Evaluation of cerebral AVM's using transcranial Doppler ultrasound

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Blood flow velocities in basal cerebral arteries were recorded noninvasively in 28 patients with cerebral arteriovenous malformations (AVM's) and were correlated with the angiographic findings. In normal arteries remote from the AVM, flow velocities ranged from 44 to 94 cm/sec (median 65 cm/sec) with pulsatility indexes from 0.65 to 1.10 (median 0.87). This is consistent with findings in normal individuals. Arteries feeding the AVM's were identified by the high flow velocities (ranging from 75 to 237 cm/sec, median 124 cm/sec). The pulsatility index ranged from 0.22 to 0.74 (median 0.48). The difference of these results from findings in normal remote arteries was highly significant (p < 0.001). Hyperventilation tests illustrated the hemodynamic difference between an AVM and normal cerebrovascular beds.

Flow velocity measurements permitted noninvasive diagnosis of AVM's in 26 of the 28 patients. Furthermore, the identification of individual feeding arteries permitted good definition of the anatomical localization of individual AVM's. Flow velocity measurements combined with computerized tomography scans are useful in the diagnosis of AVM's. With the feeding artery's configuration identified on angiography, flow velocity measurements permit a new insight into the "hemodynamic dimension" of an AVM and its possible effects on adjacent normal brain-tissue perfusion in the individual patient.

KEY WORDS □ arteriovenous malformation □ basal artery □ cerebral artery □ ultrasound □ blood flow velocity
characteristics of AVM’s and normal remote vascular beds.

The intention of the present study was to investigate
the hemodynamics of AVM-feeding vessels by means
of transcranial Doppler ultrasound and to compare
these findings to recordings from normal remote cere-
bral vessels shown angiographically as not involved in
the AVM complex. Emphasis was placed on practical
methods for the clinical assessment of individual he-
modynamic states.

Theoretical Considerations

There is an increased viscous drag in AVM-feeding
arteries because of increased flow velocities. As a con-
sequence, blood pressure in these feeders is lower than
in normal remote arteries. However, the problems
encountered when attempting to describe arterial blood
flow in exact fluid dynamic terms are considerable. For
practical working purposes, the application of relatively
simple physical principles nevertheless reveals some
clues to the understanding of important hemodynamic
mechanisms. In steady nonpulsatile parabolic flow, the
pressure loss due to viscous drag \( \Delta P \), at volume flow \( Q \),
through a length of tube \( L \), with the radius \( r \), is ex-
pressed by the Hagen-Poiseuille equation as follows:

\[
\Delta P = Q(8L\rho/\pi r^4),
\]

with \( \rho \) the viscosity coefficient. Substituting for \( Q \) the
lumen area multiplied by cross-sectional average flow
velocity \( \bar{V} \):

\[
Q = \bar{V}(\pi r^2)
\]
gives

\[
\Delta P = \bar{V}(8L\rho/r^2).
\]

The pressure loss due to viscous drag is proportional to
flow velocity and the length of the conduit and is
inversely related to the square of the lumen radius. For
calculations of \( \Delta P \), Equations 1 and 3 require correction
in view of the following considerations: 1) brain arteries
are not infinitely long and unbranched tubes; 2) blood has complicated rheological properties; and 3) arterial
flow is not steady but pulsatile. Moreover, in transcran-
ial Doppler investigations the outline of the recorded
flow velocity spectrum (that is, the instantaneous max-
imum flow velocity) is actually being measured. The
cross-sectional average flow velocity in intracranial ar-
teries (ACA’s), and posterior cerebral arteries (PCA’s)
and from the distal extracranial internal carotid artery
(ICA) have been described in previous reports. Flow
velocities were measured as being the time-mean value
of the Doppler velocity spectrum outline. The Doppler
pulsatility index of the velocity spectrum outline (sys-
tolic velocity minus diastolic velocity divided by the
time-mean value) was used to further describe each
recording. All values were the averages from 10 con-
ceutive cardiac cycles.

The Wilcoxon rank test for two samples was used for
statistical evaluation of differences.

Hyperventilation Test

The 11 most recent patients were investigated further
while end-tidal CO\(_2\) from expired air (PaCO\(_2\)) was mon-
tored using an infrared CO\(_2\) analyzer. Five of these
patients had a medium-sized AVM and six had a large
AVM. Flow velocity and end-tidal PaCO\(_2\) levels from
expired air were determined at normoventilation (rest-
ing values). The patients were then instructed to hyper-
ventilate. The intention was to achieve and secure
steady-state hyperventilation, which is essential for the
estimation of arterial CO\(_2\) partial pressure from end-
tidal CO\(_2\) measurements. Strenuous hyperventilation
was discouraged.

