Occlusive hyperemia: a theory for the hemodynamic complications following resection of intracerebral arteriovenous malformations

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An alternative theory is proposed to explain the brain edema and hemorrhage that may occur after resection of high-flow intracerebral arteriovenous malformations (AVM's). This theory, termed "occlusive hyperemia," is based on a retrospective analysis of operative dictations along with postoperative imaging studies (191 angiograms and 273 computerized tomography scans) in 295 cases of intracerebral AVM's operated on at the Mayo Clinic between 1970 and 1990. In this series, 34 cases (12%) of postoperative deterioration were documented, of which 15 were due to incomplete resection of the AVM. Of the remaining 19 cases, six had brain edema alone and 13 had hemorrhage with edema, despite complete excision of the AVM. In these 19 cases, the AVM's were greater than 6 cm in diameter in 10 patients, between 3 and 6 cm in six, and less than 3 cm in three. Obstruction of the venous drainage system was observed in 14 (74%) of the 19 cases. Ten of these 14 were due to obstruction of the primary venous drainage of the brain parenchyma immediately surrounding the lesions, while four were due to obstruction of other venous structures. In no case was a rapid circulation identified on postoperative angiograms. The flow pattern was slow or stagnant in former AVM feeders and their parenchymal branches. It is proposed that postoperative intracranial hemorrhage and/or brain edema in AVM patients may be due to: 1) obstruction of the venous outflow system of brain adjacent to the AVM, with subsequent passive hyperemia and engorgement; and 2) stagnant arterial flow in former AVM feeders and their parenchymal branches, with subsequent worsening of the existing hypoperfusion, ischemia, and hemorrhage or edema into these areas. Supportive hemodynamic evidence for this theory was derived from the literature.

KEY WORDS • arteriovenous malformation • cerebral hemodynamics • autoregulation • intracerebral hemorrhage • cerebral venous occlusion • cerebral ischemia

Despite the advances in microneurosurgical techniques to deal with large intracerebral arteriovenous malformations (AVM's), these lesions continue to present a major challenge. One of the recognized complications that may follow the embolization and/or surgical obliteration of large AVM's is the development of brain edema and/or hemorrhage in the normal brain parenchyma that surrounds the bed of the excised AVM, despite the complete obliteration of the AVM nidus. While there is widespread support for the concept that the disruption of local hemodynamics is responsible for these complications, the precise underlying mechanism(s) remain poorly understood. Cerebral hypoperfusion induced by high-flow AVM's (that is, steal) and the subsequent improvement in perfusion after the excision of the AVM was first proposed by Olivcrona and Riives in 1948 and by Norlén in 1949, while the role of cerebral hyperemia in producing neurological disability was hinted at by Gowers as early as 1888. A pioneering theory that has provided a plausible explanation for this phenomenon was proposed in 1978 by Spetzler and coworkers, and was termed "normal perfusion pressure breakthrough." The basic concept of the theory is that the parenchyma surrounding high-flow AVM's is chronically hypoperfused and has an impaired autoregulation mechanism that renders it vulnerable to the restoration of normal perfusion after AVM excision. The combination of disturbed autoregulation and normal perfusion pressure is thought to result in disruption of local capillary beds with subsequent edema and/or hemorrhage. Based on this theory,
staged resection of AVM's was proposed. While the mechanism underlying this theory may contribute to the problem. Spetzler, et al. have often stated that the incidence of normal perfusion pressure breakthrough is small and that it occurs infrequently. This raises the possibility of other mechanisms as the cause(s) for some of these complications.

In this report, a theory is proposed to explain some of the hemodynamic complications of cerebral AVM resections. This theory, termed "occlusive hyperemia," is based on the analysis of records and imaging studies in 293 patients with intracerebral AVM, 19 of whom developed postoperative complications of edema and/or hemorrhage despite the complete excision of the AVM. This theory is supported by hemodynamic evidence presented in the literature.

Clinical Material and Methods

Patient Population

The records and imaging studies in 293 patients with 295 intracerebral parenchymal AVM's or arteriovenous fistulae (AVF's) who underwent surgery at the Mayo Clinic between 1970 and 1990 were reviewed. Patients who had angiographically occult lesions or dorsal AVM's were excluded from the study.

