intraoperative or postoperative angiography.

In case of a giant AVM (case 4), extracranial carotid Selverstone clamps were applied to reduce the blood flow immediately after the AVM was excised. These clamps were removed on the 37th postoperative day, when the size and the configuration of the cerebral arteries returned to normal as indicated by cerebral angiography.

Results
Cortical Blood Flow before and after AVM Excision
The clinical characteristics of the 16 patients are summarized in Table 1. The patients were divided into the
Cerebral Hyperperfusion in High Flow AVM

Table 1. Clinical Summary of Intraoperative Blood Flow Monitoring in Patients with AVMs

<table>
<thead>
<tr>
<th>Group</th>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Size (cm)</th>
<th>Location</th>
<th>Feeding arteries*</th>
<th>Flow modulation</th>
<th>Postoperative complication*</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>46</td>
<td>Male</td>
<td>4.0 x 3.5 x 3.0</td>
<td>Lt. parietal</td>
<td>ACA, MCA</td>
<td>No</td>
<td>ICH</td>
<td>Fair</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>14</td>
<td>Male</td>
<td>4.0 x 3.5 x 2.5</td>
<td>Rt. frontal</td>
<td>ACA, MCA, Heubner a.</td>
<td>No</td>
<td>ICH</td>
<td>Fair</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>42</td>
<td>Male</td>
<td>4.0 x 3.0 x 3.0</td>
<td>Lt. temporal</td>
<td>MCA</td>
<td>No</td>
<td>None</td>
<td>Good</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>62</td>
<td>Male</td>
<td>8.0 x 5.5 x 4.5</td>
<td>Rt. temporal</td>
<td>MCA, Ach, PCA</td>
<td>Yes+</td>
<td>None</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>29</td>
<td>Male</td>
<td>4.0 x 2.5 x 2.5</td>
<td>Rt. parietal</td>
<td>MCA, PCA</td>
<td>Yes+</td>
<td>None</td>
<td>Excellent</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>25</td>
<td>Male</td>
<td>4.0 x 2.0 x 1.5</td>
<td>Lt. parietal</td>
<td>PCA</td>
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<td>Excellent</td>
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<tr>
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<td>7</td>
<td>61</td>
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<td>5.0 x 4.0 x 3.0</td>
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<td>Yes+</td>
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<tr>
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<td>13</td>
<td>Male</td>
<td>1.0 x 1.0 x 1.0</td>
<td>Rt. frontal</td>
<td>ACA</td>
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<td>None</td>
<td>Excellent</td>
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<tr>
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<td>9</td>
<td>29</td>
<td>Female</td>
<td>1.5 x 1.0 x 1.0</td>
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<td>10</td>
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<td>PCA</td>
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<td>None</td>
<td>Excellent</td>
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<tr>
<td></td>
<td>11</td>
<td>38</td>
<td>Female</td>
<td>1.5 x 1.0 x 1.0</td>
<td>Corpus callosum</td>
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<td>None</td>
<td>Excellent</td>
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<tr>
<td></td>
<td>12</td>
<td>20</td>
<td>Male</td>
<td>3.0 x 2.5 x 3.0</td>
<td>Lt. occipital</td>
<td>PCA</td>
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<td>Excellent</td>
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<tr>
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<td>13</td>
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<td>2.5 x 2.0 x 2.5</td>
<td>Lt. cerebellum</td>
<td>AICA, PICA</td>
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<td>None</td>
<td>Excellent</td>
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<tr>
<td></td>
<td>14</td>
<td>39</td>
<td>Male</td>
<td>2.0 x 3.0 x 2.0</td>
<td>Rt. parietal</td>
<td>ACA, MCA, PCA</td>
<td>No</td>
<td>None</td>
<td>Excellent</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>20</td>
<td>Male</td>
<td>2.0 x 3.0 x 2.0</td>
<td>Lt. occipital</td>
<td>PCA</td>
<td>No</td>
<td>None</td>
<td>Excellent</td>
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<tr>
<td></td>
<td>16</td>
<td>23</td>
<td>Male</td>
<td>2.0 x 1.5 x 1.5</td>
<td>Rt. Insular</td>
<td>MCA, LSA</td>
<td>No</td>
<td>None</td>
<td>Good</td>
</tr>
</tbody>
</table>

Note: ACA, anterior cerebral artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; Ach, anterior choroidal artery; LSA, lenticulostriate artery.

