Intracranial Arteriovenous Malformation: Contralateral Steal Phenomena


Division of Neurological Surgery, *Department of Radiology, and **Division of Epidemiology, the University of Texas Southwestern Medical Center at Dallas, Dallas, Texas, U.S.A.

Abstract

Sixty-two patients with radiographically proven intracranial arteriovenous malformations underwent preoperative regional cerebral blood flow measurement with $^{133}$Xe single-photon emission computed tomography. Contralateral regions of hypoperfusion were detected in all cases. Steal severity was assessed according to the contralateral steal index ($I_{\text{Steal(c)}}$). $I_{\text{Steal(c)}}$ was <0.7 (severe) in 22 (35%), 0.7-0.8 (intermediate) in 18 (29%), and >0.8 (mild) in 22 (35%). $I_{\text{Steal(c)}}$ was more frequently severe or mild in females and more often intermediate in males ($p < 0.05$). Hyperemic complications were encountered more frequently in patients with intermediate $I_{\text{Steal(c)}}$ ($p = 0.086$). An unfavorable outcome was associated with less severe contralateral steal ($p = 0.12$). A detailed clinical, radiographic, and hemodynamic profile may help to preoperatively identify patients at high risk for a poor surgical outcome.

Key words: arteriovenous malformation, cerebral blood flow, cerebrovascular steal

Introduction

Intracranial arteriovenous malformations (AVMs), by virtue of their extremely low resistance characteristics, severely derange cerebral circulatory dynamics. High rates of blood flow with rapid arteriovenous shunting can result in hypoperfusion and ischemia in adjacent brain tissue. Patients who demonstrate clinical evidence of progressive cerebral ischemia and are found to harbor AVMs in corresponding vascular territories are commonly referred for neurological evaluation. Norlen noted that angiographic hypoperfusion of tissue surrounding an AVM can be reversed by resection of the lesion. Feindel and Perot coined the term “steal” to denote absent or delayed angiographic perfusion. Hemodynamic alterations have been documented in the contralateral cerebral tissue of AVM patients, by means of flow velocity measurements as well as regional cerebral blood flow (rCBF) techniques. This study was undertaken to determine the frequency and severity of contralateral hypoperfusion in AVM patients and to ascertain if the clinical and radiographic features are associated with preoperative contralateral steal, as measured by $^{133}$Xe inhalation and single-photon emission computed tomography (SPECT).

Patients and Methods

Between 1983 and 1986, 62 patients with radiographically proven intracranial intradural AVMs were evaluated with SPECT and surgically treated at the University of Texas Southwestern Medical Center. Pre- and postoperative clinical and conventional radiographic data were collected and analyzed. In addition to sex, age, and handedness, presenting signs and symptoms were noted, including intracranial hemorrhage (ICH), seizures, headaches, and progressive neurological deficits.

CT and angiographic studies were evaluated for features that would be expected to influence CBF patterns and surgical results. These data included
location, angiographic AVM diameter (<3 cm, 3–6 cm, >6 cm),33) presence of an intracerebral hematoma greater than 2 cm in diameter, and the number of major feeding vessels, including the middle cerebral artery, anterior cerebral artery, posterior cerebral artery, vertebral artery (including the posterior inferior cerebellar artery), basilar artery (including the anterior inferior and superior cerebellar arteries), and external carotid artery. The recruitment of perforating arteries into the feeding system was noted, as was the pattern of venous drainage: superficial, deep, or both. The sum of the diameters of all angiographically visible feeding arteries was determined by direct measurement, with correction for magnification as each vessel entered the nidus. Angiographic evidence of steal was defined as a paucity of opacified normal vessels adjacent to the nidus in the mid-arterial phase. Redistribution phenomena were not considered evidence of steal, i.e., flow reversal in an A1 segment reverting to normal after resection of a frontal AVM. Perioperative hyperemic complications were liberally defined as the occurrence of unexpected or abnormal degrees of intraoperative brain swelling or hemorrhage unrelated to technical error or occult ventricular hemorrhage. This designation also included postoperative CT evidence of edema unrelated to inadvertent proximal vascular occlusion or brain retraction, and hemorrhage after angiographically proven complete AVM resection. The outcome, determined at the 6-month follow-up examination, was rated as good (capable of independent life), poor (not capable of independent life), or death.