Data Analysis

An analysis of the case histories, operative dictation (initial and/or reoperation), postoperative computerized tomography (CT) scans, postoperative angiograms (when available), and eventual functional outcome formed the basis of this report. Cases in which residual AVM was found to be the cause of postoperative deterioration were considered separately.

On postoperative neuroradiographic studies (CT in 273 cases and angiography in 191 cases) or reoperation, the presence of hemorrhage, edema, residual AVM, and/or occlusion of the venous outflow system were assessed as well as the circulation times in former AVM feeders and their parenchymal branches. Examination of the CT scans and angiograms was performed by a neuroradiologist who was blinded to the clinical outcome in all cases. While pathological specimens were examined for all cases, no meaningful data could be obtained regarding thrombosis because most specimens were coagulated and mutilated at the end of surgery.

Terminology

The various complications developing after total AVM resection are defined as follows.

Slow Circulation Time. This was defined by our neuroradiologists to be greater than 7 to 10 seconds. Exact circulation times for each case could not be determined by this retrospective analysis since precise measurements require the use of timing techniques during angiography.

Impairment of Venous Drainage. This denoted the presence of clot in venous structures as seen on CT scans or lack of filling of the venous vascular tree on postoperative angiograms.

Results

Among the 293 patients with 295 AVM's, postoperative neurological deterioration was documented in 34 cases (12%). Of the 34 cases, 28 had hemorrhage with edema and six had edema without evidence of hemorrhage. This was determined either by CT or angiography or at reoperation.

Incomplete Resection Group

In the 28 patients with postoperative hemorrhage, the AVM resections were incomplete in 15 (determined either by angiography or at reoperation), necessitating a return to the operating room for complete excision. These 15 cases were therefore excluded from the study.

Complete Resection Group

Nineteen cases developed postoperative neurological deterioration despite complete AVM resection. These 19 cases are the subject of the analysis in this study and are referred to as "the complete resection group."

Of the 19 cases, 13 had hemorrhage and edema and six had brain edema alone. Most of these AVM's tended to be large with high flow, with 10 AVM's greater than 6 cm in diameter, six 3 to 6 cm, and three less than 3 cm. The timing of deterioration varied among the cases. Neurological deterioration was observed at less than 3 hours postoperatively in eight cases, between 3 and 24 hours in four, between 1 and 3 days in four, between 4 and 7 days in two, and on the 11th postoperative day in one.

Circulation Time. Review of the postoperative angiograms in the complete resection group consistently revealed average to slow or very slow circulation time, with prolonged vascular transit times of 7 to 10 seconds or greater from the time of injection to the appearance of cortical veins and dural sinuses. No case showed a fast circulation time. In several cases, severe slowing described as "stagnant flow" was observed in former AVM arterial feeders and their parenchymal branches.

Venous Drainage System. Impairment of the venous drainage system from hemorrhage and/or edema was noted in 14 of the 19 cases in the complete resection group (Fig. 1). Impairment in 10 of the 14 cases (eight with hemorrhage and edema and two with edema alone) was due to obstruction of the venous circulation in the brain parenchyma immediately surrounding the bed of the AVM (primary venous drainage). In addition to these 10 cases, two had occlusion of the straight sinus, one a thrombosis in a cortical draining vein, and one a thrombosis of the vein of Galen (nonprimary veins).
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The thrombosis was identified on CT scans in nine cases, on angiography in four cases, and on both in one case. Imaging studies in three other patients who bled due to incomplete AVM resection also showed venous obstruction, while another three patients had asymptomatic thrombi in their venous outflow systems. The relative frequency of asymptomatic thrombi in this series could not be determined from the retrospective analysis.

Clinical Outcome

Results were classified according to four categories: 1) excellent outcome, a patient who had a normal neurological function and enjoyed normal daily activities; 2) good outcome, a patient with a minor neurological deficit functioning normally or with a minor disability; 3) poor outcome, a patient with a significant neurological deficit; and 4) death.