Blood flow modulation with constriction of the common carotid artery.

Selective blood flow modulation with constriction of feeding arteries.

following three groups. Group 1 consisted of 3 patients with AVMs that were 4 cm in diameter and no special procedures was used to minimize the increase in cortical blood flow after the AVM was excised. Group 2 consisted of 4 patients with AVMs ranging in diameter from 4 to 8 cm (mean 5.0 cm). In this group the sudden increase in blood flow in the surrounding cortex was modulated by constricting either the dilated carotid artery (case 4) or the proximal segment of the dilated feeding arteries (cases 5–7) until the ratio decreased to below 1.5. Group 3 consisted of 9 patients with AVMs ranging from 1.0 to 3.0 cm (mean 2 cm). Blood flow was not modulated in these patients.

There were two patients in group 1 who developed a postoperative hemorrhagic complication (Cases 1 and 2) even though the complete removal of the AVM was confirmed by an intraoperative or postoperative angiography. The remaining 14 patients had no hemorrhagic complication (Cases 3–16).

Preexcision and postexcision intraoperative cortical blood flow values in each group are shown in Figure 2. A decrease in blood flow was observed in some part of the cortex before excision in cases 1, 5, 6, and 16. Most of the cortex surrounding the AVMs revealed an increase in blood flow after excision in groups 1 and 2.

The relationship between AVM size and the P–P ratio is shown in Figure 2. The P–P ratio increased more than 2.0 as the AVM diameters exceeded 4.0 cm. Two of three group 1 patients developed a postoperative intracerebral hematoma. The flow ratio of these two patients was greater than 1.9. The P–P ratio of group 3 (closed triangle) was below 1.5 except in one case. The mean values and the standard deviations of the ratio of the large AVMs (group 1 plus 2) were 2.2 ± 0.5 and those were significantly higher than group 3 with values of 1.3 ± 0.2 (p < 0.001). These results suggested that the critical P–P ratio above which the postexcision hemorrhage likely to occur is approximately 1.9. Any case with a ratio of less than 1.5 is considered to be low risk for postexcision hemorrhage.

In the group 2 patients, blood flow was reduced from an initial ratio >2.0 to <1.5 using these special clamps described extracranial carotid Selverstone clamps as is shown Figure 3 (downward arrows). Patients treated with these clamps did not develop any hemorrhagic complication.

Discussion

Hyperemic complications, defined as unusual hemorrhage or edema occurring immediately after excision of high-flow AVMs were described as the normal perfusion pressure breakthrough (NPPB) syndrome [26]. The incidence of this complication varied from 1.4 to 21% of
patients. Hemorrhagic events occur approximately in more than one-half of the cases of the NPPB syndrome [22,28], and should be distinguished from severe edema, which can usually be controlled by conservative methods [2,7,12]. The combined mortality and serious morbidity rate of these complications was 50% [4,8,10,22].

Factors hypothesized to contribute to the development of NPPB include (1) large arteriovenous shunts, (2) angiographic evidence of steal, (3) recruitment of the perforating vessels, (4) low cortical blood flow around an AVM, (5) impaired vascular autoregulation, (6) low cortical artery pressure before excision, (7) increased pressure in the cortical arteries after excision, and (8) a sudden and excessive increase in cortical blood flow after excision [3,4,13,15,16,23,24,27].

Various factors will be related to the postoperative hemorrhagic complication. Residual daughter nidus is one of the causes of postoperative hemorrhage. In recent years, we have been routinely using an intraoperative digital subtraction angiography to reduce such a risk. It has been reported that blood flow is significantly decreased in the cortex, which surrounds an AVM [3,11], and that the vascular response to CO\textsubscript{2} is markedly deranged [3,9]. A strong correlation has also been demonstrated between AVM size and the degree of hypoperfusion or dysautoregulation in the surrounding cortex [11,21].

