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The Effect of Arteriovenous Malformations on the Distribution of Intracerebral Arterial Pressures

Patricia Fogarty-Mack, John Pile-Spellman, Lotfi Hacein-Bey, Andre Osipov, John DeMeritt, Ethan C. Jackson, and William L. Young

PURPOSE: To examine the distribution of arterial hypotension surrounding arteriovenous malformations (AVMs) using a standardized system of vascular zones. **METHODS:** Mean arterial pressures were recorded during superselective cerebral angiography in 96 patients with AVMs (before they underwent liquid polymer embolization) with the use of a system of vascular zones: E = extracranial internal carotid or vertebral artery; I = intracranial internal carotid or basilar artery; T = transcranial Doppler insonation site (A1, P1, M1); H = halfway to feeder, perfusing normal tissue and shunt; and F = feeder at site of *N*-butyl cyanoacrylate injection. Distal arterial pressure was measured contralateral to the AVM in an additional 12 patients (zone H_c). **RESULTS:** Zone pressures (mm Hg ± SD) were E = 76 ± 16, I = 69 ± 15, T = 59 ± 16, H = 47 ± 13, and F = 39 ± 15 mm Hg. Vessel/systemic ratios for the zones were E = 0.97 ± 0.05, I = 0.86 ± 0.08, T = 0.75 ± 0.12, H = 0.61 ± 0.13, and F = 0.50 ± 0.18. Measurements were obtained in 29 patients in all five zones and all had similar mean values. Zone H_c pressure was 66 ± 17 mm Hg and the ratio was 0.78 ± 0.12, both greater than zone H values. **CONCLUSION:** Using a standardized system of anatomic vascular zones, we found a progressive and significant decrease in intracerebral arterial pressure in patients with AVMs that proceeded from the circle of Willis to the nidus. Large areas of parenchyma sharing the same parent arterial supply may be subject to chronic hypotension.

Index terms: Arteries, cerebral; Arteriovenous malformations, cerebral; Brain, pressure

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It has been well documented that arteriovenous malformations (AVMs) induce arterial hypotension in feeding vessels as an effect of the high-flow, low-resistance AVM shunt (1–5). The extent of normal parenchyma that is perfused at chronically hypotensive levels is of considerable interest, because brain regions subject to chronic hypotension may be unable to adapt to a return of normal perfusion pressure after treatment, resulting in hemorrhage or brain swelling. This is the basis of the normal perfusion pressure breakthrough theory (6). The purpose of this study was to examine the distribution of arterial hypotension in brain surrounding AVMs by using a standardized system of anatomic vascular zones.

Materials and Methods

From March 1992 to August 1995, arterial pressure in one or more of five ipsilateral vascular zones (described below) was measured during diagnostic superselective angiography or immediately before initial embolization with a liquid polymer (*N*-butyl cyanoacrylate) in 96 patients with AVMs. In 29 of those patients measurements were obtained in all five ipsilateral vascular zones. In an additional 12 patients, distal arterial pressures in vessels contralateral to the AVM were measured.

Under fluoroscopic guidance, a 7F coaxial catheter was placed in either the cervical internal carotid or the vertebral artery through a 7.5F femoral introducer sheath. An intra-

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cranial microcatheter, 1.5F at its distal tip (Magic, Balt, Montmorency, France), was passed through the coaxial catheter into the intracranial circulation as described previously (4).

Systemic, coaxial, and intracerebral pressures were measured simultaneously with strain gauge pressure transducers (Transpac, Abbott Critical Care, North Chicago, Ill) relative to the right atrium, displayed in real time on a monitor (Merlin, Hewlett-Packard, Waltham, Mass) and digitally recorded (until June 1993) with a MacLab system (AD Instruments, Adelaide, Australia). Mean pressure data were used, as validated by Duckwiler et al (1). In addition, for each patient, the microcatheter pressure was validated against the coaxial catheter pressure while the microcatheter was located in the extracranial carotid or vertebral artery.

The size of the AVM was assessed by a neuroradiologist from the patient's initial magnetic resonance imaging scan, contrast-enhanced computed tomographic studies, or angiogram and classified on the basis of largest diameter (small, less than 2.5 cm; medium, 2.5 to 4.0 cm; and large, more than 4.0 cm).

