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Pressure Changes in the Arterial Feeder to a Cerebral AVM as a Guide to Monitoring Therapeutic Embolization

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Blood pressure changes, or lack of them, in the feeding pedicle to a cerebral arteriovenous malformation appear to be a more sensitive guide by which to monitor the progress of a therapeutic embolization than are fluoroscopic or serial angiographic changes. We have measured the feeder pressure in three patients before embolization with particulate materials and compared the pressure with subsequent measurements obtained during the progress of the procedure. When the feeder pressure has increased after particle infusion, even if fluoroscopic slowing of flow is not apparent, additional particulate infusion has soon resulted in slowing of flow and, finally, hemostasis. When the feeder pressure has not increased, we have changed to a larger-sized particle and then continued the procedure. Monitoring changes in feeder pressure is particularly helpful during the early stages of embolization; however, our end point is still the angiographic demonstration of flow arrest.

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Treatment of cerebral arteriovenous malformations (AVMs) with endovascular techniques (therapeutic embolization) may be facilitated with serial measurements of the pressure in the pedicle being treated. Several authors have documented blood pressure elevations in a feeding artery to an AVM as the distal run-off is decreased by either standard neurosurgical (clipping or ligating vessels) [1–3] or endovascular (embolization) [4] techniques. We treated three patients with brain AVMs in whom we used the change in pressure in the feeding vessel as an indication to either select a different material or to continue the procedure with the same material.

Materials and Methods

Three patients undergoing embolization for treatment of cerebral AVMs were studied. The procedures were performed via the femoral artery. A standard cerebral catheter was placed into the brachiocephalic vessel of interest and a Tracker-18 microcatheter* was then coaxially introduced into the intracranial circulation. Intracranial pressure measurements of the feeding arteriole were accomplished by connecting the microcatheter to a pressure transducer and digital pressure monitor.† An initial preembolization measurement was obtained in all cases. A brachial sphygmomanometer measurement‡ was obtained at each microcatheter measurement as a baseline control. Polyvinyl alcohol (PVA) particles§ were used in all three cases and were suspended in a solution of iopamidol (300 mg I/ml) diluted to half strength with saline for injection. PVA was used in conjunction with 4-0 silk suture in one patient (case 2). Local anesthesia and mild IV sedation were used as necessary, but all patients were awake and alert in the angiography suite.

Case 1

A 55-year-old man presented with a subarachnoid hemorrhage and evaluation demonstrated a large parietal AVM. The patient was referred to us for embolization. The arterial

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§Pacific Medical Industries, San Diego, CA 92122.