According to our previous report, the rate of satisfactory outcome was significantly higher (88%) in patients with small AVMs than in those with AVMs greater than 4 cm in diameter (63%). In this series of 81 consecutive cases of total AVM removal without preoperative endovascular treatment, no symptomatic hemorrhage occurred with any AVM less than 4 cm in diameter, but did occur with 4 of 22 (18%) AVMs greater than 4 cm in diameter [30]. Furthermore, preoperative carotid Doppler flowmetry demonstrated that blood flow increased gradually as the AVM diameter exceeded 4 cm and approached two times the normal common carotid blood flow when it exceeded 6 cm [31].

It is also known that the pressure of the feeding arteries is low and well below the systemic arterial blood pressure. Following the occlusion of such arteries at their entrance to the AVM, the pressure in their stump rises suddenly [3,6,18]. According to their data, intraoperative monitoring had some prognostic value. However, if a sudden increase in arterial pressure is observed during operation, then a decision concerning the next step will become difficult. Monitoring of cerebral blood flow seems to be more important to protect the surrounding dysautoregulated cortex in such cases.

Recent developments of various intraoperative monitoring systems including laser and thermal blood flow measurement [3,17,29] and Doppler sonography [9,18,20] enable us to monitor cerebral blood flow before, during, and after AVM excision. Intraoperative cortical blood flow measurement in this study demonstrated that the postexcision blood flow increased to more than twice its preexcision values for AVMs greater than 4 cm in size. These results suggest that increased blood flow rate seen after excision correlates closely with AVM size, and is associated with greater risk of postoperative hemorrhagic complications.

To prevent NPPB, the following strategies have been tried: (1) multiple-staged operations, (2) preoperative embolization of the feeding arteries followed by surgical excision, (3) intraoperative embolization combined with feeding artery ligation followed by surgical excision, and (4) ligation of several feeding arteries followed at a later time excision [1,19,21,25].

Gradual stepwise reperfusion of the surrounding ischemic hemisphere after a staged reduction of shunt flow seems a reasonable approach to reestablishing autoregulation. In contrast, the hemodynamic change induced by preoperative embolization may cause an additional risk of ischemic or hemorrhagic complications [5]. Some surgeons also question the benefits of staged surgical excision of an AVM, since this may induce acute intraoperative hypoperfusion superimposed on chronic preoperative hypoperfusion [14,15]. There are few re-
ports published regarding the difference in overall mortality and morbidity among staged operation and one-stage operation with or without preembolization using randomized trials in a reasonable number of cases. Decision making for treatment of high-flow AVMs is still controversial and should be based on understanding cerebral hemodynamics.

Partial occlusion of the carotid artery has previously been used to prevent or to treat NPPB in the one-stage excision of high-flow AVMs [6,22,29]. We have advocated a one-stage operation for large, high-flow AVMs while modulating blood flow with extracranial carotid clamps if the size of the AVMs is larger than 6 cm, because these giant AVMs are usually fed by multiple main arteries and because the extracranial carotid arteries in these cases carry blood at a remarkably high flow rate [30,31]. Selective flow modulation by constricting of the proximal segments of the primary feeding arteries should be indicated if the size of an AVM is between 4 and 6 cm and if the cortical blood flow after excision is more than twice the preexcision value as measured intraoperatively. This flow modulation technique allows the suddenly increased blood flow from dysautoregulated vessels to be redistributed to the intact arterial system. Using intraoperative monitoring, cortical blood flow surrounding the AVM can be reduced to a safe level after excision. Thereby, postoperative hemorrhagic complications due to hyperperfusion after excision of high-flow AVM will be markedly reduced.

Figure 3. The relationship between the size of the AVM and the maximum postexcision/preexcision cortical blood flow ratio (P-P ratio). Group 1: large AVMs without flow modulation (closed box). Group 2: large AVMs with flow modulation after excision of AVMs. Group 3: small AVMs with a diameter of less than 4 cm (closed triangle).

References

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