Patients received sedation with neuroleptic anesthetic (fentanyl citrate, midazolam hydrochloride, droperidol) and low-dose propofol (10 to 25 μg/kg per minute). The anesthetic was titrated to allow the patients to be comfortable but easily roused for neurologic testing.

A standardized system of vascular zones was developed for use in comparison measurements, as shown in Table 1. Zone E corresponds to the extracranial carotid or vertebral artery, and zone I indicates the supraclinoid (intracranial) carotid or basilar arteries. The T designation indicates a measurement obtained at the A1, M1, or P1 segments (7), which are typically insonated during transcranial Doppler measurements (4). The zone H pressure reflects the pressure in a vessel that supplies both AVM and functional brain, whereas zone F reflects pressure in a vessel supplying only the AVM.

To normalize the pressure in the main extracranial vessel, we calculated the ratio of the microcatheter mean pressure to the simultaneously measured systemic (coaxial catheter) mean pressure for each zone (microcatheter mean pressure/coaxial catheter mean pressure). We did not systematically evaluate distal arterial pressure in the hemisphere contralateral to the AVM; however, in 12 patients in whom contralateral vessels were catheterized during the course of angiography, distal intracerebral arterial pressure (denoted as zone H_c) was measured at the A3 to A4, M3 to M4, or P3 to P4 branch level, which corresponds to the zone H measurement in the hemisphere harboring the AVM.

Data, expressed as mean ± SD, were analyzed by using repeated-measures analysis of variance (ANOVA), factorial ANOVA, and linear regression, where appropriate. Statistical significance was set at $P < .05$.

TABLE 1: Definitions of vascular zones

E	Extracranial: systemic pressure at level of coaxial catheter in extracranial vertebral or internal carotid artery
I	Intracranial: supraclinoid internal carotid or basilar artery
T	Transcranial Doppler insonation site: A1, M1, or P1 (long circumferential nomenclature for anterior, middle, and posterior cerebral arteries as proposed by Fischer [7])
H	Halfway: arbitrarily halfway between T and F; supplies normal tissue and shunt
F	Feeder: feeding artery at liquid polymer injection site before deposition
H_c	Contralateral distal arterial pressure at A3 to A4, M3 to M4, or P3 to P4 (homologous position to H but in the normal hemisphere)

Results

Demographics

Pressure measurements in one or more of the five ipsilateral vascular zones (E, I, T, H, F) were recorded in 96 patients (54 men and 42 women) before treatment. Mean patient age was 37 ± 13 years. Thirty-nine patients had hemorrhages, 44 had seizures, 32 had headaches, and 14 had focal neurologic deficits (some patients presented with more than one sign or symptom). The primary locations of the AVMs were as follows: temporal, 28; frontal, 27; parietal, 22; cerebellum, seven; basal ganglia, five; occipital, five; corpus callosum, one; thalamus, one. There was no predominance of left-sided or right-sided lesions. Small AVMs accounted for 6% of lesions, 67% were medium, and 27% were large.

Complete pretreatment measurements in all five ipsilateral zones were obtained for 29 patients. In this subset, the mean age was 35 ± 12

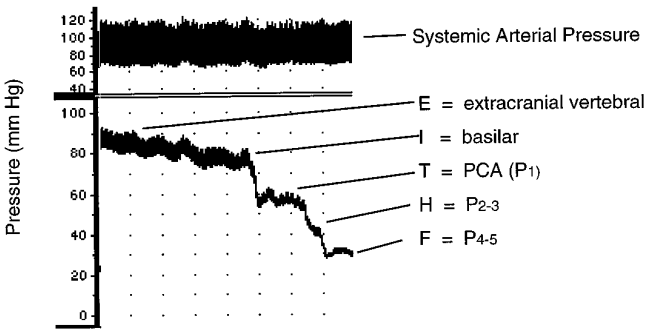

Fig 1. Continuous pressure tracing in a 40-year-old man with seizures and a left-sided, 3.5 × 2.5 × 7.0-cm temporooccipital AVM fed by branches of the middle cerebral and posterior cerebral arteries. This tracing documents the pressure in the vertebral artery (zone E), basilar artery (zone I), P1 segment (zone T), P2 to P3 segment (zone H), and P4 to P5 segment (zone F). Note the gradual decline in pressure, which is accentuated at major branch points, and that all areas distal to P1 (zone T) are relatively hypotensive.