Among the 12 cases of hemorrhage with venous obstruction, 10 were supratentorial AVMs: the eventual outcome was excellent in four, good in four, poor in one, and one death. Of the other two patients, both with posterior fossa AVMs, one had a poor outcome and one died. The one patient with hemorrhage without venous obstruction achieved an excellent outcome. In the six cases with brain edema, two had venous obstruction; outcome was excellent in one and good in one. The other four cases were not associated with venous obstruction and resulted in an excellent outcome in two patients, a good outcome in one, and a poor outcome in one.

Illustrative Cases

Case 1

This 56-year-old man, with a long-standing history of persistent headache, suffered subarachnoid hemorrhage with a left hemiparesis several weeks before transfer to our facility. Angiography revealed a large AVM in the right temporal lobe along with a 7-mm right middle cerebral artery aneurysm (Fig. 2). Comparison of the angiograms and CT scans led to the conclusion that the aneurysm was the probable source of hemorrhage. On arrival, he was found to have a mild left hemiparesis, parietal sensory defect, and a left superior homonymous quadrantanopsia.

Operations. The patient first underwent one-stage particulate embolization in an effort to reduce flow to the malformation. This procedure was followed by a right frontotemporal craniotomy with resection of the AVM and clipping of the middle cerebral artery aneurysm.

Postoperative Course. On awakening from the operation, the patient suffered a generalized seizure. An immediate CT scan revealed the presence of hemorrhage in the bed of the malformation. The patient was returned to the operating room where the clot was removed but no obvious source of bleeding was found. There was marked brain edema. A repeat CT scan showed edema and thrombus in the draining varix (Fig. 3 left). Cerebral angiography confirmed total extirpation of the AVM with no evidence of shunting (Fig. 3 center). The circulation time was very slow, with stagnant flow in former arterial feeders and their parenchymal branches. There was retrograde filling of the right angular artery by pial collateral vessels consistent with a proximal occlusion (Fig. 3 right).

The patient was kept intubated and sedated with barbiturate protection for approximately 72 hours following surgery and thereafter allowed to wake up slowly. During this period, he had several seizures. He was dismissed from the hospital alert, oriented, seizure-free on anticonvulsant therapy, and with the persistent left-sided deficit and homonymous field defect that was present preoperatively. At follow-up examination, he had made a good functional recovery but did not return to his previous employment as a welder.

Comment

Several hemodynamic factors were present in this case: 1) venous outflow system obstruction; 2) stagnant postoperative arterial flow in former AVM feeders and their parenchymal branches, with arterial thrombosis in the angular artery; and 3) marked brain edema and hemorrhage despite complete AVM resection as confirmed on angiography and at reoperation. These fac-
tors suggest the presence of arterial and venous stagnation with secondary hypoperfusion and subsequent ischemia and hemorrhage.

**Case 2**

This 44-year-old physical therapist was being followed for many years at the Mayo Clinic for a known left hemispheric AVM, complicated by severe migraine headaches. Over a period of 6 months prior to this definitive evaluation, she had developed a slowly progressing difficulty with speech and short-term memory. She found herself unable to read a book and retain its contents. Angiography demonstrated a high-flow parenchymal AVF between the left posterior cerebral artery and the draining venous complex in the inferior temporal lobe (Fig. 4).

**Operations.** On the day of surgery, a nondetachable balloon was placed and inflated in the left posterior cerebral artery. This would not remain in place and another balloon was navigated into the lesion. A left subtemporal craniotomy was then performed and the fistula was occluded with two clips, after which the balloon was deflated and removed.

**Postoperative Course.** The patient awoke from surgery with no neurological deficit. On the 3rd postoperative day, she complained of severe headache and blurred vision and suffered a seizure. On examination she was stuporous but arousable, with nystagmus, mild ataxic dysarthria, and poor language comprehension. A CT scan showed marked brain edema with thrombosis of cortical veins near the posterior parietal cortex (Fig. 5 left); no hematoma was seen. Postoperative angiography confirmed complete closure of the fistula with a slow circulation time and stagnant flow in former feeders and their parenchymal branches (Fig. 5 right).