Dynamic SPECT: rCBF was determined by monitoring of the cerebral transit of $^{133}$Xe with the Tomomatic 64 SPECT (Medimatic A/S, Copenhagen, Denmark) designed by Stokely et al.36) It consists of four detector arrays, each containing 16 NaI(T1) scintillation crystals. The arrays are mounted in a hollow square configuration that rotates around the subject's head at 6 rpm. Special focused collimators define three transverse tomographic cross-sections with centers 4 cm apart. $^{133}$Xe was administered in an air/oxygen mixture (10 mCi/l) during the first minute of a 4-minute wash-in/wash-out procedure. During the 4-minute measurement period, activity in the lung was monitored by a scintillation probe placed on the chest; this activity was assumed to correspond to the arterial blood concentration of $^{133}$Xe in the brain. rCBF was calculated in ml/100 gm/min according to the double-integral method described by Kanno and Lassen.14) Celsis et al.6) and Smith et al.32) The reproducibility and reliability of this method have been evaluated in normal subjects by Devous et al.9)

Voxel flow values were displayed in a $64 \times 64$ matrix with a 16-shade scale that can be normalized to the highest flow value. CBF was also numerically recorded, with values characterizing the entire hemisphere. Total brain flow (TBF) was calculated by averaging the right and left hemispheric flow values. rCBF in a hand-drawn region of hypoperfusion in the contralateral hemisphere of each patient was divided by TBF to yield a contralateral steal index [ISteal(c)] as a measure of steal severity, i.e.,

$$I_{\text{Steal}(c)} = \frac{r\text{CBF(steal area)}}{TBF}$$

The system resolution (full width at half maximum), measured with a $^{133}$Xe line source in water, varied from 1.7 cm at the center of each slice to 1.0 cm at the edge transversely. The patients were positioned in the tomograph so that three transverse cross-sections were located 2, 6, and 10 cm above and parallel to the canthomeatal line. These rCBF studies were conducted after informed consent had been given by the patients, in accordance with the policies of the Institutional Review Board of the University of Texas Southwestern Medical Center. rCBF was not determined prior to a 3-week waiting period in patients who had suffered ICH. Statistical significance was determined by the Chi-square test. In the event of small numbers, significance was determined by Fisher's exact test.

Results

I. Clinical profile

Twenty-five (40%) of the patients were under 30 years of age, 28 (45%) were 30–50, and nine (15%) were over 50. Thirty-six (58%) were male. Fifty-nine (95%) were right handed. Twenty-eight AVMs (45%) were right hemispheric, 32 (52%) were left hemispheric, and two (3%) were midline or bilateral. Four AVMs were located in the posterior fossa. Nineteen (31%) were less than 3 cm in diameter, 26 (42%) were 3–6 cm, and 17 (27%) were larger than 6 cm. Thirty patients (48%) presented with ICH and 21 (34%) with progressive deficits. A history of headache was recorded in 11 (18%), and seizure disorder in 27 (44%). Preoperative radiographic studies disclosed a hematoma greater than 2 cm in eight (13%), recruitment of perforators in 19 (31%), and hypoperfusion of surrounding tissue in 23 (37%). The sum of the diameters of all feeding vessels at entry to the AVM nidus was less than 8 mm in 28 (45%), 8–15 mm in 23 (37%), and greater

Neurol Med Chir (Tokyo) 29, May 1989
than 15 mm in 11 (18%). Venous drainage was superficial in 30 (48%), deep in 13 (21%), and both in 19 (31%).

Five patients (8%) were treated only by surgical ligation of feeding vessels or intravascular embolization; definitive resection was not performed because of the patients' wishes or medical contraindications. All other patients underwent radiographically documented microsurgical resection. Hyperemic complications occurred in 13 (21%). The final outcome 6 months postoperatively was good in 51 (82%), poor in four (6%), and death in seven (11%).

II. ISteal(c)

Lower ISteal(c) values were associated with more severe depression of flow in steal regions and higher values (approaching 1.0) reflected less severe flow depression. All patients were found to have a steal region contralateral to the AVM. The distribution of ISteal(c) values is given in Table 1.

