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Blood Pressure Changes in Feeders to Cerebral Arteriovenous Malformations During Therapeutic Embolization

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We recorded blood pressures in feeding arteries of cerebral arteriovenous malformations with microcatheters before, during, and after embolization in awake patients. Our embolization results corroborate reports of intraoperative measurements that show an immediate increase in blood pressure after ligation. Demonstration of abrupt hemodynamic changes gives additional credence to the theory of normal perfusion pressure breakthrough and to theories of neurologic change based on vascular steal phenomena. Furthermore, pressure changes occurred earlier than visible slowing of flow during the embolization, suggesting that this is a more sensitive guide with which to monitor the progress of the embolization than is serial angiography.

A devastating complication of treatment of arteriovenous malformations (AVMs), whether by endovascular techniques or with conventional neurosurgery, is the normal perfusion pressure breakthrough syndrome [1–4].

Other investigators have measured intraoperatively an elevation of the pressure in feeding arteries after surgical ligation, and "steals" from adjacent normal parenchyma have been discussed [5–8]. However, a recent report appeared to contradict some findings regarding changes in perfusion of surrounding parenchyma [9].

In an effort to better understand the hemodynamics of brain AVMs and the effects of endovascular therapy we recorded pressures in arteries feeding AVMs before, during, and after therapeutic embolization.

Materials and Methods

We studied six vessels in five patients with cerebral AVMs who were referred to us for embolization. All patients were awake in the angiography suite, and IV sedation was used as necessary. Pressures were recorded through a Tracker-18 microcatheter* coupled to a transducer and digital pressure monitor.[†] The microcatheter was positioned intracranially with a coaxial assembly system that consisted of an introducer sheath in the femoral artery through which a standard angiographic catheter was passed. The microcatheter was then advanced through the standard catheter to its final intracranial position. Each vessel was measured before and after embolization. Case 2 was also measured after we infused some emboli, because we were uncertain whether the first visible flow changes were occurring. Case 5 was measured when the first visible changes occurred in addition to the measurements taken before and after embolization. A systemic (brachial) blood pressure was obtained with a Dinamap Automatic Sphygmomanometer[‡] during each microcatheter measurement. Pressure tracings from the microcatheter were also recorded during measurements. The embolization was performed through the same microcatheter once satisfactory positioning was accomplished.

Results

Table 1 shows that in each case systolic, diastolic, and mean arterial pressure of the feeder rose during embolization to a final higher pressure despite a relatively

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TABLE 1: Patient Data

Patient No.	Age	Sex	Nidus Location	Nidus Size	Embolized Vessel	Feeder Blood Pressure (S/D M)*	Feeder Pulse Pressure	Cuff Blood Pressure (S/D M)*	Embolic Material
1	33	F	Right parietal	3 cm	Parietal branch of MCA	55/45, 50 Preembo.	10	126/71, 88	PVA
						85/74, 80 Postembo.	11	130/77, 100	
2	30	F	Right parietal	3 cm	Angular branch of MCA	79/68, 72 Preembo.	11	121/84, 100	PVA
						90/83, 88 Flow slightly diminished?		120/70, 83	PVA
						106/97, 102 Postembo.	9	126/68, 88	
3	56	M	Right parietal	4 cm	Parietal branch of MCA	58/43, 50 Preembo.	15	132/87, 95	4-0 silk
						90/66, 76 Postembo.	24	121/79, 89	
4	31	M	Posterior frontal	5 cm	Branch of pericallosal	55/41, 48 Preembo.	14	130/81, 102	PVA
						81/56, 69 Postembo.	25	116/74, 86	
					Branch of MCA	60/45, 53 Preembo.	15	110/73, 88	PVA
						91/64, 75 Postembo.	27	116/73, 89	
5	67	M	Posterior fronto-parietal	6 cm	ACA	73/54, 64 Preembo.	19	126/74, 104	PVA and 4-0 silk
						95/67, 81 Flow slowing		134/83, 111	
						104/79, 91 Postembo.	25	130/78, 105	

* S = systolic (mm Hg), D = diastolic, M = mean.

constant systemic pressure. In the feeding vessel the average rise in systolic pressure was 29.5 mm Hg, the average rise in diastolic pressure was 23.3 mm Hg, and the average rise in mean arterial pressure was 27.3 mm Hg. Pressure tracings showed good wave forms before and after embolization when distal occlusion of the feeder was achieved (Fig. 1).