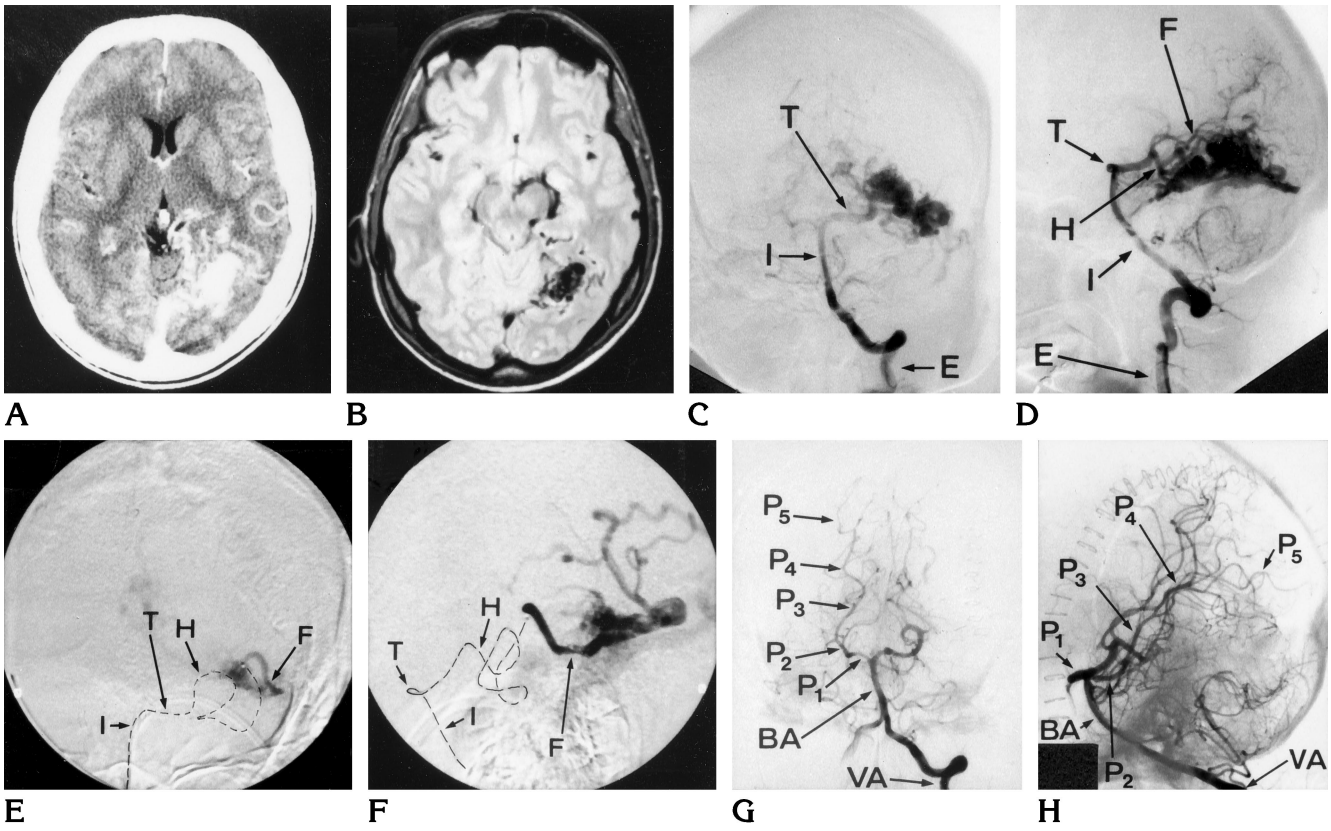

Fig 2. Imaging studies in same patient as in Figure 1 show AVM and are labeled with anatomic vascular zones.

A, Axial contrast-enhanced computed tomogram shows medium-sized left-sided temporooccipital AVM.

