Cerebral hemodynamics in angioma patients: an intraoperative study

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Local hemodynamics were investigated during 33 operations for cerebral arteriovenous malformation (AVM). In all cases, microvascular Doppler sonography was used to measure flow velocities and vasomotor reactivity to CO2 changes. Intravascular pressure recordings were performed in six patients. The AVM feeders had low intravascular pressure, high flow velocity, low peripheral stream resistance, and very poor vasomotor reactivity. Remote brain arteries showed no abnormalities. Doppler findings in arterial branches of AVM feeders that supplied normal brain indicated arteriolar dilation in their peripheral distribution. On removal of the angiomas, the arteries that formerly supplied them showed a return to normal intravascular pressure, whereas flow velocities dropped far below normal in these vessels. Remote arteries and branches of the former AVM feeders supplying the brain did not show any signs of impaired vasomotor reactivity following angioma removal. The results are in contrast to the normal perfusion pressure breakthrough theory.

KEY WORDS • cerebral angioma • cerebral hemodynamics • autoregulation

The hemodynamic aspects of cerebral arteriovenous malformations (AVM's) are currently the subject of increasing interest. Recently developed methods such as single-photon emission tomography, transcranial Doppler sonography, and superselective angiographic catheterization have promoted a more dynamic diagnostic and therapeutic approach to the problem.

Anatomically, the angioma nidus consists of primitive shunting vessels measuring 50 to 200 µ in diameter.18 This system is supplied by a number of AVM-feeding arteries which also emit "normal" nutrifying arteries to adjacent brain regions. Hemodynamically, the AVM-feeding arterial system is characterized by low intravascular pressures15 due to abnormally low stream resistance in the angioma nidus, which lacks vascular autoregulation. The intravascular pressure in brain-nutrifying branches of the angioma feeders is therefore also low. According to the model of Nornes and Grip,15 pressure in such branches decreases progressively the more distally they branch off the feeder. As a result of downstream autoregulation, the arterioles should be dilated in order to prevent underperfusion in such critical areas. Intracerebral steal phenomena are expected to occur when the autoregulative capacity of these resistance vessels is exhausted and fails to compensate further for low intravascular pressure. However, this phenomenon has been demonstrated by only a few authors.2,6,9,12,20

With the surgical occlusion of AVM feeders, pressure in these vessels increases,15 leading to "normal perfusion pressure" of adjacent brain regions supplied by branches of the former AVM feeders. Spetzler, et al.,21 described a "normal perfusion pressure breakthrough theory" based on experimental findings; according to this, arterioles in these areas may be unable to constrict after protracted previous dilation. Arterial pressure would then break through into the capillary bed and probably result in brain swelling and/or hemorrhage (Fig. 1) — events that are still the major complications in angioma surgery. In this paper, we contribute to the discussion about the pathophysiological concepts in this important field.

Clinical Material and Methods

Operative Procedure

Intraoperative studies were performed in six small (< 2 cm in greatest angiographic diameter), 18 medium-sized (2 to 4 cm), and 10 large (> 4 cm) cerebral angiomas. Measurements were carried out immediately after exposure of the AVM, during stepwise occlusion of feeding arteries, and following AVM removal. In all cases, Doppler sonographic examinations of blood flow
TABLE 1

Arterial pressures before and after AVM removal correlated with AVM size and the length and diameter of feeding arteries*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Location</th>
<th>Diameter† (cm)</th>
<th>Volume† (cu cm)</th>
<th>Artery</th>
<th>Feeder Pressure Pre-AVM Removal</th>
<th>Vessel Stump Pressure</th>
<th>% Radial Artery Pressure Increase (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>parietal</td>
<td>3.0 x 2.0 x 1.5</td>
<td>9.9</td>
<td>MCA</td>
<td>2.7</td>
<td>16</td>
<td>73</td>
</tr>
<tr>
<td>2</td>
<td>temporoparietal</td>
<td>4.7 x 3.2 x 4.0</td>
<td>60.2</td>
<td>MCA, ACA, PCA</td>
<td>(MCA)</td>
<td>12</td>
<td>96</td>
</tr>
<tr>
<td>3</td>
<td>temporoparietal</td>
<td>5.0 x 4.8 x 3.8</td>
<td>91.2</td>
<td>MCA</td>
<td>4.0</td>
<td>8</td>
<td>76</td>
</tr>
<tr>
<td>4</td>
<td>temporo-occipital</td>
<td>4.0 x 4.5 x 5.0</td>
<td>90.0</td>
<td>MCA, PCA</td>
<td>(MCA)</td>
<td>10</td>
<td>71</td>
</tr>
<tr>
<td>5</td>
<td>parietal</td>
<td>6.0 x 3.5 x 4.2</td>
<td>88.2</td>
<td>MCA, ACA</td>
<td>3.5</td>
<td>17</td>
<td>85</td>
</tr>
<tr>
<td>6</td>
<td>temporal</td>
<td>7.0 x 5.5 x 4.0</td>
<td>154.0</td>
<td>MCA</td>
<td>5.0</td>
<td>7</td>
<td>77</td>
</tr>
</tbody>
</table>

