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# Particulate Embolization of the Anterior Choroidal Artery in the Treatment of Cerebral Arteriovenous Malformations

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The anterior choroidal artery is commonly recruited to supply arteriovenous malformations in the temporal lobe, basal ganglia, lateral ventricle, and internal capsule. Embolization of this artery is theoretically hazardous owing to its supply to important motor territory, relative lack of collaterals, and small caliber. However, this procedure can be a beneficial adjunct to surgery or radiation in the treatment of arteriovenous malformations.

We describe our experience in performing particulate embolization of the anterior choroidal artery in 15 patients (16 procedures). Significant flow reduction was achieved in 14 patients. Two symptomatic and two asymptomatic complications arose, including permanent hemiparesis in one patient.

The anterior choroidal artery (AChA) supplies the internal capsule, basal ganglia, medial temporal lobe, optic pathways, cerebral peduncle, and choroid plexus of the lateral ventricle [1]. This artery may be recruited to supply arteriovenous malformations (AVMs) that involve these structures. Embolization of the AChA can be a beneficial adjunct to surgery or radiation in the treatment of AVMs, but this procedure is theoretically hazardous because of the artery's supply to crucial structures, its relative lack of collaterals, small caliber, and abruptly angled origin at the internal carotid artery (ICA). We report our experience in performing particulate embolization of the AChA in 15 patients with cerebral AVMs.

## Materials and Methods

Fifteen patients with cerebral AVMs supplied in part by the AChA had embolization procedures involving this artery (Table 1). Patients consisted of eight females and seven males, 12–56 years old. Predominant presenting symptoms included hemorrhage (eight patients), seizures (six patients), and headaches (one patient). Of the eight patients with hemorrhage, four had intracerebral hemorrhage (ICH), three had intraventricular hemorrhage (IVH), and one had subarachnoid hemorrhage (SAH). Four patients exhibited angiographic evidence of a pseudoaneurysm in the nidus of the AVM; all four presented with hemorrhage. Indications for AChA embolization included (1) the presence of a pseudoaneurysm in the AVM nidus, which is considered a highly likely site of hemorrhage; (2) planned surgical resection of the AVM facilitated by preoperative diminution of the AChA supply, which can be difficult to control surgically; and (3) diminution of the dominant AChA supply to an AVM prior to radiosurgery.

Embolic agents included polyvinyl alcohol (PVA) particles measuring 300–1000 μm (Interventional Therapeutics Corp., So. San Francisco, CA; and Pacific Medical Industries, La Mesa, CA) (15 cases), and PVA particles and 5-mm platinum coils (Cook, Inc., Bloomington, IN) (one case). The procedure was aborted prior to embolization in one patient. Embolic agents were delivered through a 2.2-French Tracker catheter with an 18-cm distal segment (Target Therapeutics, San Jose, CA), navigated to the AChA through a 5.5- or 7-French guiding catheter placed in the internal carotid artery under systemic anticoagulation. The

**TABLE 1: Summary of Patients with AVM Treated by Anterior Choroidal Artery (AChA) Embolization**

| Patient No. | Age (yrs) | Sex | Side | Location | Spetzler Classification[a] | History | Pseudoaneurysm Present | Embolic Agent | Adjunctive Therapy | Complications Resulting from AChA Embolization |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 40 | M | R | Posterior medial T lobe | 2 | ICH | No | PVA | Surgery | No |
| 2 | 21 | F | L | Thal, lentiform nucleus, IC, LV | 4 | ICH | Yes | PVA | XRT | No |
| 3 | 37 | F | R | Posterior medial T lobe, LV | 2 | IVH | No | PVA | Surgery (XRT) | No |
| 4 | 55 | M | L | Medial T lobe | 3 | SZ | No | None | None | Asymptomatic perforation |
| 5 | 33 | F | L | Posterior medial T lobe | 2 | SZ | No | PVA | Surgery | Asymptomatic perforation |
| 6 | 33 | M | L | Thal, 3V | 3 | SAH | No | PVA | Surgery | No |
| 7 | 39 | F | L | Posterior medial T lobe | 3 | ICH | No | PVA | Surgery | No |
| 8 | 43 | F | R | Medial T lobe | 3 | SZ | No | PVA | Surgery | No |
| 9[b] | 17 | M | R | Thal, LV | 4 | IVH | Yes | PVA | XRT | No |
| 10 | 36 | M | R | Anterior medial T lobe | 3 | SZ | No | PVA | Surgery | No |
| 11 | 23 | M | L | Medial T lobe, lentiform nucleus | 5 | ICH | Yes | PVA and platinum coils | XRT | No |
| 12 | 56 | F | R | Medial T-P lobes, peduncle | 4 | HA | No | PVA | Surgery | Transient hand weakness |
| 13 | 12 | F | L | Medial T lobe | 2 | IVH | Yes | PVA | XRT | Hemiparesis |
| 14 | 52 | M | R | Anterior medial T lobe | 2 | SZ | No | PVA | Surgery | No |
| 15 | 31 | F | R | T-O lobes, thal, CC | 4 | SZ | No | PVA | XRT | No |