B, Axial T2-weighted magnetic resonance image of the lesion.

C, Left vertebral anteroposterior angiogram obtained before treatment, with zones E, I, and T labeled. Zones H and F are not easily distinguished in this view.

D, Left vertebral lateral angiogram obtained before treatment, with zones E, I, T, H, and F labeled.

E and *F*, Superselective anteroposterior and lateral angiograms, respectively, of left posterior cerebral artery show microcatheter in basilar artery and proximal posterior cerebral artery, with contrast material filling distal posterior cerebral artery; zones I, T, H, and F are labeled.

G and *H*, Vertebral anteroposterior and lateral angiograms, respectively, obtained immediately after resection of AVM to clarify the vascular anatomy. As compared with *C* and *D*, note appearance of normal right posterior cerebral artery with indicated Fischer zones P1 to P5 labeled. *BA* indicates basilar artery; *VA*, vertebral artery.

years; 14 patients were women, 15 were men. Nine had hemorrhage, 16 had seizures, eight had headaches, and five had focal neurologic deficits. AVMs were primarily located as follows: frontal, eight; temporal, 12; parietal, six; basal ganglia, two; and occipital, one. Two AVMs were small, 20 were medium, and seven were large.

In the group of 12 patients (eight women and four men; mean age, 42 ± 11 years) in whom distal contralateral vessels were catheterized, five AVMs were parietal, two were frontal, one was thalamic, one was occipital, one was temporal, one was cerebellar, and one was dural. Six patients had hemorrhage: three with seizures, two with headaches, and one with focal neurologic deficit. Of the nondural lesions, one was small, three were medium, and seven were large.

Pressure Measurements in Ipsilateral Vascular Zones

Pressure decreased gradually along the arterial tree. Pressure changes seemed to be exaggerated at major branch points, as shown for a representative patient in Figure 1. Carotid and superselective angiograms of this patient are presented in Figure 2.

The mean arterial pressure measurements in each of the ipsilateral vascular zones (n = 96) and in the vessels in the distal contralateral zone

TABLE 2: Mean arterial pressures in vascular zones E through F (n = 96) and H_c (n = 12)

Zone	No. of Zones Measured	Microcatheter Pressure,* mm Hg ± SD	Microcatheter/ Systemic Pressure, mm Hg ± SD
E	84	76 ± 16	0.97 ± 0.05*
I	78	69 ± 15	0.86 ± 0.08*
T	62	59 ± 16	0.75 ± 0.12*
H	39	47 ± 13	0.61 ± 0.13*
F	82	39 ± 15	0.50 ± 0.17*
H_c	12	66 ± 17	0.78 ± 0.12†

* E, I, T, H, and F zones are different from one another, $P < .05$.

† Zone H_c (distal contralateral vessels) is different from zones H and F, $P < .001$, and from zone E, $P < .05$, but it is not significantly different from zones I and T.

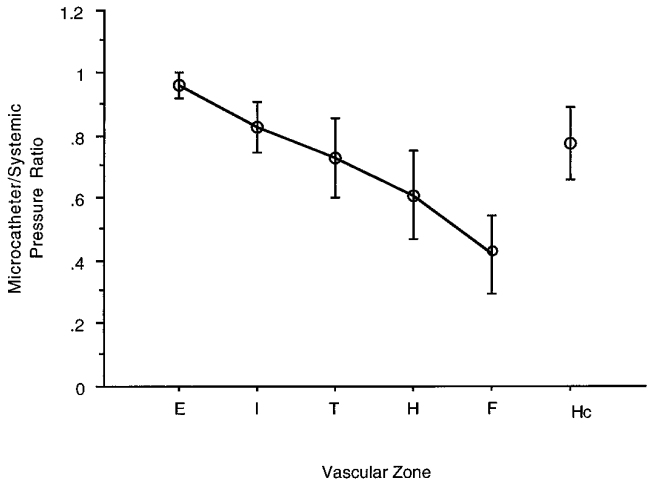

Fig 3. Microcatheter/systemic pressure ratios (mean ± SD) in zones E, I, T, H, and F for 29 patients in whom measurements were obtained in all ipsilateral zones and in 12 patients in whom contralateral zone (H_c) measurements were obtained.