The patient was maintained on Decadron (dexamethasone) and anticonvulsant (Dilantin (phenytoin sodium) and Tegretol (carbamazepine)) therapy, and over the next few days continued to become more alert with no further seizures. At follow-up evaluation, she continued to complain of intermittent vascular headaches and some persistent ataxic dysarthria, but was seizure-free on anticonvulsant therapy, with no focal deficit. Six months following surgery, the patient returned to full employment with no focal deficit and normal mentation.

**Fig. 3.** Case 1. *Left:* Nonenhanced postoperative computerized tomography scan, obtained after evacuation of the hematoma, showing thrombus in the right posterior temporo-occipital draining cortical vein (*arrow*) depicted in Fig. 2. Note the surrounding edema/venous infarction and mild mass effect. *Center:* Postoperative right common carotid angiogram demonstrating no residual arteriovenous malformation (AVM). *Right:* Postoperative left vertebral angiogram demonstrating stagnant arterial flow and no residual AVM. Note retrograde filling of the angular branch of the middle cerebral artery (*arrowhead*).
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FIG. 5. Case 2. Left: Postoperative computerized tomography (CT) scan demonstrating enhancement in the draining veins consistent with stagnant flow and/or thrombosis and low attenuation changes in the parietal lobe consistent with edema/venous infarction. Multiple areas of calcification were present on the preoperative CT (not shown). The low attenuation resolved over a period of several weeks. Right: Postoperative left vertebral angiogram demonstrating successful clipping of the left posterior cerebral artery arteriovenous fistula. Stagnant flow was noted in the left posterior cerebral circulation proximal to the clip.

Comment

In this case marked brain edema with stagnant arterial flow and venous outflow obstruction were quite probably responsible for the deterioration in the patient's neurological function.

Discussion

While there is general agreement that resection of high-flow intracerebral AVM's can be associated with intra- and postoperative complications of brain edema and/or hemorrhage, the underlying mechanisms are still in dispute.

Normal Perfusion Pressure Breakthrough

An attractive explanation for postoperative brain swelling and hemorrhage in AVM patients was proposed by Spetzler, et al.,36 in 1978. As outlined earlier, the theory proposes that the combination of disturbed autoregulation with vasomotor paralysis in the face of normal perfusion results in hyperemia and disruption of local capillary beds and subsequent hemorrhage. This theory was based on four clinical cases as well as an experimental model. While the theory is based on solid physiological principles, its authors have stressed that the risk of normal perfusion pressure breakthrough is small and that it occurred infrequently.34-36 Moreover, several recent studies have questioned the concept of disturbed autoregulation.

Blood Flow Studies

Table 1 lists the various studies that have measured cerebral blood flow (CBF) focally before and after AVM resection. These studies have been performed using different techniques, making direct comparisons inaccurate. This is complicated by the lack of three-dimensional discrimination in the case of xenon measurements; because of the "look-through phenomenon," areas of hypoperfusion are not revealed due to impairment in the delivery of the indicator to the region of interest.

While circulation times assessed in our study could not quantify CBF, no patient who developed postoperative hemorrhage and/or brain edema showed a fast circulation time. On the contrary, the usual pattern was one of slow stagnant flow in former AVM feeders and their parenchymal branches.

Postendarterectomy Reactive Hyperemia

The CBF patterns observed after resection of an AVM are in sharp contrast to the reactive hyperemic states that have been documented to occasionally follow carotid endarterectomy. In the latter, there may be marked increases in CBF and velocity after the correction of the stenosis. In these hyperperfusion syndromes, CBF increases to between 100% and 200% of baseline values.3,30,31,39,40 These hyperemic flows, referred to as "luxury perfusion" by Lassen,19 are manifested by very fast circulation times on postoperative angiograms. Complications associated with reactive hyperemic states are well known and include vascular headaches, paroxysmal lateralizing epileptiform discharges, and catastrophic cerebral edema and hemorrhage.38,39