* Arterial pressures: intravascular systolic = diastolic mean pressures. AVM = arteriovenous malformation; MCA = middle cerebral artery; ACA = anterior cerebral artery; PCA = posterior cerebral artery.
† Data obtained angiographically.

velocity and CO₂ reactivity in vessels supplying brain and/or the angioma were performed. In six of these patients additional recordings were made of intravascular pressure in the AVM feeding arteries. The angiomas were carefully dissected as far as possible from the surrounding tissue without early occlusion of the main feeding or draining vessels. After dissection, the measurements were again performed. The feeders were then excluded one by one to allow time for the brain vessels to adjust to the changing hemodynamic situation. Measurements were repeated during and after this procedure.

**Intravascular Pressure Measurement**

Simultaneous recordings of intravascular pressure were performed in the radial artery and in angioma feeders within 1 cm before entry into the AVM. The vessels were punctured using 0.45 x 13-mm needles (much less than half the vessel diameter). Systolic, diastolic, and mean pressure values were recorded and displayed on a Hellige monitor. Feeding arteries were punctured before and after AVM removal.

**Measurement of Blood Flow Velocity**

The application of intraoperative Doppler ultrasound for the measurement of blood flow velocity in single intracranial vessels was first reported by Norrnes, et al.,15,16 using 6- and 10-MHz pulsed systems. This study was performed using a device* originally developed by Cathignol, et al.3,4,5 The high pulsed transmitter frequency of 20 MHz provides this system with a good resolution. Pulse durations are 250/450/850 nsec, axial resolution 0.4/0.7/1.3 mm, lateral resolution 0.5/1.1

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* Microvascular Doppler ultrasonograph, Model MF 20, manufactured by EME, Überlingen, Federal Republic of Germany.
FIG. 2. Intraoperative Doppler sonograms showing blood flow velocities before removal of the arteriovenous malformation. Typical tracings are illustrated from feeding arteries 1, 2, 3 and 4 with high systolic and diastolic velocities and low pulsatility. Draining veins 7 and 8 show arterialized spectra. A remote brain artery (9) not originating from a feeder has normal Doppler patterns, whereas the nutrifying distal branch of the feeding artery (5) shows relatively elevated diastolic velocity.

mm, and pulse repetition frequencies 25/50/100 kHz. Depth of measurement can be adjusted in steps of 0.1 mm to a maximum of 15 mm. Due to the filter arrangements, the upper limit of the recording range was at 12.5 kHz of Doppler shift. The spectra were evaluated by a real-time frequency analyzer.†

Intraoperative application of the miniaturized autoclavable probes measuring 10 mm in length and 1 or 2 mm in diameter was quite simple. The angle of insonation between the instrument-held probes and the vessel was set between 40° and 60°; probe position and gate depth were adjusted visually and under acoustic control of the signals until the highest frequencies were obtained. The smallest vessels thus evaluated measured 1 mm in diameter. Application of this Doppler technique in neurosurgery was first described by Gilsbach for operations on aneurysms and extracranial-intracranial bypass procedures.

According to Pourcelot, the relationship between the Doppler sonographic systolic peak velocity and the end-diastolic velocity of an artery is a measure of the vascular resistance in the vessel's peripheral distribution. The ratio between end-diastolic (D) and systolic (S) velocity amplitude is normally 0.43 to 0.47:1 in intracranial arteries. According to Pourcelot's formula $R = (S - D)/S$, the resistance index (R) depends mainly on the relative magnitude of the end-diastolic velocity.
Cerebral hemodynamics

A low R value corresponds to a low resistance, so that relatively elevated end-diastolic velocities indicate low peripheral stream resistance.

**Measurement of \( CO_2 \) Reactivity of Blood Flow Velocity**

Cerebral blood flow (CBF) is greatly influenced by the arterial \( CO_2 \) acting upon the resistance vessels. Hypercapnia results in arteriolar dilation and CBF increase, whereas in hypocapnia the arterioles constrict and CBF decreases. As the diameter of large cerebral arteries does not react to \( CO_2 \), the flow in these vessels accelerates during hypercapnia and decelerates during hypocapnia. Changes of CBF measured by nitrous oxide uptake and xenon-133 clearance run almost parallel to the reported velocity changes in cerebral arteries demonstrated by transcranial Doppler sonography. Velocity changes in these vessels are therefore considered to correlate strongly with CBF changes at varying arterial \( CO_2 \) levels. Thus, Doppler studies of \( CO_2 \) reactivity represent a valuable means for the assessment of hemodynamic situations by determining the vasomotor capacity of arterial systems.