(H_c) (n = 12) are presented in Table 2, as is the ratio of the microcatheter mean pressure to the simultaneously measured systemic (coaxial catheter) mean pressure. The decrease between each zone was statistically significant for both the mean pressure and the pressure ratio; however, the contralateral pressure (zone H_c) was not significantly different from zone T pressure.

Figure 3 depicts the decrease in the mean arterial pressure ratio from zone E to zone F for the patients in whom pressures in all five ipsilateral zones were measured (n = 29). All zones were different from one another with the exception of the mean pressure ratio for the contralateral vessels, which was not significantly different from the zone T pressure.

TABLE 3: Mean pressures of feeding arteries by location (n = 82)

Artery	No. of Arteries Measured	No. of Arteries in Which Zone F Pressure Was Measured	Zone F Mean Pressure, mm Hg ± SD
Middle cerebral	49	41	38 ± 17
Posterior cerebral	26	24	39 ± 16
Anterior cerebral	11	9	37 ± 12
Anterior choroidal	3	3	49 ± 14
Superior cerebellar	6	4	39 ± 4
Pontine	1	1	58

Note.—There was no significant difference among arteries.

Feeding Vessel, AVM Size, Presence of Hemorrhage, and Feeding Artery Pressure

Feeding artery (zone F) pressures were obtained in only 82 patients, and factorial ANOVA revealed no significant effect of the parent conductance artery (anterior cerebral, middle cerebral, posterior cerebral, and so on) on feeding artery pressure. Values for mean arterial pressures are listed in Table 3. Microcatheter/systemic pressure in zone F and AVM size (largest diameter) were not related; however, there was a weak inverse correlation between mean pressure in zone F and AVM size ($y = -2.9x + 50.4$, $r = .22$, $P < .05$). Finally, those patients who had hemorrhage (n = 36) had significantly higher zone F pressure than those without hemorrhage (n = 46): 45 ± 19 mm Hg versus 35 ± 11 mm Hg ($P < .002$, two-tailed t test).

Contralateral Vessel Pressure Measurement

In the 12 patients in whom distal contralateral vessels were catheterized, the mean pressure was 66 ± 17 mm Hg at zone H_c, compared with a mean of 47 ± 13 mm Hg for ipsilateral zone H ($P < .0001$). The zone H_c microcatheter/systemic pressure ratio was 0.78 ± 0.12, compared with 0.61 ± 0.13 for the zone H ratio ($P = .0002$) (Table 2). The angiogram of one of these 12 patients is shown in Figure 4.

Discussion

Progressive Decrease in Cerebral Arterial Pressure

Relative hypotension at the AVM nidus is a well-documented finding. The results of the present study show that this hypotension is progressive, proceeding distally from the circle of

Willis to the pial level along the intracerebral circulation feeding both the AVM and the adjacent functional tissue. Distal contralateral pressures served as surrogate control measurements in this study. Ideally, control pressures would be obtained in normal cerebral vessels in patients without intracranial disease; however, such patients were not available during the course of this study. The finding that the zone H_c pressure was significantly higher than the zone H pressure confirms that the parenchyma perfused at the H pressure is indeed hypotensive compared with the usual arterial pressure at that distance from the circle of Willis.

Several investigators have determined distal cortical arterial pressures (pial or M5 level) to be approximately 90% of systemic pressure in patients without occlusive cerebrovascular disease (3, 8–10). To our knowledge, normal vessels in patients without intracranial disease have not yet been studied with neuroradiologic microcatheter techniques. Handa et al (11), in a study of patients with glioblastoma who were undergoing superselective chemotherapy as a control group for patients with AVMs, found that the ratio of pressures in the M1 to M3 or P1 to P3 branches relative to systemic pressure was 0.75 to 0.85, whereas in patients with AVMs this ratio was 0.38 to 0.46. The findings of the present study are consistent with these results. Although both the zone H_c vessels in patients in the present study and the vessels in the patients with glioblastoma (11) were "normal," they were situated in patients with intracranial disorders, and therefore may not accurately reflect distal cortical pressures in subjects without intracranial disease, but should underestimate rather than overestimate normal pressures.