Increased pulse pressure (amplitude) as well as absolute pressure increases developed as the embolization proceeded. The average preembolization pulse pressure (amplitude) was 14 mm Hg. The average postembolization pulse pressure was 20.2 mm Hg, resulting in an average change in pulse pressure of +6.2 mm Hg (range, −2 to +12). Five vessels showed an increase in pulse pressure while one vessel (patient 2) showed no increase in pulse pressure. In fact, in this case there was a slight decrease (−2 mm Hg). Nevertheless, the postembolization feeder pressure did rise substantially and was the only case in which the mean feeder pressure became greater than the mean cuff pressure.

Discussion

The hemodynamics of AVMs and surrounding brain parenchyma have been debated for years [5, 6, 7, 9]. Intuitively, one would suppose that a low-resistance pathway would have high flow. Mean arterial pressure would be expected to be lower than other similar normal vessels, and "steals" from surrounding tissue are plausible [8]. If surrounding ischemic tissue were suddenly to be perfused at higher pressures one might predict the onset of a normal perfusion pressure breakthrough [1–4].

The fundamental property of AVMs that supports the above concepts is that there is a low-resistance pathway with a low pressure and high flow that is rapidly normalized by embolization. We have documented these pressure changes with intraarterial measurements before, during, and after emboli-

zation. Our data are in agreement with some previous reports of intraoperative measurements [5–7], but our recordings were made in awake patients in whom blood pressures had not been pharmacologically altered.

The changes in pressure are inversely related to flow. As the feeder pressure increased, the flow through it decreased. We have been impressed that pressure changes can be detected earlier than the visually apparent slowing of flow seen fluoroscopically. This information aided us in one patient (case 2) in whom we continued to inject small particles, although we were initially unable to detect flow changes fluoroscopically. The feeder pressure, however, began to increase very early and ultimately the flow began to decrease. Statis was finally achieved. If the pressures had not been increasing we would have altered our procedure, probably by resorting to larger-sized particles and risking a too proximal occlusion.

In this same vessel (case 2) we observed a slight decrease in pulse pressure although the absolute pressure did rise with embolization. We are uncertain why the change in pulse pressure in this case was different from the others. Possibly there are variations in vessel elasticity that are pressure dependent.

Inaccuracies in our recordings could have been from several sources. For example, we did not obtain blood gases during these procedures. Carbon dioxide is a strong determinant of cerebrovascular perfusion; yet, we have assumed that in these stable, awake, alert patients it remained relatively constant and was therefore not a factor. Another source of error could be from vasospasm induced by the procedure. Although we did not observe this, a visual evaluation may not reflect minor variations. Also, wedging the microcatheter in a vessel or against a wall can cause low readings. We specifically tried to avoid this situation, since we were relying on flow for particle delivery and frequently checked the flow under fluo-

Fig. 1.—Posterior parietal arteriovenous malformation (AVM) (case 3).

A, Lateral view of right internal carotid artery angiogram (*arrow* = position finally attained with microcatheter).

B, Lateral view with microcatheter in major feeder (*arrow* = tip of microcatheter). This is the position used for pressure measurements and embolization.

C, Lateral view of internal carotid angiogram postembolization. Only a small amount of filling of the AVM remains.