* Sirecust System 400 with CO\(_2\) analyzer module manu-
factured by Siemens AG, D-8520 Erlangen, Federal Republic
of Germany.
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FIG. 1. Angiograms in a 27-year-old man. Left: Right carotid angiogram showing a large arteriovenous malformation of the right parietal region filling from the enlarged nontapering right middle and posterior cerebral arteries. Right: Left carotid angiogram showing cross-filling to the right middle cerebral artery (MCA) through the anterior circle of Willis. The left MCA illustrates progressive lumen reduction toward the periphery, which is characteristic of normal brain arteries.

The blood pressure was measured using a conventional manometric cuff. Mean blood pressure was defined as the diastolic value plus one-third of the amplitude value, using the average from three successive readings.

Results

The results from angiography and Doppler investigation were interpreted independently.

Normal Arteries

In 27 patients, the MCA contralateral to the AVM represented normal remote intracranial arteries shown angiographically as not involved in the AVM complex. The PCA recording was used in one patient with a bilateral subfrontal AVM. Flow velocity levels ranged from 44 to 94 cm/sec (median 65 cm/sec). The pulsatility index values ranged from 0.65 to 1.10 (median 0.87). In the 14 patients in whom one ICA was not associated with the AVM, the ratio of MCA flow velocity to the ICA flow velocity on the same side (\(V_{MCA}/V_{ICA}\)) was from 1.1:1 to 2.1:1 (median 1.5:1). These findings are within normal ranges (Tables 1 and 2). There was no significant difference between patients with medium and large AVM's (p > 0.2).

AVM-Feeding Arteries

Recordings from intracranial arteries feeding medium-sized or large AVM's showed flow velocities from 75 to 237 cm/sec (median 124 cm/sec), with pulsatility index values ranging from 0.22 to 0.74 (median 0.48). The difference between these findings and those from normal remote arteries is highly significant (p > 0.001).

### TABLE 1

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Range</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA flow velocity (cm/sec)</td>
<td>52–88</td>
<td>67</td>
</tr>
<tr>
<td>MCA flow velocity (%)</td>
<td>84–116</td>
<td>100</td>
</tr>
<tr>
<td>MCA pulsatility index (%)</td>
<td>0.54–0.89</td>
<td>0.72</td>
</tr>
<tr>
<td>MCA pulsatility index (%)</td>
<td>93–107</td>
<td>100</td>
</tr>
<tr>
<td>ICA flow velocity (cm/sec)</td>
<td>32–54</td>
<td>42</td>
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<tr>
<td>ratio (V_{MCA}/V_{ICA})</td>
<td>1.3–2.0</td>
<td>1.6</td>
</tr>
</tbody>
</table>

* MCA = middle cerebral artery; ICA = distal extracranial internal carotid artery; \(V\) = flow velocity. Values adapted from Lindegaard, et al.\(^1\) Percentages denote flow velocity and pulsatility index values from the left MCA expressed as a percentage of flow velocity and pulsatility index values for the right MCA in individual patients.

### TABLE 2

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Normal Remote MCA Feeders</th>
<th>Tapering AVM Feeders</th>
<th>Nontapering AVM Feeders</th>
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<tbody>
<tr>
<td>flow velocity (cm/sec)</td>
<td>Range 44–94</td>
<td>Median 65</td>
<td>Range 75–124</td>
</tr>
<tr>
<td>flow velocity (%)</td>
<td>Range 0.65–1.10</td>
<td>Median 0.87</td>
<td>Range 0.41–0.74</td>
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<td>pulsatility index</td>
<td>Range 52–79</td>
<td>Median 67</td>
<td>Range 32–66</td>
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</table>

* Data for four patients with recent arteriovenous malformation (AVM) hemorrhage and two others with small AVM's are not included. MCA = middle cerebral artery. Percentages denote AVM feeding-artery flow velocity and pulsatility index values expressed as percentages of values recorded from the normal remote MCA in individual patients.
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FIG. 2. Flow velocity recordings from the same patient illustrated in Fig. 1. The recordings from the right middle and posterior cerebral arteries (MCA and PCA) show very high flow velocities (upper and lower left). The low pulsatility also clearly identifies these vessels as arteriovenous malformation-feeding arteries. Recordings from the normal remote left MCA and PCA show flow velocity and pulsatility within the normal range (upper and lower right). Recording from the left anterior cerebral artery (ACA) also has high flow velocity and low pulsatility (center right); this recording is shown below the zero line to indicate flow toward midline which is the normal direction for ACA flow. Recording from the right ACA shows retrograde flow with high flow velocity and a low pulsatility (center left), revealing its role as a feeding artery collateral to the right MCA. Flow velocities in the distal extracranial internal carotid artery were 85 cm/sec on the right side and 46 cm/sec on the left side (not shown).