Note.—T = temporal, O = occipital, P = parietal, thal = thalamus, IC = internal capsule, LV = lateral ventricle, 3V = third ventricle, CC = corpus callosum, ICH = intracerebral hemorrhage, PVA = polyvinyl alcohol particles, XRT = radiation therapy, IVH = intraventricular hemorrhage, SZ = seizures, SAH = subarachnoid hemorrhage, HA = headaches.
[a] See [2].
[b] This patient was embolized twice via the AChA.

Tracker catheter tip was positioned at or distal to the plexal point of the AChA (the point at which this artery enters the choroidal fissure) directly supplying the AVM. Functional testing with sodium amytal was not done in any case. Embolization was halted when a pseudoaneurysm was eradicated or when AChA flow was diminished such that further embolization might result in retrograde AChA or critical branch thrombosis. Nine patients were embolized in preparation for surgical resection and six patients in preparation for radiation therapy, which was not performed in one patient because the AVM was considered too large.

### Representative Case Reports

*Case 11*

A 23-year-old man without previous symptoms suffered massive intraparenchymal hemorrhage involving the left internal capsule and lentiform nucleus, with intraventricular extension (Fig. 1A), resulting in right hemiplegia, aphasia, and somnolence. The mass effect from the hematoma required emergent craniotomy for clot removal. Arteriography several days later showed an AVM involving the left medial temporal lobe and lentiform nucleus supplied predominately by the AChA and lenticulostriate arteries (Fig. 1B). As a pseudoaneurysm in the AVM nidus arising from the AChA was considered a likely site of hemorrhage, AChA embolization was performed through a Tracker catheter positioned at the plexal point (Fig. 1C). Delivery of PVA particles (500–700 μm) and four 5-mm platinum coils resulted in obliteration of the pseudoaneurysm and diminished flow to the AVM (Fig. 1D) without change in neurologic status. Particulate embolization of a posterior thalamoperforating branch was also carried out. As the AVM was not deemed surgically resectable, radiosurgery was per-

formed. The patient continues to have dense aphasia and right hemiparesis 9 months after hemorrhage. He will undergo follow-up arteriography 2 years after radiosurgery (or sooner if MR imaging demonstrates a significant diminution in AVM size) to monitor therapeutic response.

*Case 12*

A 56-year-old woman with a history of headaches and visual disturbances underwent surgical clipping of a single feeding artery to a right medial temporooccipital AVM 8 years prior to current admission. In preparation for definitive surgical resection of the AVM (second operation), branches of the right occipital and posterior cerebral arteries were embolized with PVA particles, but surgical resection was subtotal. Therefore, with the catheter positioned at the plexal point, embolization of the right AChA was performed with PVA particles (500–700 μm) to facilitate control of deep arterial supply to the AVM before the third operation (Figs. 2A–2C). This procedure dramatically reduced AVM flow (Figs. 2D and 2E), and the cisternal AChA segment was preserved; however, the patient suffered transient mild weakness of the left hand, which resolved completely over 4 hr after vigorous IV volume expansion. Repeat surgery allowed complete removal of the AVM, which was found to extend minimally into the cerebral peduncle, and resulted in left spastic hemiparesis.