CO2 Reactivity Studies

The pre-excision CO2 reactivity has been shown by several investigators (Table 2) to be either impaired4,16,31,36 or normal.3,4,44,45 Except for one study that showed an abnormal postexcision CO2 reactivity,4 there is almost unanimous agreement that the postexcision CO2 reactivity is restored to normal.4,16,31,36,44,45 In five of the studies cited,1,16,30,44,45 CO2 reactivity measurements were obtained at two time points during surgery: at the beginning of the craniotomy and at the end of the procedure. In one study,36 in addition to intraoperative measurements, repeat measurements were made at intervals thereafter; in one other study,4 pre- and postoperative measurements were recorded. These observations therefore question the concept of disturbed

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**TABLE 1**

**CBF measurements in brain adjacent to AVM**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Pre-excision</th>
<th>Post-excision</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spetzler, et al., 1978</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Nornes &amp; Grip, 1980</td>
<td>-</td>
<td>increased</td>
</tr>
<tr>
<td>Yamada, 1982</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Okabe, et al., 1983</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Barnett, et al., 1987</td>
<td>decreased (far sites)</td>
<td>normal (near sites)</td>
</tr>
<tr>
<td>Batjer, et al., 1988</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Young, et al., 1983 &amp; 1984</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>Young, et al., 1983 &amp; 1984</td>
<td>normal</td>
<td>normal</td>
</tr>
</tbody>
</table>

*Abbreviations: CBF = cerebral blood flow; AVM = arteriovenous malformation; "normal" = defined by each study; - = no data available.*
autoregulation after AVM excision as measured by CO2 reactivity.

Arterial Stagnation and Obstruction

Abnormalities of AVM feeders are common (Table 2). The observations on postoperative angiograms of stagnant or very slow arterial flow described in this study support the previous studies of a number of investigators.6,21,22 Intraoperative Doppler ultrasonographic examinations by Hassler, et al.15,16 and Petty, et al.,20 showed severe stagnation of flow in former AVM feeders and their parenchymal branches after AVM resection.

Miyasaka, et al.,21 reported very slow flow in former feeders on postoperative angiography; in severe cases of stagnation, retrograde thrombosis of former feeders was observed in five (6.6%) of 76 patients. The incidence increased to 14% when only medium-sized and large AVM's were considered. These authors also found that retrograde thrombosis and severe stagnation were more likely to occur in older patients. Other incidents of stagnation and retrograde thrombosis were reported by Luessenhop and Rosa,20 Pertuiset, et al.,24 and Solomon and Stein.33

Stagnation and occasional feeder thrombosis are likely due to several factors: 1) increased resistance to flow; 2) abnormal endothelium with intimal thickening and elastic and medial degeneration from long-standing high flow-induced mechanical stresses;37 and 3) a reflex arterial vasoconstriction to compensate for normal or increased perfusion pressures in the face of normal autoregulatory mechanisms, not unlike that occasionally seen after carotid endarterectomy.28 While impaired perfusion secondary to arterial stagnation alone can occur, this could be compounded by passive hyperemia and engorgement induced by obstruction to the venous outflow system of brain adjacent to the AVM (see below).

An obvious weakness of this report relates to the fact that postoperative angiograms were not available in all cases for retrospective analysis and, accordingly, the relative frequency of these findings in patients who did not deteriorate following surgery is unknown. This, nevertheless, does not detract from the fact that these findings were prominent in the patients in whom deterioration did occur.

Venous Obstruction

Abnormalities of the venous system in AVM's (Table 3) are also common.1,9,11,13 Yasargil42 reported that 30% of his AVM cases had preoperative abnormalities in their venous drainage and that in large AVM's the incidence was 100%. These abnormalities have taken the form of agenesis, stenosis, or occlusion of major venous sinuses.

Viñuela, et al.,40 studied the preoperative angiograms in 53 cases of deep-seated cerebral AVM's and found vessel wall irregularities and/or stenosis of the system of the vein of Galen in 14 cases and occlusion of the deep venous system in seven cases. It is likely that one reason for hemorrhage from intracranial AVM's in the first place is thrombosis or further obstructive changes in their venous drainage system. In the series of Viñuela, et al., the high incidence of hemorrhage in deep-seated AVM's and the frequency of venous obstruction in these lesions may support this idea.