In the individual patient, this difference was derived from the real-time spectral display, as well as from the Doppler audio signal (Figs. 1 and 2). The diagnosis of an AVM could thus be obtained from transcranial Doppler investigation in the 26 patients with medium or large AVM's. In some dilated feeding vessels, flow velocity was relatively low; nevertheless, the low pulsatility index value revealed the identity of these vessels.

Nontapering feeding arteries showed flow velocities ranging from 90 to 237 cm/sec (median 136 cm/sec), with pulsatility index values ranging from 0.22 to 0.70 (median 0.44). In tapering feeding arteries, flow velocities ranged from 75 to 124 cm/sec (median 95 cm/sec), with pulsatility index values from 0.41 to 0.74 (median 0.62). The difference between findings in tapering and nontapering feeding vessels was significant (p < 0.003). There was, however, no significant difference between medium-sized and large AVM's with regard to feeding-artery flow velocity or pulsatility index value (p > 0.11). In patients with multiple feeding arteries, the feeder with the highest flow velocity also had the lowest pulsatility index value (Fig. 3). Evaluation of the individual angiograms confirmed that feeders with the highest recorded flow velocities also conveyed a particularly rapid angiographic passage of contrast material through the shunt and into the AVM-draining veins; however, this angiographic feature is difficult to express in exact terms.

When the MCA was the artery feeding a medium or large AVM, the $V_{MCA}/V_{ICA}$ ratio ranged from 1.4:1 to 3.4:1 (median 2.1:1). These data are significantly different from findings in normal remote vessels (p < 0.001).

Two small AVM's were not demonstrable. In arteries feeding these AVM's, flow velocity and pulsatility index values were indistinguishable from findings in normal arteries. Moreover, angiography showed that flow in the feeding arteries investigated was predominantly nutritional flow.

Localization of AVM's

Localization of AVM's was achieved by combining findings from individual basal cerebral arteries, following the basic rule that blood supply to an AVM is through the specific arteries normally supplying this brain region (Fig. 4). In four patients, the anterior segment of the circle of Willis was a collateral vessel.
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Fig. 3. Flow velocity and Doppler pulsatility index from recordings from 22 patients with medium-sized and large unruptured arteriovenous malformations (AVM's). Data from four patients with recent AVM hemorrhage are not included. There is a marked difference between values recorded from normal arteries remote from the AVM (squares), tapering feeding vessels (dots), and nontapering feeding vessels (circles). Lines connect values from each individual patient. In patients with multiple feeding vessels, the vessel having the highest flow velocity also showed the lowest pulsatility index. Collateral feeding vessels are not shown.

from the opposite carotid artery. This was evidenced from findings of retrograde ACA flow on the recipient side, and confirmed angiographically (Figs. 1 and 2). Flow velocity in the distal extracranial ICA was clearly higher on the recipient side in these patients. These findings show that the pressure is lower in AVM feeders even extracranially (see Equation 3).

The AVM was delineated directly in three patients. Recordings from the AVM itself revealed broad flow velocity spectra simultaneously directed toward and away from the probe (Figs. 5 and 6). The corresponding Doppler audio signal had a "machine-hall" quality which was clearly different from the smooth signal recorded from normal arteries and AVM feeders. In some patients, recordings from Sylvian fissure veins were obtained with the probe placed in front of the position used for MCA recordings. The AVM-draining veins had a higher flow velocity and a pulsatile pattern that clearly differed from that of normal cerebral veins (Fig. 6). Veins draining to the sagittal or sigmoid sinus were beyond the reach of current Doppler equipment.