*Case 13*

A 12-year-old girl had an intraventricular hemorrhage involving the trigone and temporal horn of the left lateral ventricle and the third

Fig. 1.—Case 11: 23-year-old man with massive intraparenchymal and intraventricular hemorrhage.

*A*, Axial noncontrast CT scan shows acute parenchymal hematoma with mass effect involving left internal capsule and lentiform nucleus; intraventricular extension is seen in both occipital horns. Patient had had emergent craniotomy for clot removal to reduce mass effect.

*B*, Left internal carotid injection, lateral projection, shows AVM in medial temporal lobe.

*C*, Left anterior choroidal artery injection, lateral projection, prior to embolization, with catheter tip at plexal point. Note pseudoaneurysm in AVM nidus (*arrow*).

*D*, Postembolization left internal carotid injection, lateral projection, shows obliteration of posterior aspect of AVM. Platinum coils were placed in distal anterior choroidal artery.



A



B



C



D
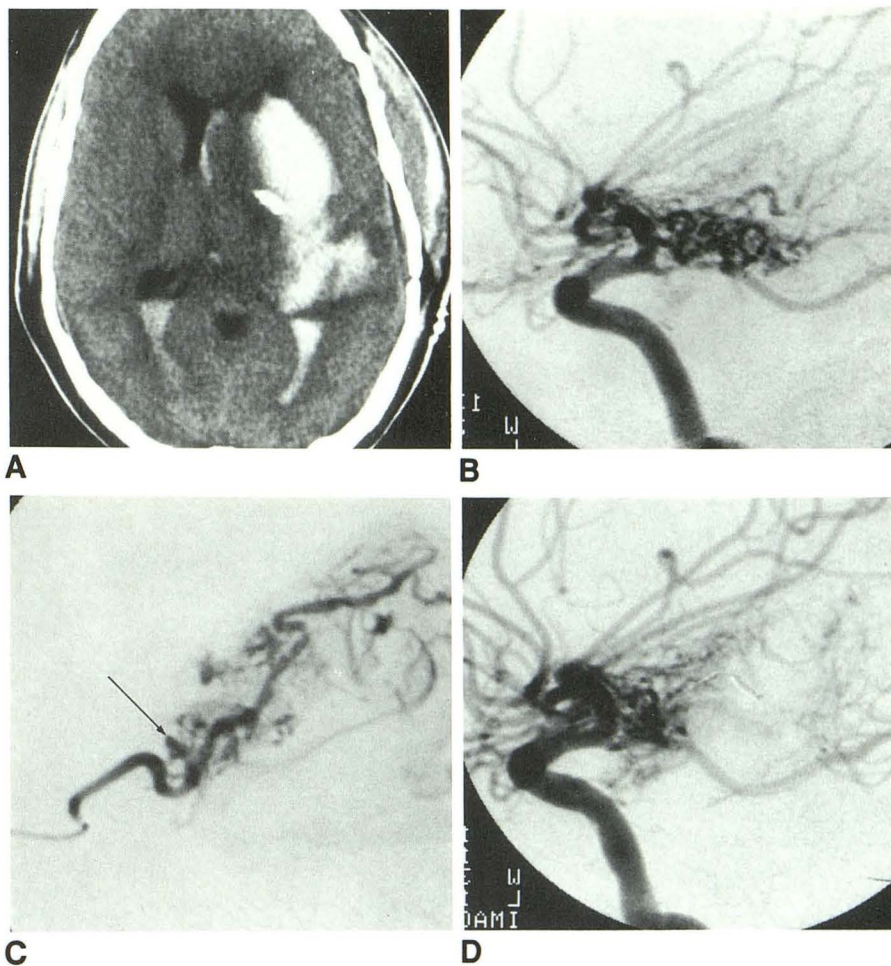
ventricle (Fig. 3A), resulting in somnolence and moderate right arm and leg weakness. Arteriography showed a left medial temporal lobe AVM (Fig. 3B), supplied by the AChA (Fig. 3C) and left lateral posterior choroidal artery. A small pseudoaneurysm seen on AChA injection prompted embolization of this artery (PVA particles, 500–700 $\mu$m). Although the Tracker catheter was positioned at the plexal point for embolization, retrograde thrombosis to the proximal cisternal AChA segment (Fig. 3D) resulted in complete right hemiplegia, which improved to moderate arm and mild leg weakness over the next several weeks. Infarction of the posterior limb of the left internal capsule was documented by MR (Fig. 3E). Radiosurgery was performed; follow-up arteriography will be needed to monitor therapeutic response. Nine months after embolization, the patient continues to have mild to moderate right arm and mild right leg weakness.