D, Pressure tracing. *Solid line* = preembolization, *dotted line* = postembolization. Note that good wave form remains but is increased in absolute value.
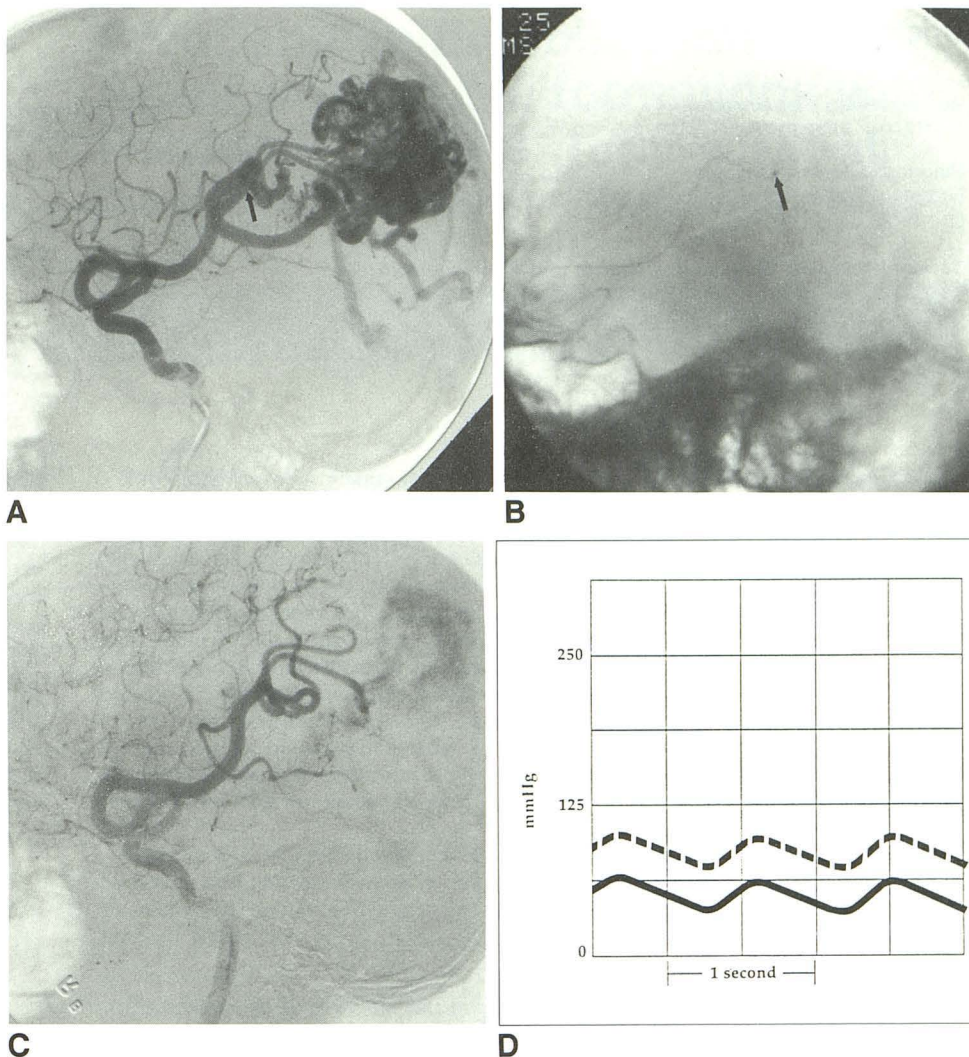
roscopy. Finally, catheter occlusion by emboli could affect subsequent measurements but would tend to decrease the measured values. Yet in all cases increases occurred, making this an unlikely source of error.

Not all patients suffer "steals" or normal perfusion pressure breakthrough, but documenting pressure changes is important if we are to hypothesize and alleviate such effects. Much work still remains to elucidate the other factors (such as size of AVM, speed of flow, etc.) that combine to determine the course of these lesions.

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REFERENCES

1. Spetzler RF, Wilson CB, Weinstein P, Mehdorn M, Townsend J, Telles D. Normal perfusion pressure breakthrough theory. *Clin Neurosurg* **1978**; 25:651–672
2. Solomon RA, Michelsen WJ. Defective cerebrovascular autoregulation in regions proximal to arteriovenous malformations of the brain: a case report and topic review. *Neurosurgery* **1984**;14:78–82
3. Batjer HH, Devous MD, Meyer YJ, Purdy PD, Samson DS. Cerebrovascular hemodynamics in arteriovenous malformation complicated by normal perfusion pressure breakthrough. *Neurosurgery* **1988**;22:503–509
4. Halbach VV, Higashida RT, Hieshima GB, Norman D. Normal perfusion pressure breakthrough occurring during treatment of carotid and vertebral fistulas. *AJNR* **1987**;8:751–756
5. Nornes H, Grip A. Hemodynamic aspects of cerebral arteriovenous malformations. *J Neurosurg* **1980**;53:456–464
6. Barnett GH, Little JR, Ebrahim ZY, Jones SC, Friel HT. Cerebral circulation during arteriovenous malformation operation. *Neurosurgery* **1987**;20: 836–841
7. Spetzler RF, Martin NA, Carter LP, Flom RA, Raudzens PA, Wilkinson E. Surgical management of large AVM's by staged embolization and operative excision. *J Neurosurg* **1987**;67:17–28
8. Homan RW, Devous MD, Stokely EM, Bonte FJ. Quantification of intracerebral steal in patients with arteriovenous malformation. *Arch Neurol* **1986**;43:779–785
9. Hassler W, Steinmetz H. Cerebral hemodynamics in angioma patients: an intraoperative study. *J Neurosurg* **1987**;67:822–831