Hyperventilation Test

The 11 patients who underwent a hyperventilation test had resting end-tidal PaCO₂ levels ranging from 37 to 43 mm Hg (median 39 mm Hg). A total of 26 vessels were investigated. Investigation of two different feeding vessels was performed in four patients. Resting mean blood pressure ranged from 78 to 104 mm Hg (median 96 mm Hg). Hyperventilation reduced end-tidal PaCO₂ levels between 8 and 15 mm Hg (median 11 mm Hg). Vascular reactivity was determined as the percentage velocity change during the hyperventilation test, resting flow velocity always representing 100%. This percentage was divided by the difference (in mm Hg) in end-tidal PaCO₂. The MCA of the hemisphere contralateral to the AVM represented normal remote arteries not involved in AVM blood supply.

Figure 7 shows recordings obtained during the hyperventilation test. During hyperventilation, the blood pressure fell between 8 and 12 mm Hg in four patients, remained within 5 mm Hg of resting value in three patients, and rose between 6 and 14 mm Hg in four patients. Normal arteries showed vascular reactivity between 2.3% and 4.0%/mm Hg (median 3.1%/mm Hg; mean ± standard deviation was 3.1% ± 0.5%/mm Hg). The vascular reactivity in these arteries illustrates the vasomotor mechanisms of normal brain.

Flow velocity recordings from the nine nontapering feeding vessels showed vascular reactivity from -0.6% to 1.1%/mm Hg (median 0.0%/mm Hg), the negative sign indicating that despite the drop in end-tidal PaCO₂,
flow velocity in fact increased when blood pressure increased concomitantly (Fig. 8). The response to CO₂ variation seemed to be minimal in these feeding arteries, and was probably induced by the presence of small nutrient branches distal to the recording point. By contrast, recordings from the six tapering feeding arteries clearly indicated the presence of a CO₂-dependent flow component in these arteries. The vascular reactivity determined for these vessels was from 0.9% to 2.4%/mm Hg (median 1.4%/mm Hg). This range probably reflected differences in the ratio of nutritional flow to AVM shunt flow.

**AVM Hemorrhage**

The four patients with recent AVM rupture were admitted between Day 1 and Day 3 after hemorrhage and underwent surgery on Day 9 or later. Flow velocities in normal remote arteries on Day 3 were not significantly different from findings in patients with no bleed (p > 0.02). Statistical evaluation of flow velocities in nontapering feeding vessels showed that patients with recent hemorrhage had significantly lower flow velocities in the feeding arteries on Day 3 after the bleed (p < 0.005). The difference on Day 9 was not significant (p > 0.05). Moreover, there seemed to be a parallel increase in MCA and extracranial ICA flow velocity in two of these patients. These findings are suggestive of a temporarily reduced AVM flow in the acute phase following hemorrhage. Pressure effects operative locally or generally may have contributed to this situation. It is clinically well known that an AVM may be partly and temporarily inactive in the acute or subacute stage of hemorrhage.

Computerized tomography in three of the patients with recent AVM hemorrhage showed intracerebral hematoma of varying size. In the fourth patient, whose CT scans revealed clots in the basal cisterns and ventricles, flow velocities in the MCA remote from the AVM increased from 81 to 158 cm/sec between Days 3 and 9. In the distal extracranial ICA on the same side, flow velocity decreased from 39 to 30 cm/sec, increasing the VMcA:VICA ratio from 2.1:1 to 5.3:1. In this patient, the findings on Day 9 were clearly out of the normal range, probably reflecting moderate vasospasm.

**Clinical Observations**

In one patient, brain swelling developed in the MCA territory proximal to the site of ligation after the excision of a large parietal AVM. Symptoms and signs of a possible hematoma developed within hours. Reoperation revealed a taut brain with only a small clot in the resection cavity. The postoperative hemiparesis and aphasia gradually subsided, and the patient ultimately resumed his former occupation. Control angiography at 3 months confirmed total removal of the AVM. It is noteworthy that this AVM received three long enlarged MCA feeding vessels, each giving off several nutrient branches toward the periphery, and had an MCA flow velocity of 186 cm/sec, which was the highest MCA flow velocity recorded in the present series. Even higher flow velocities, 200 cm/sec or more, were recorded preoperatively from PCA feeding vessels to the AVM's of two other patients; however, these vessels were comparatively short. There was no sign of postoperative brain swelling in these patients.