Vessel Location, AVM Size, and Feeding Pressure

As might be expected, the variance in systemic arterial pressures was less than the variance of pressures measured in the distal feeding arteries. Although there appears to be a tendency for perforator arteries (ie, anterior choroidal and pontine feeding vessels) to have higher mean pressures (Table 3), this difference was not statistically significant owing to the small sample of patients with these feeding arteries. The weak inverse correlation found between feeding artery pressure and size of AVM as measured by greatest diameter as well as the relationship between hemorrhagic presentation and zone F pressure has been described previously by Kader et al (12).

Methodological Considerations

In theory, there is a concern that the microcatheter might impede flow through the vessel. However, since the outer diameter of the 1.5F microcatheter measures 0.5 mm, in a typical feeding vessel with a diameter of 2.0 to 2.5 mm, the microcatheter would occupy only about 5% of the calculated cross-sectional area (πr^2) of the vessel. Wedging of the microcatheter or vasospasm caused by microcatheter manipulation could affect pressure measurements; however, injection of contrast material before pressure measurements were taken revealed no evidence of obstruction to flow, catheter wedging, or vasospasm.

It is not only arterial pressure that determines cerebral perfusion pressure but also cerebral venous pressure or draining vein pressure. If draining vein pressure is elevated, mean arterial pressure measurements will overestimate cerebral perfusion pressure. Although draining vein pressure was not directly measured in this series of patients, superficial draining vein pressure and feeder mean artery pressure (zone F) were positively correlated in a previous series in which they were measured simultaneously (13). Thus, it is unlikely that our measurements substantially or systematically underestimated cerebral perfusion pressure.

The ipsilateral vascular zones designated as pressure measurement points in this study were used to simplify data collection of intracranial arterial pressures. The zones make no attempt to take into account the often complex angioarchitectural characteristics of many AVMs, and the position of zone H is dependent on the distance of the feeder from the circle of Willis; however, the simplicity of the system and the fact that zone H, by definition, perfuses normal parenchyma allows identification of presumably functional parenchymal areas subject to chronic hypotension.

Adaptation to Chronic Hypotension

Several studies have estimated the lower limit of cerebral pressure autoregulation to be between 50 and 60 mm Hg (14–16), even up to 75 mm Hg (17) systemic mean pressure. On the

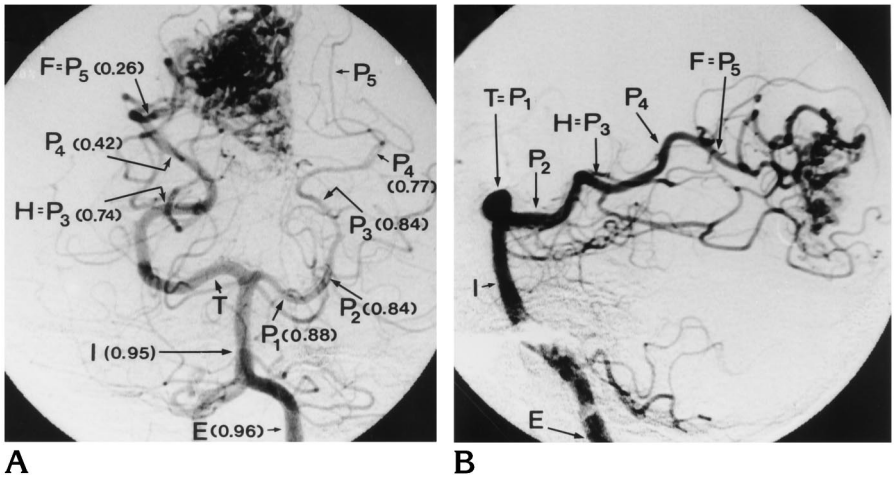

Fig 4. *A*, Anteroposterior angiogram of patient with AVM in right occipital P5 zone. For the right posterior cerebral artery, zones E, I, T, H, and F are labeled, as is the corresponding Fischer nomenclature, and the microcatheter/systemic pressure ratios, when obtained, are indicated. The left posterior cerebral artery is a normal vessel and is labeled according to the Fischer nomenclature, with microcatheter/systemic pressure ratios where obtained. The discrepancy in arterial pressures is most evident in the P4 segments: on the side of the AVM the ratio is 0.42, whereas in the normal hemisphere the P4 ratio is 0.77. The pressure ratio in the feeding artery (F = P5), is 0.26. P5 pressure was not measured in the left posterior cerebral artery.