*Case 14*

A 52-year-old man suffered generalized seizures from a right anterior temporal lobe AVM (Fig. 4A) supplied by the AChA and several middle cerebral artery branches (Figs. 4B–4D). PVA embolization (300–500 $\mu$m) of the AChA was performed (Figs. 4E and 4F) to facilitate surgical control of these deep arterial feeders at surgical resection. Several anterior division middle cerebral AVM feeding

arteries were also embolized. Complete surgical resection, documented angiographically, left the patient seizure-free and neurologically intact 17 months after treatment.

**Results**

Angiographically evident flow reduction to the AVM was achieved by AChA embolization in 14 patients (15 procedures). The end point for embolization is as described earlier: either pseudoaneurysm eradication or flow diminution to the point at which further embolization might result in vessel thrombosis or reflux, as determined visually. Pseudoaneurysms identified in the AVM nidus in four patients were obliterated. In one of these patients, four platinum coils were placed in the distal AChA to complete pseudoaneurysm obliteration after PVA embolization. As it is difficult to quantify percentage of AVM flow diminution or proportion of nidus obliteration, visual inspection, both at real-time fluoroscopy and at film review, provided the basis for judging whether there was reduction in flow.

Embolization reduced the Spetzler-Martin classification [2] in one patient (case 12) by diminishing the size of the AVM nidus. Within this study, impact of preoperative embolization
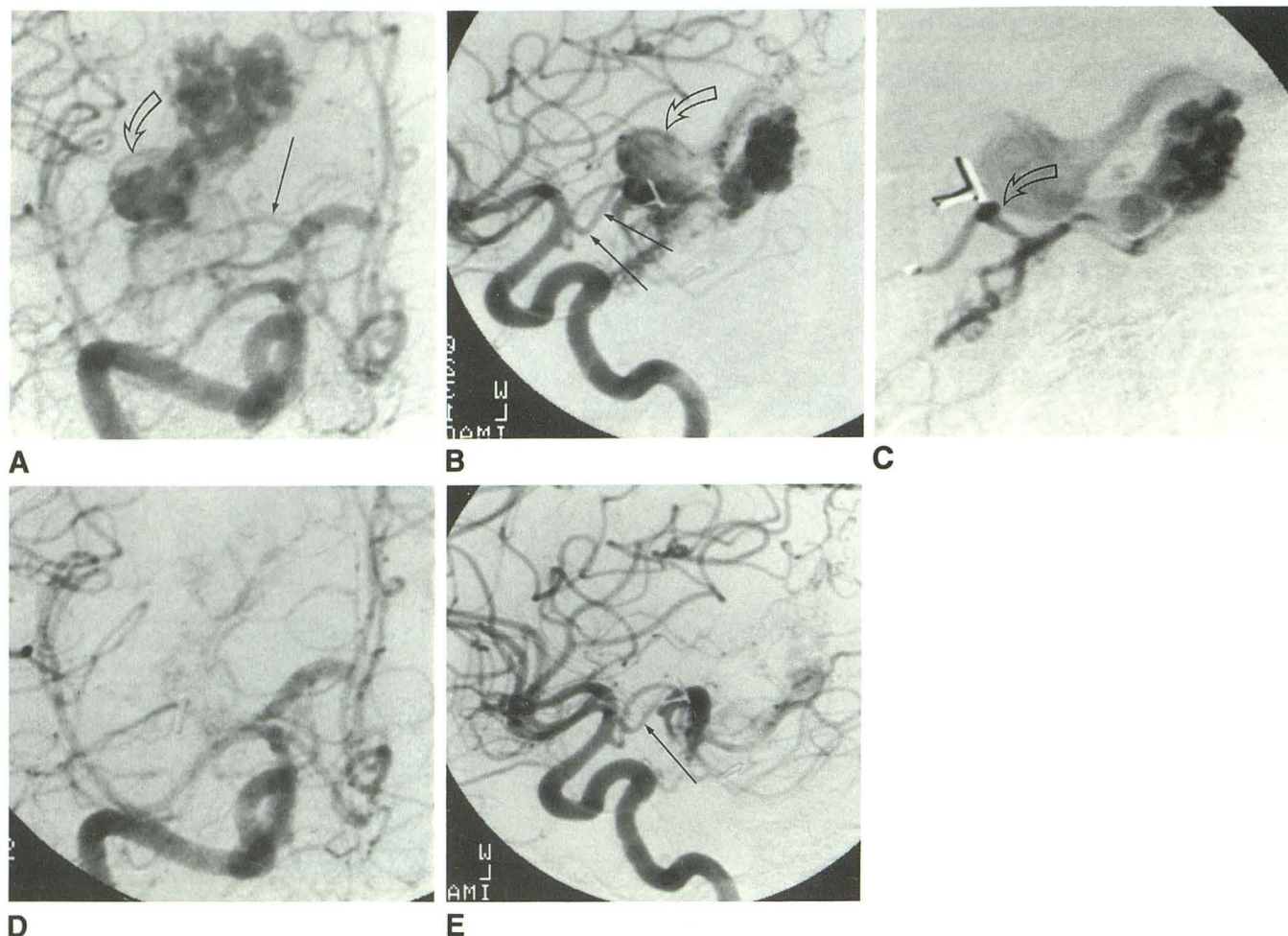
Fig. 2.—Case 12: 56-year-old woman with long history of headaches and visual disturbances, who had undergone surgical clipping of AVM feeding artery 8 years prior to current embolization.