For the intraoperative studies of \( CO_2 \) reactivity, the end-tidal partial volume of \( CO_2 \) in the expired air was

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**FIG. 3.** Intraoperative Doppler sonograms showing flow velocities in response to a \( CO_2 \) reactivity test. The arteriovenous malformation-feeding artery (1) exhibits flow acceleration during hypercapnia and no response to hypocapnia. There is normal reactivity of a remote artery (9). The response to hypocapnia in distal nutrifying branch 5 of the feeder is impaired.
FIG. 4. Intraoperative Doppler sonograms following arteriovenous malformation removal. There are low flow velocities in the former feeding artery (1 and 2), which maintains its previous diameter. The pattern of the remote artery (9) remains unchanged. The nutrifying distal branch (5) of the former feeding artery shows flow deceleration when compared to Fig. 2. Increased stream resistance in this vessel is indicated by the waveform (see text). P = pressure.

documented using infrared analysis.‡ This parameter is widely accepted as an indirect measure of the arterial CO₂ pressure (PaCO₂). Doppler curves were documented each time a desired steady state of PaCO₂ had been reached.

Results

Intravascular Pressure

Intravascular pressure measurements were made in six angiomas of varying sizes. Mean pressures in the feeding branches ranged between 45.1% and 61.8% of the mean systemic arterial blood pressure (SAP) measured in the radial artery. Normally, these values are approximately 90%.¹ Pressure pulsations were also much lower than in the systemic circulation. The pressure losses in the AVM feeders compared to SAP did not correlate with angioma size or with the length or diameter of the evaluated feeding vessel (Table 1).

After AVM removal, the measurements were repeated at the same recording sites. The pressures had increased by 52.3% to 68.7% (Table 1) to about 90% of the SAP values which, along with PaCO₂, had been held stable. The pressure increases in the vessel stumps of former feeders did not depend on the size of the resected AVM or feeder length.

Flow Velocity Before AVM Removal

Figure 2 shows flow velocity tracings before removal of the AVM. Angioma-feeding arteries exhibit very high flow velocities that, in the case of large feeders, often exceed the recording range of the Doppler MF 20 ultrasonography device. Doppler spectra are elevated in their systolic components and are even more pronounced in their diastolic components, thus indicating low peripheral stream resistance. Brain-supplying arteries not issuing from AVM feeders show normal Doppler patterns upon intraoperative investigation. Evaluation of brain-nutrifying branches of the angioma feeders, however, reveals slightly abnormal spectra with high diastolic and end-diastolic velocities. This flow pattern indicates reduced stream resistance in the peripheral vascular bed of these arteries.

‡ Normocap infrared device manufactured by Datex Instruments, Helsinki, Finland.
**Cerebral hemodynamics**

**CO₂ Reactivity Before AVM Removal**

Figure 3 shows the CO₂ reactivity pattern before removal of the AVM. Responses to PaCO₂ changes in the angioma-feeding arteries are almost abolished. Velocities only slightly increase with hypercapnia and remain stable in hypocapnia. In contrast, normal brain arteries not originating from AVM feeders have normal reactivities with marked hypocapnic flow deceleration and hypercapnic acceleration, indicating undisturbed vasomotor reactivity in their peripheral distribution. Brain-supplying arterial branches of the feeders demonstrate almost normal flow acceleration in hypercapnia but clearly impaired reduction in hypocapnia due to a lack of arteriolar constriction in their peripheral distribution.

**Flow Velocity After AVM Removal**

With advancing exclusion of angioma feeders, the flow velocities in these vessels drop (Fig. 4). After removal of the malformation, velocities in the vessel stumps (former AVM feeders) are extremely low. Doppler patterns are characterized by small systolic peaks and very low diastolic and sometimes unmeasurable end-diastolic signals indicating high peripheral stream resistance. Normal brain arteries not issuing from former AVM feeders maintain normal flow patterns. Doppler waveforms from brain-supplying arterial branches of former AVM feeders, however, indicate slightly elevated peripheral stream resistance. Systolic peaks are steep and end-diastolic velocities are lower than before AVM removal. Flow accelerations were never observed in such vessels (Fig. 4).

**CO₂ Reactivity After AVM Removal**

The normal vasomotor response of all brain-supplying arteries to PaCO₂ changes is established immediately following angioma removal (Fig. 5). This applies in particular to arterial branches of the vessels that formerly fed the AVM. Previously impaired hypocapnic vasomotor responses in these vessels immediately normalized in all cases and even tended to overshoot following occlusion of the angioma feeders (Fig. 6).