Albert1 studied the relationship between the number of draining veins of an AVM and the incidence of intracranial hemorrhage. He proposed that the higher the number of draining veins in an AVM, the less likely it is to bleed; for example, AVM's with three or more draining veins were less likely to bleed than those with one or two, further implicating the role of the venous system in AVM-induced hemorrhage.

Nornes and Grip25 reported that draining vein pressures before occlusion were 8 to 23 mm Hg (average 15

### TABLE 2

**Characteristics of AVM feeding arteries as described in various series**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Pre-excision</th>
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<td>morphology</td>
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<tr>
<td>Pertuiset, et al., 1982</td>
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<td>thrombosed</td>
</tr>
<tr>
<td>Luesenhop &amp; Rosa, 1984</td>
<td>-</td>
<td>thrombosed</td>
</tr>
<tr>
<td>Solomon &amp; Stein, 1986</td>
<td>-</td>
<td>thrombosed</td>
</tr>
<tr>
<td>Hassler &amp; Steinmetz, 1987</td>
<td>dilated</td>
<td>dilated, thrombosed</td>
</tr>
<tr>
<td>Miyasaka, et al., 1990</td>
<td>-</td>
<td>thrombosed</td>
</tr>
<tr>
<td>pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nornes &amp; Grip, 1980</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Hassler &amp; Steinmetz, 1987</td>
<td>decreased</td>
<td>normal</td>
</tr>
<tr>
<td>Petty, et al., 1990</td>
<td>increased</td>
<td>decreased</td>
</tr>
<tr>
<td>velocity</td>
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<td></td>
</tr>
<tr>
<td>Nornes &amp; Grip, 1980</td>
<td>increased</td>
<td>-</td>
</tr>
<tr>
<td>Hassler &amp; Steinmetz, 1987</td>
<td>increased</td>
<td>decreased</td>
</tr>
<tr>
<td>Petty, et al., 1990</td>
<td>increased</td>
<td>decreased</td>
</tr>
<tr>
<td>CO2 reactivity</td>
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<td></td>
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<td>Scott, et al., 1978</td>
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<td>-</td>
</tr>
<tr>
<td>Spetzler, et al., 1978</td>
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<td>impaired</td>
</tr>
<tr>
<td>Barnett, et al., 1987</td>
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<td>normal</td>
</tr>
<tr>
<td>Hassler &amp; Steinmetz, 1987</td>
<td>impaired</td>
<td>normal</td>
</tr>
<tr>
<td>Baijjer, et al., 1988</td>
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<td>normal</td>
</tr>
<tr>
<td>Young, et al., 1988</td>
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<td>Young, et al., 1990</td>
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<td>normal</td>
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* Abbreviations: AVM = arteriovenous malformation; "normal" = defined by each study, - = no data available.

### TABLE 3

**Characteristics of AVM draining veins as described in various series**

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<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Pre-excision</th>
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<tr>
<td>morphology</td>
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<tr>
<td>Age &amp; Greer, 1967</td>
<td>stenosis/thrombosis</td>
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<tr>
<td>Garcia, et al., 1975</td>
<td>-</td>
<td>stenosis/thrombosis</td>
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<td>Dobbeliaku, et al., 1979</td>
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<td>-</td>
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<tr>
<td>Brain &amp; Samec, 1983</td>
<td>stenosis/thrombosis</td>
<td>-</td>
</tr>
<tr>
<td>Jomin, et al., 1985</td>
<td>stenosis/thrombosis</td>
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<tr>
<td>Viñuela, et al., 1987</td>
<td>normal</td>
<td>stenosis/thrombosis</td>
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<td>Yasargil, 1987</td>
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<td>-</td>
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<td>pressure</td>
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<tr>
<td>Nornes &amp; Grip, 1980</td>
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</table>

* AVM = arteriovenous malformation; - = no data available.
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**Fig. 6.** Diagrammatic representation of the proposed theory of occlusive hyperemia over three stages: preoperative (A), intraoperative (B), and postoperative (C).

mm Hg) and fell to zero in all patients after the excision of the AVM. They further studied the flow pattern in these draining veins and reported that, in distal portions of the draining veins, regular parabolic patterns with stable laminar flow were seen while the more proximal veins showed highly irregular and turbulent flow. Barnett, et al., observed elevated pressures in draining veins before AVM occlusion and rapid reduction after occlusion. Local endothelial damage in AVM draining veins due to pressure and shearing stress was described by Fry. Such changes are likely to predispose to increased incidence of stenosis or thrombotic occlusion. This is especially likely after AVM excision, as the pressure in the draining veins drops to zero. Venous thrombosis may also be compounded by embolization from the bed of the AVM during excision or inadvertent therapeutic embolization intended for the AVM feeders.