The clinical characteristics of these patients were associated with patterns of contralateral steal severity. As shown in Table 2, female patients tended to have either severe [ISteal(c) <0.7] or mild [ISteal(c) >0.8] contralateral steal, whereas males more commonly had intermediate degrees (0.7-0.8) (p < 0.05). Older patients (>50 years) tended to have less severe contralateral steal. In 56% of older patients, ISteal(c) was >0.8, and only 22% had severe contralateral steal. Statistically, however, this group was not significantly different from the other age groups. Patients who had suffered ICH tended to have less severe contralateral steal than those who had not bled. Whereas 43% of patients who presented with progressive deficits had severe contralateral steal, only 32% of those without deficits had similarly low ISteal(c), although this difference was not significant. No relationship was noted between ISteal(c) and a history of headaches or seizures.

The angiographically determined size of the AVM had no significant relationship with contralateral CBF distribution, although 47% of the small AVMs (<3 cm), but only 29% of large AVMs (>6 cm), were accompanied by mild contralateral steal. AVMs with only one major feeding vessel tended to exhibit severe or mild contralateral steal, while 57% of lesions fed by three or more major vessels were associated with intermediate ISteal(c) (p = 0.07) (Table 3). Among patients with intracranial hematomas >2 cm, 63% had ISteal(c) >0.8. Only 31% of patients without intracerebral hematoma had mild contralateral steal. However, the small number of patients with hematomas precluded statistical significance. AVMs with perforator recruitment tended to be associated with intermediate contralateral steal (p = 0.11). The pattern of venous drainage, the sum of diameters of feeding vessels, and angiographic evidence of hypoperfusion surrounding the AVM had no impact on the severity of contralateral steal.

As seen in Table 4, the development of hyperemic complications was associated with intermediate ISteal(c) (p = 0.086). Patients who had good outcomes were fairly evenly distributed over the range of contralateral steal severity (Table 5). Patients with unfavorable outcomes, however, tended to have intermediate or mild ISteal(c) (p = 0.12).

**Discussion**

Cerebral hemodynamics have been studied in AVM

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**Table 1** Preoperative contralateral steal severity (steal index)

<table>
<thead>
<tr>
<th>ISteal(c)</th>
<th>No. of patients</th>
</tr>
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<tbody>
<tr>
<td>&lt;0.7</td>
<td>22 (35%)</td>
</tr>
<tr>
<td>0.7-0.8</td>
<td>18 (29%)</td>
</tr>
<tr>
<td>&gt;0.8</td>
<td>22 (35%)</td>
</tr>
<tr>
<td>Total</td>
<td>62</td>
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Steal index = rCBF (ml/100 gm/min) in hand-drawn region of hypoperfusion divided by TBF.

**Table 2** Relationship of contralateral steal severity to sex

<table>
<thead>
<tr>
<th>ISteal(c)</th>
<th>Female</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.7</td>
<td>12 (46%)</td>
<td>10 (28%)</td>
</tr>
<tr>
<td>0.7-0.8</td>
<td>3 (12%)</td>
<td>15 (42%)</td>
</tr>
<tr>
<td>&gt;0.8</td>
<td>11 (42%)</td>
<td>11 (31%)</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>36</td>
</tr>
</tbody>
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*Neurol Med Chir (Tokyo) 29, May 1989*
patients both invasively and noninvasively. Studies of ipsilateral flow dynamics have demonstrated manifestations of a high flow-low resistance shunt. Blood flow as high as 550 ml/min has been measured in feeding vessels, with flow through the AVM as high as 900 ml/min. This high rate of non-nutritive flow with arterialized venous pressures can lead to hypoperfusion of adjacent brain regions. Pathological changes suggestive of chronic ischemia have also been described.

In some cases, abrupt occlusion of the fistula leads to hyperemia with edema and hemorrhage from dysautoregulated adjacent regions. Radiographic evidence of hypoperfusion, large AVM size, and long, tortuous feeding arteries have all been implicated in the development of these hyperemic states. Recent intraoperative measurements have disclosed mean cortical flow rates several centimeters from the AVM of 43 ml/100 gm/min, which increased to 57 ml/100 gm/min following its excision. Pre-resection cortical flow rates of 18.6-48 ml/100 gm/min were measured with laser Doppler velocimetry, and they increased to 60-168 ml/100 gm/min following resection.