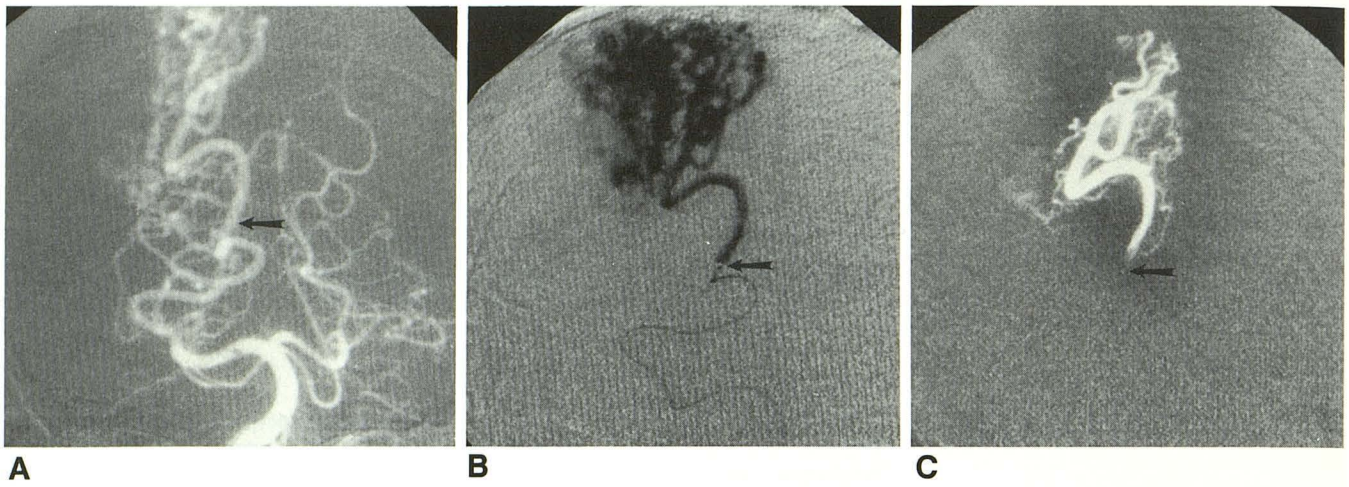

Fig. 1.—Case 1. Parietal AVM.
***A*, Towne view of vertebral angiogram shows enlarged right posterior cerebral artery (*arrow*) feeding AVM.**
***B*, Microcatheter in feeder (*arrow* = tip of catheter in posterior cerebral artery) prior to embolization. This is the position used for pressure measurements and embolization.**
***C*, Postembolization angiogram in same position as 1B.**

feeders from the middle cerebral artery had been occluded during a first-stage procedure 6 weeks previously, and the patient was now returning for a second-stage embolization. This time, embolization of the posterior cerebral artery contribution was planned (Fig. 1A), and, therefore, the microcatheter was placed into the right posterior cerebral artery (Fig. 1B).

The initial preembolization values in the feeder were 52/41 mm Hg (mean = 46). After infusion of one bottle of 300–500 μm of PVA, the pressure was 54/44 mm Hg (mean = 47). Our conclusion at this time was that the particles were too small and were flowing through the malformation to the lungs. Therefore, slightly larger PVA particles were selected (500–700 μm). Infusion of the new suspension did not reveal an obvious fluoroscopic flow change, but after approximately a quarter of the bottle, the pressure rose to 80/62 mm Hg (mean = 72). We therefore continued to infuse this mixture. Visible slowing occurred after approximately two thirds of the bottle had been infused, and hemostasis was finally obtained after the full bottle had been infused (Fig. 1C). Pressure measurement at the conclusion was 105/77 mm Hg (mean = 90). The brachial pressures remained essentially unchanged throughout the procedure at approximately 145/90 mm Hg (mean = 116). The patient tolerated the procedure well and is currently being considered for surgical resection.

Case 2

F. B. is a 38-year-old man whose AVM was diagnosed and partially treated with IBCA 11 years ago. He did well until the past year, at which time seizures, increasing spasticity, and suicidal depression became manifest.

Repeat angiography showed a large AVM supplied principally by branches of the right anterior cerebral artery (Fig. 2A). A microcatheter was positioned into the anterior cerebral artery (Fig. 2B). The initial pressure in this pedicle was 45/37 mm Hg (mean = 41). The systemic pressure at this time was 100/48 mm Hg. Infusion of one bottle of PVA (300–500 μm) showed no pressure changes, and no fluoroscopic changes were evident. A second bottle of PVA (500–700 μm) was infused, again without either pressure or fluoroscopic change. The pedicle pressure at this time remained 45/38 mm Hg (mean = 41), with a systemic pressure of 108/71 mm Hg. Therefore, we injected three short lengths (about 5-cm each) of 4-0 silk suture and noted a modest slowing of flow fluoroscopically. Further embolization with PVA (300–700 μm) steadily slowed the flow and rapidly caused complete occlusion of that pedicle to the AVM (Fig. 2C).

Case 3

A 23-year-old man sustained his first seizure, and evaluation disclosed a left temporal AVM supplied by branches of the middle cerebral, posterior cerebral, and anterior choroidal arteries (Fig. 3A). In a first-stage embolization, the middle cerebral supply was occluded. Pressure monitoring was not available during this stage owing to an equipment malfunction. A second-stage procedure 2 days later consisted of superselective catheterization of the anterior choroidal artery (Fig. 3B). The initial pressure in this vessel was 85/62 mm Hg (mean = 72), with the systemic cuff pressure at 122/61 mm Hg (mean = 85). Infusion of a suspension of PVA (300–500 μm) did not appear to change the fluoroscopic appearance after half the bottle had been infused. Yet a feeder pressure at this time was 98/76 mm Hg (mean = 89) without any significant change in systemic pressure (124/66 mm Hg, mean = 86). Therefore, we continued the infusion. When another half bottle (total of one bottle) had been infused, still no fluoroscopic changes were apparent, but the feeder pressure continued to increase (101/80 mm Hg, mean = 91). Infusion of only a small amount more emboli finally resulted in visually apparent slowing of flow. When a total of one and one-half bottles had been infused, hemostasis was accomplished (Fig. 3C). Feeder pressure at the conclusion was 107/78 mm Hg (mean = 93), with a systemic pressure of 125/71 mm Hg (mean = 88).