B, Lateral angiogram in the same patient. Owing to vessel overlap in this view, only the right posterior cerebral artery is labeled, with zones E, I, T, H, F, and Fischer nomenclature.

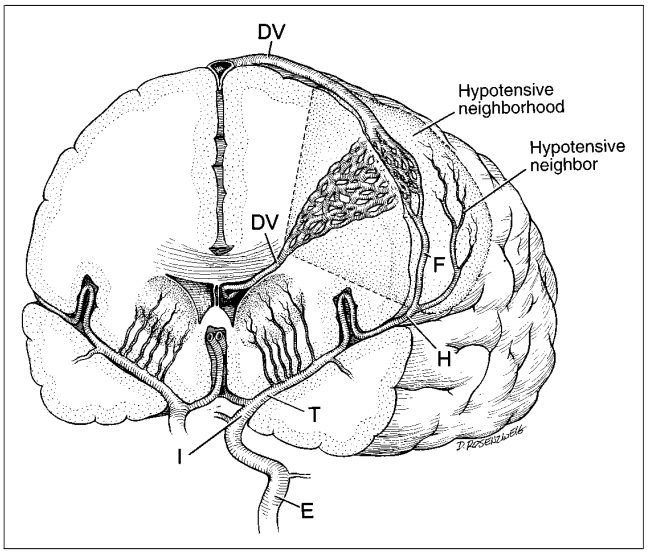

Fig 5. Coronal oblique depiction of intracranial circulation to AVM shows anatomic vascular zones (see Table 1) and surrounding functional area subject to chronic hypotension (*hypotensive neighborhood*). One vessel perfusing the hypotensive neighborhood, labeled the *hypotensive neighbor*, is illustrated. *E* indicates extracranial; *I*, intracranial; *T*, transcranial Doppler insonation site; *H*, pressure measured at half the distance from zone T to F, supplying functional tissue and shunt; zone *F*, feeder; and *DV*, draining veins. There is also a hypotensive neighborhood, perfused by hypotensive neighbors, in the volume of brain that has been cut away for illustrative purposes.

basis of these studies and the present findings we speculate that there are large areas of functional parenchyma adjacent to AVMs that are chronically perfused at relative hypotension. Such an area is depicted in Figure 5 and is termed the *hypotensive neighborhood*. It is perfused by a vessel labeled the *hypotensive neighbor*.

Previous work has shown that the areas adjacent to the AVM, which correspond to the hypotensive neighborhood, maintain their ability to autoregulate; however, the lower limit of autoregulation is shifted to the left (18). The means by which the hypotensive neighborhoods accomplish this shift have not yet been determined. It may be that the adaptive mechanisms have distant effects on the cerebral vasculature, which would account for the global (or hemispheric) rather than local nature of the normal perfusion pressure breakthrough phenomenon both in patients with AVMs (19) and in those undergoing carotid endarterectomy (20).

Systematic study is required to confirm that the hypotensive neighborhood in fact subserves the functions traditionally thought to be located there. In one reported case, intact language function was documented in an area supplied at a mean pressure of 25 mm Hg (systemic mean, 55 mm Hg) (21). Once intact function has been confirmed, studies to elucidate the mechanisms by which brain tissue adapts to chronic hypotension are warranted. Understanding such mechanisms may be important in the treatment of ischemic cerebral syndromes.

Conclusions

First, there was a progressive reduction in cerebral arterial pressure distally from the circle of Willis along vessels feeding both the AVM and

adjacent functional tissue. Second, this reduction in arterial pressure was not seen in vessels contralateral to the AVM. Finally, the mechanisms by which the hypotensive neighborhood adapts to and functions appropriately in an environment of chronic hypotension are presently unknown and warrant further investigation.

Acknowledgments

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