A and B, Anteroposterior (A) and lateral (B) projections, right internal carotid injections, show posterior medial temporal lobe AVM supplied by anterior choroidal artery (long arrows). A large varix (open arrows) participates in venous drainage.

C, Right anterior choroidal artery injection, lateral projection, shows AVM supply. Catheter tip was advanced to a more distal location prior to embolization (arrow). Metallic clips remain from prior surgery.

D and E, Anteroposterior (D) and lateral (E) projections, postembolization right internal carotid injections, show markedly diminished arteriovenous shunting, with preservation of proximal anterior choroidal artery (arrow in E). The patient suffered mild left-hand weakness, which cleared within 4 hr following the procedure.

on surgical removal of the AVM (diminished blood loss, operative time, percentage of AVM able to be resected) cannot be determined as there is no control group without embolization.

In those patients who underwent surgical resection, control angiography was performed within 1 week of the operation to exclude residual AVM. Complete resection was verified angiographically in eight of nine patients; one patient had a small residual AVM that was treated with radiosurgery 6 weeks after resection (case 3). In patients treated with embolization and radiosurgery, control angiography is performed when follow-up MR imaging shows a definite change in the AVM nidus or 2 years after radiation if no demonstrable change is seen on MR images.

There was one permanent and three transient complications among the 16 procedures. Retrograde thrombosis of the AChA immediately after embolization resulted in hemiplegia in a 12-year-old girl, which improved to a mild hemiparesis over the next several weeks. Transient hand weakness after AChA embolization in another patient resolved completely over a period of 4 hr. Perforation of the distal AChA during embolization resulted in asymptomatic contrast extravasation into the trigone of the lateral ventricle in a third patient. In the fourth patient, perforation of the AChA origin during attempted catheterization resulted in asymptomatic parenchymal contrast extravasation into the medial temporal lobe, aborting the procedure prior to embolization. The AChA remained patent.
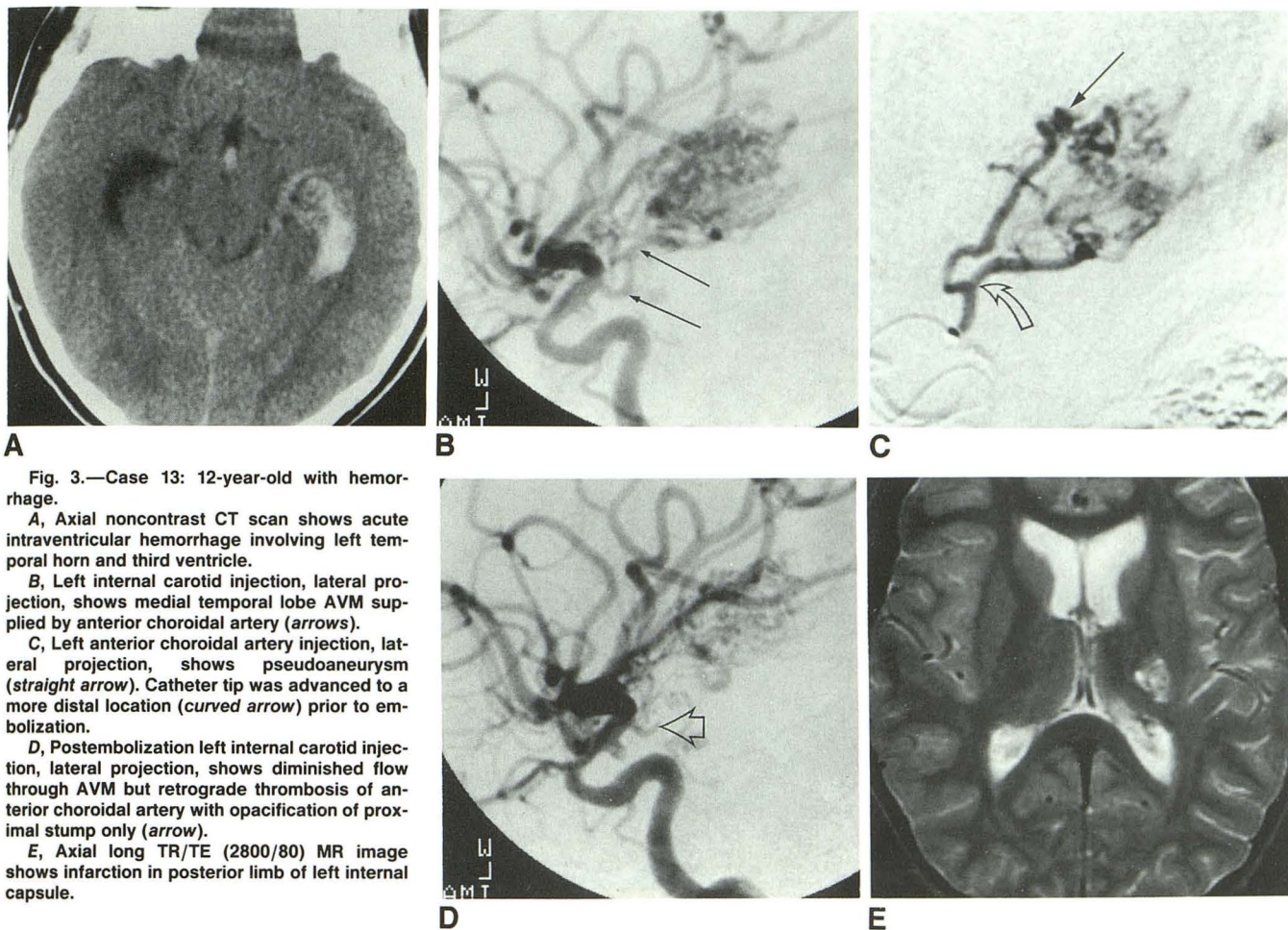
**A**

**B**

**C**

**D**

**E**

Fig. 3.—Case 13: 12-year-old with hemorrhage.

*A*, Axial noncontrast CT scan shows acute intraventricular hemorrhage involving left temporal horn and third ventricle.

*B*, Left internal carotid injection, lateral projection, shows medial temporal lobe AVM supplied by anterior choroidal artery (*arrows*).

*C*, Left anterior choroidal artery injection, lateral projection, shows pseudoaneurysm (*straight arrow*). Catheter tip was advanced to a more distal location (*curved arrow*) prior to embolization.

*D*, Postembolization left internal carotid injection, lateral projection, shows diminished flow through AVM but retrograde thrombosis of anterior choroidal artery with opacification of proximal stump only (*arrow*).