**Postoperative Findings**

Eleven patients were investigated again at about 3 months postoperatively. Postoperative recordings from normal remote arteries did not differ significantly from those obtained preoperatively. The pulsatility index

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**Figure 4.** Location of 25 large and medium-sized cerebral arteriovenous malformations (AVM's) obtained from computerized tomography scans and angiograms (drawing of the left hemisphere used for graphic simplicity). Symbols denote AVM-feeding arteries; see below for explanation. The feeding arteries with the highest and the next highest flow velocities are indicated in patients with multiple feeding arteries to the AVM. Identification of individual feeding arteries permitted good definition of the anatomical location of individual AVM's. Arrows indicate three AVM's that were delineated directly. One cerebellar AVM fed by the superior cerebellar artery and two small AVM's that were not demonstrable from flow velocity measurements are not included. MCA = middle cerebral artery; PCA = posterior cerebral artery; ACA = anterior cerebral artery; V = velocity.
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FIG. 5. Left carotid angiograms in a 32-year-old woman. Left: Arterial phase showing an arteriovenous malformation in the left Sylvian fissure fed from the middle cerebral artery (MCA) trifurcation branches. Right: Venous phase showing normal distal MCA branches and draining veins with simultaneous opacification, illustrating rapid shunt flow. The arrow indicates the Sylvian fissure vein, and the arrowheads the sphenoparietal and superior petrous sinuses.

FIG. 6. Recordings from the same patient illustrated in Fig. 5. Recording from the left middle cerebral artery (MCA) (upper left), at a sampling depth of 45 mm from the transducer, shows higher flow velocities and lower pulsatility than the recording from the normal right MCA (upper right). Recording at a sampling depth of 30 mm (at the arteriovenous malformation (AVM), center) reveals a broad Doppler spectrum with flow velocity directions toward and away from the probe simultaneously. The corresponding Doppler audio signal had a “machine-hall” quality due to the broad velocity spectrum. Recording from the Sylvian fissure veins (lower) had a “humming” audio-signal quality. Recording from the draining vein on the left side shows a higher flow velocity, about 40 cm/sec (lower left), and is more pulsatile than the signal recorded from the normal vein on the right side (lower right). This illustrates clear hemodynamic differences between AVM’s and normal vascular beds. Flow velocities in the distal extracranial internal carotid arteries were 40 cm/sec on the right side and 46 cm/sec on the left side (not shown).
FIG. 7. Recordings from a 53-year-old man in whom vertebral angiography had revealed a medium-sized arteriovenous malformation (AVM) of the left occipital region filled rapidly via an enlarged nontapering left posterior cerebral artery (PCA). A left carotid angiogram revealed filling of the AVM by a peripheral middle cerebral artery (MCA) branch with a diameter much smaller than the diameter of the proximal MCA (tapering feeder). The right carotid angiogram was normal. Upper: The recordings obtained at rest show typical differences between a nontapering feeder (left), tapering feeder (center), and normal remote arteries (right). Lower: On test hyperventilation, flow velocity level in the left PCA (left) increased from 132 to 134 cm/sec (2%) despite the end-tidal PaCO₂ reduction from 38 to 28 mm Hg. At the same time, mean blood pressure increased from 93 to 100 mm Hg, which probably obscured any effect from CO₂-reactive nutrient vessels distal to the measurement point. Flow velocity in the tapering feeder (left MCA, center) fell from 86 to 72 cm/sec (13% reduction), identifying this tapering feeder as also conveying nutritional flow. Flow velocity in the normal remote right MCA (right) decreased from 50 to 34 cm/sec (32% reduction), showing CO₂ reactivity typical of normal brain flow.

values determined for the arteries that had previously fed AVM’s ranged from 0.80 to 1.04 (median 0.86), not significantly different from the value for normal arteries (p > 0.30). However, flow velocities in these previous feeding arteries (ranging from 40 to 62 cm/sec, median 50 cm/sec) were significantly lower than in normal arteries (p < 0.014). Control angiography confirmed total AVM removal. Some of the previous feeding arteries showed residual enlargement, and this probably explains why flow velocities were lower in these arteries.

**Discussion**

The high flow velocities in AVM-feeding arteries are of the same magnitude as is seen in arteries with vasospasm following subarachnoid hemorrhage (SAH) from ruptured aneurysms. After SAH, volume flow is normal or even below normal limits. Therefore, flow velocity of the ICA in the neck is not increased correspondingly. Thus, the ratio of MCA:ICA flow velocity on the same side is of particular value in distinguishing between high flow velocities signaling cerebral arteries in vasospasm and the increased flow velocity that accompanies high-volume flow in AVM-feeding arteries. This ratio may exceed 10:1 in patients with angiographically verified vasospasm after aneurysm hemorrhage (KF Lindegaard, et al., in preparation). Vasospasm after AVM hemorrhage is considered rare, but was probably present in the patient whose CT scans revealed extensive basal cistern hemorrhage.