Few data are available regarding the impact of AVM circulatory dynamics on the contralateral hemisphere. The patency of the circle of Willis as well as bihemispheric sharing of superficial and deep venous drainage provide the anatomical substrate for derangement of contralateral CBF. Conventional angiography commonly demonstrates contralateral irrigation of supratentorial AVMs via the anterior communicating artery or the P1 segments of the posterior cerebral artery. Transcranial Doppler studies have confirmed flow reversal in anterior cerebral (A1) segments, reflecting collateral flow from the contralateral side. In AVMs fed by both internal carotid arteries, Pertuiset et al. demonstrated elevated diastolic flow velocities in the contralateral carotid, which normalized after clipping of the anterior communicating artery. Using stable xenon-enhanced CT rCBF measurements, Okabe et al. noted that mean gray matter flow rates contralateral to AVMs were significantly depressed relative to those of control subjects. Using 133Xe inhalation SPECT, Homan et al. noted hypoperfusion in structurally normal brain regions contralateral to the AVM. Also using SPECT, Takeuchi et al. demonstrated focal contralateral hypoperfusion.

While noninvasive three-dimensional rCBF measurement is an ideal means to evaluate contralateral flow dynamics, the clinical implications of detectable steal are uncertain. A recent patient of ours who suffered fatal postoperative hyperemia underwent pre- and postembolization SPECT rCBF measurements as well as pharmacological measurement of vasoreactivity. Interestingly, the contralateral hemisphere mirrored the ipsilateral hemisphere during each step of evaluation procedure, in terms of both rCBF and vasoreactivity.

Patients with occlusive cerebrovascular disease are frequently found to have depressed CBF values contralateral to the infarction. In a recent study involving fluorine-18-fluoromethane inhalation and positron emission tomography, six of seven patients with strictly unilateral cerebrovascular disease and infarction were noted to have rCBF reduction in the contralateral hemisphere. The investigators concluded that transcallosal neuronal disconnection (diaschisis) was the most likely explanation for their findings. It is possible that the consistent finding of ipsilateral steal regions in each of the AVM patients in this series is causally related to the presence of contralateral flow reduction by a similar neural mechanism, with depressed metabolic activity in the ipsilateral steal regions.

In addition to methodological concerns, such as Compton scattered tissue overlap in a SPECT study, the heterogeneity of the AVM population presents problems of interpretation of a steal index. Concerns with the determination of rCBF in a hand-drawn region relate to variations in the size of each steal area as well as the severity of rCBF depression.
denominator in the steal index quotient, TBF, is also jeopardized by the presence of ipsilateral and contralateral steal regions as well as AVM contamination.

It is possible that the apparently lesser severity of contralateral steal in our older patients relates to a diminution of TBF with advancing age, which we have also noted in normal subjects. Similarly, we found patients with ICH to have decreased TBF, even though no CBF studies were obtained within 3 weeks of the most recent bleeding episode. This finding may explain the high incidence of mild contralateral steal in patients who had previously bled. The trend toward less favorable outcomes in patients with less severe steal also may reflect decreased preoperative TBF in those with complicated postoperative courses. The failure of angiographic features to predict contralateral rCBF patterns of hypoperfusion is consistent with our findings in the ipsilateral hemisphere.

Although our understanding of the significance of contralateral hemispheric physiology and hemodynamics is limited in all forms of cerebrovascular disease, SPECT offers a noninvasive, easily reproducible means to quantitatively study these regions, and is capable of monitoring the hemodynamic impact of therapeutic manipulations. Our preliminary data suggest that, in AVM patients, global autoregulatory disturbances may be present that ultimately culminate in hyperemic catastrophe.

Analysis of clinical, radiographic, and CBF data has allowed identification of patients at risk for hyperemic complications and unfavorable outcomes\(^2\)\(^-\)\(^4\) (Table 6).

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### References


Address reprint requests to: H.H. Batjer, M.D., Division of Neurological Surgery, the University of Texas Southwestern Medical Center at Dallas, 5323 Harry Hines Boulevard, Dallas, Texas 75235-8855, U.S.A.

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