*E*, Axial long TR/TE (2800/80) MR image shows infarction in posterior limb of left internal capsule.

## Discussion

The microsurgical anatomy of the AChA has been well described [1, 3–12]. Arising most commonly from the supraclinoid ICA, the AChA can be divided into two segments: the cisternal and plexal. The *cisternal* segment extends from the AChA origin to the choroidal fissure. Invariably, supply to crucial territories originates in this segment. Regularly supplied structures include the internal capsule (posterior limb and retrolenticular portion), medial globus pallidus, cerebral peduncle, optic tract, and lateral geniculate body. Occasionally, the caudate tail, amygdala, thalamus, and hypothalamus receive AChA supply. Infarction in this territory can be devastating, combining the triad of hemiplegia, hemianesthesia, and homonomous hemianopsia in its complete form [13–15]. Therefore, the catheter tip must be placed beyond this segment or completely within an AVM feeding branch prior to embolization to avoid such a complication. The *plexal* segment begins anteriorly at the plexal point, where the vessel enters the choroidal fissure to penetrate the choroid plexus of the temporal horn. The lateral posterior choroidal artery provides variable collateral supply [16].

The AChA can be recruited to supply AVMs involving these structures. Surgical access to such feeding arteries can be difficult because of their depth [17, 18]; the surgeon would encounter the AVM superficial to these choroidal feeding arteries, hindering surgical interruption. Therein lies the benefit of preoperative AChA embolization. Embolization of this artery could also complement radiosurgery by diminishing arteriovenous shunting or by providing palliation by obliterating a nidus aneurysm, as seen in four of our cases. In our series, angiographically decreased flow to the AVM was achieved in all but one patient. Although it was not a goal of this study to quantify the beneficial impact of preoperative embolization on surgical resection, embolization has led to shorter surgical procedures, better definition of operative margins, and less intraoperative blood loss [19].

Among our four complications, two were symptomatic and one was permanent. One of the factors that increases the risk of this procedure is the necessity of navigating a catheter into a small-caliber artery that has an abrupt angulation at its origin. One of our asymptomatic complications, perforation of the AChA origin, occurred while we were attempting this maneuver. Steaming a tight "C"-shaped curve into the tip of the Tracker catheter facilitates AChA catheterization. Also, the development and use of smaller-diameter catheters would
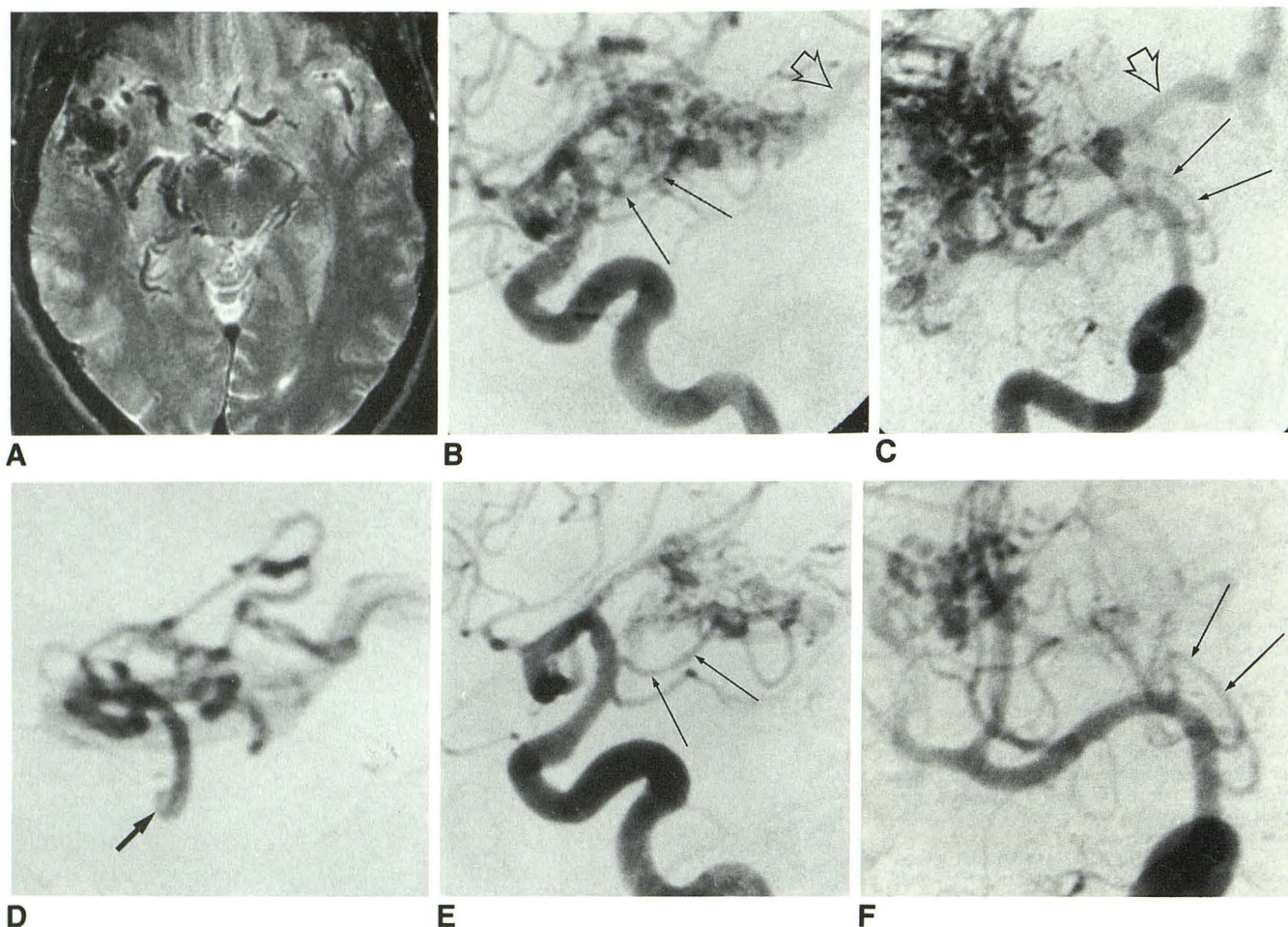
Fig. 4.—Case 14: 52-year-old man with seizures.