Discussion

Several authors [1–3] have demonstrated increases in pressure in arterial feeders to AVMs during treatment with conventional neurosurgical techniques. In a previous report [4] we documented similar changes during embolization. As the distal run-off in a vascular malformation decreases, the blood pressure in feeding pedicles increases. This is a report of three procedures during which the pressure changes or lack thereof were useful as a guide with which to choose emboli of the appropriate size.

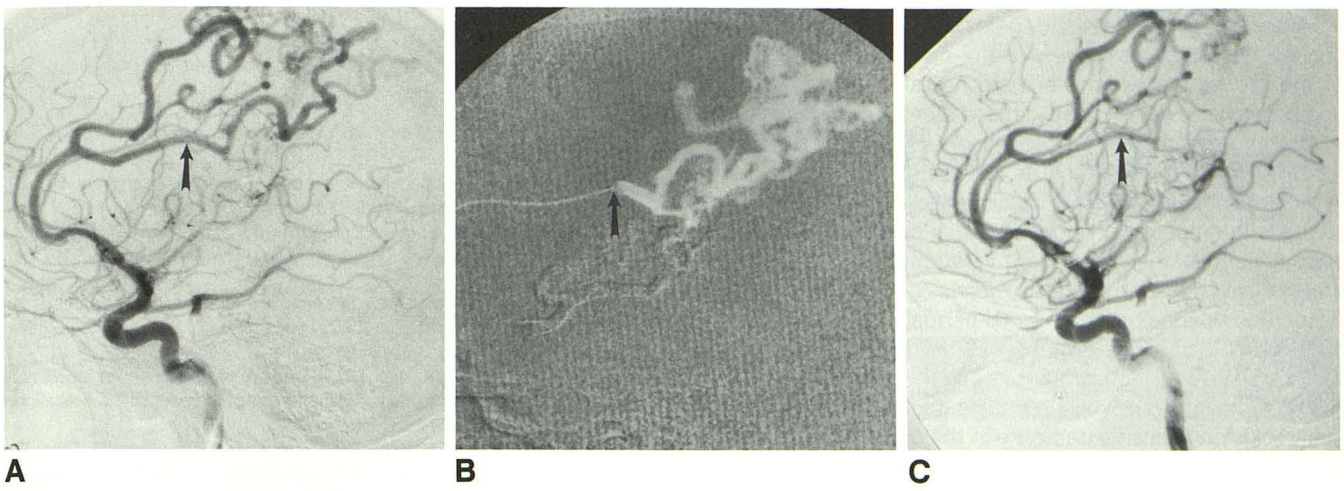

Fig. 2.—Case 2. Parietal AVM.
***A*, Early lateral view of internal carotid angiogram shows several enlarged anterior cerebral artery feeders. *Arrow* marks position eventually obtained with microcatheter.**
***B*, Microcatheter in position for pressure measurements and embolization (*arrow* = microcatheter tip).**
***C*, Postembolization carotid angiogram. Microcatheter has been removed, but *arrow* marks its previous position.**