**Discussion**

The basic assumption in theories about cerebral hemodynamics in angioma patients is that resistance...
FIG. 6. Intraoperative Doppler sonograms from Case 6 (Table 1) showing normal CO2 reactivity in the brain-nutritiving distal branch of the feeding artery following removal of a large angioma with 66% pressure increase in the arterial vessel stump. The PaCO2 values are indicated above. There is a typical finding with normal vasomotor reaction in this artery, which was previously less responsive. Note that the flow velocity in the small brain-draining vein also increases under hypercapnia and that a reflux occurs in the venous blind duct during inspiration. P = pressure; MCA = middle cerebral artery; TS = transverse sinus.

and pressure are lowered in the AVM feeding system. The intravascular measurements presented confirm the finding described by Nornes and Grip15 of abnormally low pressure in AVM feeders that rises by about 60% to normal values with surgical occlusion of these vessels. Analysis of the Doppler waveforms obtained from the feeders (resistance index) further suggests marked reduction of vascular resistance in the angioma nidus. Severely impaired Doppler responses to PaCO2 changes demonstrate a lack of vasomotor reactivity in these vessels. Following removal of the angioma, flow velocity in the vessel stumps is very low. Doppler waveforms suggest raised peripheral stream resistance while the previous vessel diameters remain unchanged. This corresponds to observations in postoperative angiography, where former feeders remain enlarged up to the 3rd postoperative week and continue to be visible often throughout the venous angiographic phase (“stagnating arteries,” Fig. 7).

Cerebral arteries not originating from angioma feeders do not show any abnormality on intraoperative investigation. Flow velocities, Doppler waveforms, and vasomotor reactivity to PaCO2 changes are normal before, during, and after angioma removal. Brain-nutritifying distal branches of the low-pressure angioma feeders, however, show some interesting characteristics in relation to the concept of normal perfusion pressure breakthrough. Doppler patterns of these vessels are initially characterized by relatively high diastolic velocities, a factor which indicates lowered vascular resist-
Cerebral hemodynamics

FIG. 7. Intraoperative recordings of intravascular pressure, Doppler sonograms of flow velocity, diagrams of arterial diameter, and pre- and postoperative angiograms in Case 1 (Table 1). The artery feeding a small arteriovenous malformation (AVM) becomes a “stagnating artery” following angioma removal. Note that the pressure increase in the arterial vessel stump is similar to that found in larger AVM’s.

ance in their peripheral distribution. This is explained by a dilation of arterioles compensating for abnormally low intravascular pressure. As further demonstrated by the CO₂ tests, these arterioles fail to constrict under hypocapnic conditions; that is, the autoregulative force of hypotension prevails over the influence of CO₂. This of course does not prove intracerebral steal, but demonstrates intact autoregulation in such critical areas. According to the theory propounded by Spetzler, et al., the long-standing dilation of resistance vessels may render them “paralytic” once normal perfusion pressure is surgically established. The breakthrough phenomenon should therefore be expected earliest in the brain regions adjacent to the former AVM’s which are supplied by distal branches of the previous feeders. Doppler spectra of these vessels, however, do not indicate hyperperfusion or vasoparalysis following angioma removal. Flow velocities decrease, waveforms indicate rising peripheral resistance, and (more important) the vasomotor responses to PaCO₂ changes are immediately established at the end of the operation. Previously dilated arterioles then constrict during hypocapnia and dilate during hypercapnia, which would be unthinkable in the case of a breakthrough situation. This was a uniform experience in investigations on AVM’s of varying size.

Theoretically, the occurrence of a breakthrough should mainly depend on the degree of preocclusive intra-arterial hypotension and the surgically induced pressure increase in vessels supplying the critical brain regions adjacent to the AVM. There is no indication from the data of Nornes and Grip or from the pressure measurements presented here that flow rate, flow velocity of AVM feeders, or the actual AVM size influence these parameters. Feeders of malformations of variable size show similar preocclusive pressure losses as well as postocclusive increases (Table 1). This finding corresponds to the impression of Nornes and Grip, who believed that the length of the feeders rather than the AVM shunt rate might be the crucial parameter for the occurrence of a breakthrough. However, all cases presented here had comparably long feeders because the measurements could only be performed in superficially located angiomas. The results therefore question the basic assumption of the “normal perfusion pressure breakthrough theory.”

Surgical complications such as brain swelling and hemorrhage may well be due to other factors. Thin-
walled coagulated vessel stumps in the surgical site may rupture under rising arterial pressure in the postoperative situation or be subject to lytic processes. Problems might also evolve from thrombotic infarction. As the flow in former AVM feeding and draining vessels is close to zero following surgery (Fig. 8), arterial and/or venous thromboses are conceivable and may lead to ischemia, swelling, and secondary hemorrhage.

References

Fig. 8. Intraoperative Doppler sonograms and arterial pressure tracings before and after angioma removal. Changes in local arterial pressure (CAP) and flow velocity occurring with occlusion of the last feeding artery are shown. SAP = systemic arterial pressure.
Cerebral hemodynamics


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