*A*, Axial long TR/TE (2800/80) MR image shows right anterior temporal lobe AVM.

*B* and *C*, Lateral (*B*) and anteroposterior (*C*) projections, right internal carotid injections, show right anterior temporal lobe AVM supplied by anterior choroidal artery (*long arrows*) and middle cerebral branch arteries. Note deep venous drainage (*open arrows*) via perimesencephalic vein.

*D*, Superselective distal right anterior choroidal artery injection, lateral projection, in preparation for embolization. Catheter tip is located at plexal point (*arrow*).

*E* and *F*, Lateral (*E*) and anteroposterior (*F*) projections, postembolization right internal carotid injections, show diminished flow to AVM and preservation of anterior choroidal artery (*arrows*). Middle cerebral branches have also been embolized. The patient underwent complete surgical resection of the AVM.

enhance the ability to catheterize the AChA more safely, but would inhibit the delivery of larger particulate emboli.

Once the catheter is positioned for embolization in the AChA, flow restriction by the catheter in the relatively small arterial lumen can hinder visual evaluation of the progress of embolization: it is difficult to determine whether slow arterial flow is caused by catheter restriction or by hemodynamically significant AVM embolization. This could lead to excessive embolization, which could result in retrograde thrombosis of the AChA (as in our case 13, which resulted in permanent neurologic deficit) or reflux of emboli into proximal AChA branches or the ICA. In such a situation, it is safer to under-embolize the AChA rather than to risk stroke by overemboli-zation.

Selection of appropriate particle size is essential to avoid occlusion of small, normal perforating branches of the AChA.

Smaller particles (less than 300 μm) should be avoided as they would be more likely to lodge in these branches. Conversely, the use of large particles (greater than 1000 μm) could result in proximal AChA occlusion, depending on AChA size and flow.

Careful particulate embolization of the AChA is possible, especially if the catheter tip is placed beyond the plexal point, as adjunctive therapy in the treatment of cerebral AVMs. However, the procedure carries increased risk compared with embolization of other arteries because of difficult catheterization and vascular supply to crucial territories.

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