**Vascular Reactivity**

Huber and Handa have shown that the caliber of large cerebral arteries is constant within the PCO₂ range from 25 to 57 mm Hg. The reactivity of normal vasculature remote from the AVM (3.1% ± 0.5%/mm Hg) is in keeping with the vascular reactivity of 3.4% ± 0.4%/mm Hg recently demonstrated in healthy volunteers. Furthermore, these findings correspond well
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![Graph](image)

**Fig. 8.** Flow velocity changes recorded during test hyper-ventilation in 11 patients expressed as the percentage velocity change per mm Hg of induced PaCO₂ reduction (vascular reactivity). The middle cerebral artery of the hemisphere contralateral to the arteriovenous malformation (AVM) represented normal arteries remote from the AVM. There is a clearly lower vascular reactivity in feeding arteries than in normal vessels. Moreover, vascular reactivity in nontapering feeding arteries differed as to whether blood pressure increased (cross-hatched bars), decreased (open bars), or remained the same (dotted bars), indicating that flow through the AVM itself does not respond to CO₂ variation. Numbers over each bar denote number of vessels. Two feeding arteries were investigated in four patients.

with the CO₂ reactivity of 4.1% ± 1%/mm Hg determined with the intra-arterial xenon method. Thus, in recordings from a given vessel segment, changes in flow velocity seem to permit a useful estimate of changes in volume flow.

**Hemodynamic Evaluation of AVM’s**

At normal rates of flow and flow velocity, very little of the potential energy that is transmitted by means of pressure is actually spent in the transport of blood from the aorta up to the circle of Willis. As flow velocity in AVM-feeding vessels is generally higher, the pressure drop due to viscous drag in these channels (see Equation 3) is increased. Lower flow velocities and clear evidence of CO₂ reactivity in tapering feeding vessels confirm that there is a relatively higher proportion of nutritional flow, and hence a higher distal resistance and less pressure loss in these than in nontapering feeding arteries.

In patients with multiple feeders, the feeder with the highest flow velocity also had the lowest pulsatile index value (Fig. 3). A low pulsatile index is an important distinguishing characteristic of AVM-feeding arteries (Table 2). However, there is too little quantitative knowledge of the pressure-flow relationship and the capacitative and resistive impedance in AVM’s for a further discussion of the present pulsatility data. The pulsatile signal in arteries feeding and veins draining AVM’s no doubt conceals hemodynamic information of clinical interest.

Chronically low perfusion pressure in areas of normal brain adjacent to an AVM is increasingly recognized as important for the occurrence of the well-known clinical steal syndromes and postoperative brain swelling. This low regional perfusion pressure is partly due to the pressure loss in AVM-feeding vessels. From Equation 3 it also follows that, with a constant vessel diameter (nontapering feeding vessel), the inflow pressure diminishes successively along the vessel’s course. The total length of each feeding vessel and the number and functional importance of nutrient branches taking off from it have been emphasized in other reports. The factor of greatest significance is probably whether these nutrient branches emerge from the feeding vessel trunk proximally, or distally close to the AVM where the arterial pressure is at its lowest.

Because of the arteriovenous shunting there is also an increased regional venous pressure. Measurements of intravascular pressure during craniotomy for cerebral AVM’s confirm that a higher proportion of the feeding-artery pressure is transmitted to draining veins from high-flow AVM’s. When seen in context with angiographic findings, measurements of flow velocity in feeding arteries provide an impression of the volume flow through the AVM (see Equation 2) and, indirectly, of pressure conditions on the venous side.

**Conclusions**

This clinical study has shown that investigations by transcranial Doppler ultrasound and contrast-enhanced CT scanning are useful in combination in the diagnosis of AVM’s. Furthermore, with the length and diameter of the feeding vessel known from angiography, the flow velocity data and the angiographic findings (see Equations 2 and 3) together provide a new level of insight on the “hemodynamic dimension” of AVM’s. We have found this combination useful in obtaining a better understanding of individual hemodynamic states and of value in the surgical management of these patients. In view of the experience from this series, and as a guideline in clinical situations, we presently consider the finding of flow velocities above 150 cm/sec in long AVM-feeding vessels as a warning sign of chronically impaired nutritional perfusion in adjacent normal brain, which moreover may be prone to swelling due to an autoregulation breakthrough when the regional perfusion pressure is restored through elimination of the arteriovenous shunt.

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