More recently, Bederson, et al. provided experimental data that emphasized the importance of venous outflow obstruction and venous hypertension to the complications associated with intracerebral AVF's. Despite detailed angiography of the venous drainage system, it is not always possible to detect abnormalities. Nevertheless, it is important not only to identify the presence of irregularity and/or occlusion but also to differentiate veins that drain the AVM as well as the surrounding brain parenchyma. Inadvertent embolization or surgical ligation of these veins is likely to precipitate passive hyperemia and engorgement with subsequent hemorrhage and brain edema and a secondary reduction in arterial perfusion.

**Risk Factors for Occlusive Hyperemia**

There are a number of risk factors that could be postulated for the development of occlusive hyperemia: 1) alterations in velocity of venous flow, particularly in veins that were converted by surgery from high-flow to low-flow conduits; 2) pre-existing venous narrowing or occlusion; 3) a small number of draining veins; 4) large and high-flow AVM's; 5) long and tortuous feeders that are subject to retrograde thrombosis; 6) preoperative clinical or radiographic evidence of a steal; 7) location of the AVM in the watershed zone(s) between the three major arterial territories, resulting in increased risk of hypoperfusion and ischemia; and 8) endothelial damage in AVM draining veins due to pressure and shearing stress. A diagrammatic representation of the theory of occlusive hyperemia is shown in Fig. 6.

**Clinical Implications**

One of the consequences of the normal perfusion pressure breakthrough theory was the recommendation for hemodynamic reasons of staged surgical resection of AVM's. Given the right hemodynamic circumstances and good surgery, resecting AVM's in one or more stages is likely to result in a favorable outcome. If, however, occlusive hyperemia is a greater risk than normal perfusion pressure breakthrough, then surgical staging may have fewer indications other than those for technical considerations.

Whether the AVM resection is accomplished in one, or multiple stages, with or without preoperative embolization, the following precautions may reduce the risk of occlusive hyperemia: 1) care to avoid injury of veins that drain both the AVM and the surrounding brain parenchyma; 2) clipping of the terminal arteries as close to the malformation as possible and protection of the nutrient transient vessels that supply normal brain beyond the malformation; and 3) avoidance of excessive intravascular hypotension because, while moderate intraoperative hypotension (70 to 80 mm Hg) is useful in some instances (such as during the coagulation of feeders from deep friable perforators), critical drops in the perfusion pressure could have undesirable ischemic consequences in the precarious surrounding parenchyma in which autoregulatory mechanisms have already resulted in maximum arterial dilatation. This could also predispose to sludging, aggregation, and clumping of formed elements (such as platelet aggregates and red blood cells) in the venous outflow system.

**Conclusions**

Based on our observations and the evidence cited, we conclude that the complications of hemorrhage and/or edema associated with the resection of high-flow
AVMs’s are the result of two separate but interrelated mechanisms involving both the arterial feeders and the venous drainage systems. These mechanisms are: 1) stagnant arterial flow in former AVM feeders and their parenchymal branches with subsequent worsening of the existing hypoperfusion and ischemia and subsequent hemorrhage and/or edema into these areas; and 2) obstruction of the venous outflow system of brain adjacent to the AVM with subsequent passive hyperemia, engorgement, and further arterial stagnation. In addition, disturbed autoregulation at the microcirculation level may play a role.

These mechanisms, however, do not negate the importance of immaculate surgical technique in the complete excision of the AVM, of minimizing trauma to the surrounding brain parenchyma, and of care in handling small nutrient vessels or thin-walled coagulated vessels around the AVM, which are inherently weakened by long-standing high-flow stresses. In our experience, these represent the most frequent source of the postoperative hemorrhage.

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