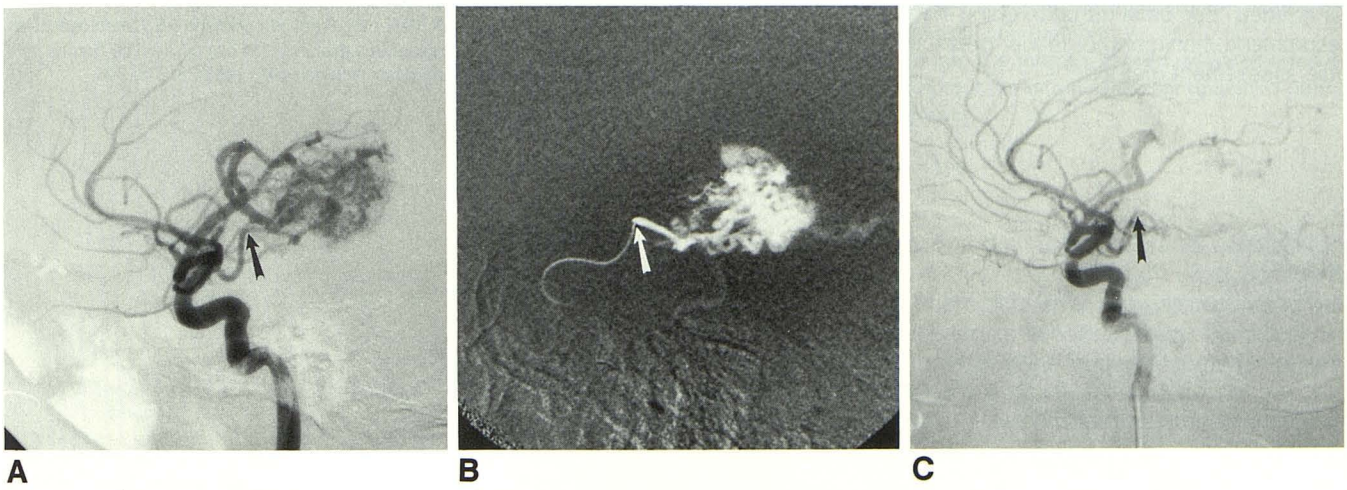

Fig. 3.—Case 3. Temporal AVM.
***A*, Lateral view of carotid angiogram demonstrating temporal AVM. *Arrow* marks position eventually obtained with microcatheter.**
***B*, Microcatheter in anterior choroidal artery (*arrow* = tip). This was the position used for pressure measurements and embolization.**
***C*, Postembolization carotid angiogram. Microcatheter has been removed but *arrow* marks its previous position. Decreased flow to AVM is apparent.**

The increase in pressure within the feeding artery is a more sensitive indicator of hemodynamic change than are visual differences seen fluoroscopically or with serial angiography. In the first two cases we increased particle size when pressures remained unchanged. Conversely, in the third case pressures increased despite an unchanging fluoroscopic appearance, and infusion of additional emboli of the same size soon led to hemostasis.

The goal of endovascular treatment of AVMs is to occlude the arteriovenous shunt in the malformation. If occlusion of feeders is too proximal, collaterals are formed that reperfuse the low-resistance pathway. Therefore, one attempts to occlude as far distally as possible in the malformation without going out the other (venous) side. To this end it is advantageous to begin particulate embolization with smaller rather than larger particles. Inherent in this reasoning is the potential for particles that are smaller than the shunting vessels to traverse the malformation and end up in the capillary bed of the lungs. When feeder pressure does not increase after particle infusion, pulmonary embolization has presumably occurred. Early recognition of this fact could limit this potential complication, and monitoring the feeder pressure seems helpful in this regard. Comforting is the empirical impression that patients appear to tolerate well the pulmonary emboli that must certainly be occurring in many embolizations. Most likely this tolerance derives from the size of the pulmonary capillary bed that is tremendous compared with the amount of errant emboli.

Measuring pressures and detecting an increase or no change has been particularly useful during the early stages of the procedure, when there are no other guidelines. Our end point, hemostasis, is still guided by visual evidence.

Also important is the further evidence that the pressure in feeders to AVMs increases as the distal runoff decreases. While previous reports have documented such hemodynamic changes intraoperatively our data have been obtained in the awake, nonanesthetized patient in whom pressures are not artificially altered. This supports theories of neurologic change based on hyperfusion or a normal perfusion pressure breakthrough in an adjacent territory [5–9].

Inaccuracies in measurements might occur if emboli occlude or partially occlude the microcatheter. However, catheter occlusions cause a decrease in the transmitted pressure and are therefore recognizable. A strong blood flow modifier is arterial pCO_2. We have not monitored the pCO_2 but have assumed it to be relatively stable in the awake, alert patient undergoing embolization.

Sometimes it is difficult to evaluate the progress of a therapeutic embolization. Currently, in cases that require a superselective catheterization, we obtain a pressure measurement prior to embolization and subsequent measurements as needed. For patients undergoing AVM embolization, we recommend monitoring of feeder pressures as a simple, reliable assessment method.

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