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Embolization of branches arising from the cavernous portion of the internal carotid artery.

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Embolization of Branches Arising from the Cavernous Portion of the Internal Carotid Artery

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Seven patients with vascular disease (four with cavernous and two with transverse sinus dural fistulas and one with a facial arteriovenous malformation, all supplied primarily from cavernous branches of the internal carotid artery) underwent subselective catheterization and embolization. Ten branches were catheterized (seven meningohypophyseal trunks and three inferolateral trunks) and eight branches were embolized. The embolic agents were as follows: polyvinyl alcohol particles in five, hypertonic glucose in two, and liquid adhesive in one. All four cavernous sinus dural fistulas were occluded after embolization. In the two transverse sinus fistulas, the goal of embolization was to obliterate the tentorial supply prior to surgery. This goal was achieved in both cases. In the remaining patient, who had a large facial arteriovenous malformation and recurrent epistaxis, the embolization obliterated the cavernous supply to the nasal cavity and the patient remains hemorrhage-free.

Despite one complication, which occurred in the first case when a thrombus formed and dislodged, resulting in an embolic stroke, this study shows that subselective catheterization and embolization can obliterate lesions of the branches arising from the cavernous internal carotid artery.

Diseases involving the cavernous sinus and parasellar regions derive their vascular supply from a number of sources. The external carotid artery has a meningeal supply to these regions that arises from branches of the internal maxillary, ascending pharyngeal, middle meningeal, and accessory meningeal arteries [1, 2]. These branches have anastomoses with cavernous branches of the internal carotid artery; namely, the inferolateral trunk (ILT) and the meningohypophyseal trunk (MHT) (Fig. 1). This balanced input from both internal and external carotid arteries allows dural supply to disease processes in the cavernous sinus and parasellar regions [3]. Embolization of the external carotid arterial supply may completely obliterate the vascular supply to a lesion in this territory, but it may also cause hypertrophy of the internal contribution. Techniques that aid obliteration of lesions with dual supply include the use of liquid adhesive agents that permeate into the nidus with systemic hypotension and/or the temporary occlusion of other shared vascular pedicles. While these techniques may assist in the embolization of the external carotid supply, anatomic variations or previous hemodynamic constraints, such as prior ligation of the external carotid artery, may favor the vascular supply arising primarily from the cavernous internal carotid artery. In such instances, superselective catheterization and embolization of the cavernous supply may obliterate these lesions. We report our experience with seven patients treated by this technique.

Materials and Methods

Over a 3-year period, seven patients were treated by superselective catheterization and embolization of the dural branches. Patients' ages ranged from 27 to 66 years (mean, 47 years). A summary of the diseases treated, embolic agents used, vessels catheterized,
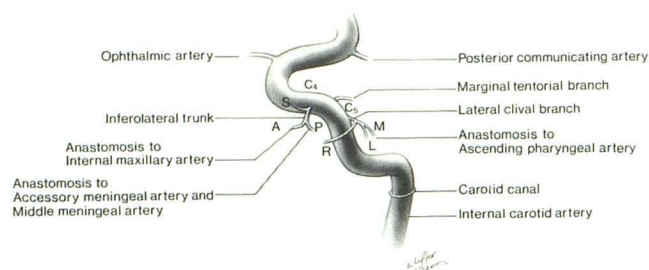
Fig. 1.—Drawing shows dural branches arising from cavernous carotid artery. Lateral clival artery has medial (M) and lateral (L) branches that anastomose with ascending pharyngeal (AP) artery. Recurrent (R) branch to foramen lacerum may have a separate origin. Marginal artery of tentorium arises superiorly from C5 portion (ascending cavernous) of internal carotid artery. Inferolateral trunk arises from C4 (horizontal cavernous carotid) and has superior (S), anterolateral (A), and posterior (P) branches that anastomose with ophthalmic, internal maxillary (IMA), accessory meningeal (AM), and middle meningeal (MM) arteries, as shown.

outcome, follow-up, and complications appears in Table 1. In six patients the procedure was performed from a transfemoral route, and all had systemic anticoagulation with 5000 units of heparin followed by 2500 units/hr during the procedure. Transfemoral catheterization was impossible in the seventh patient; therefore, a direct puncture of the involved carotid artery was performed. Systemic anticoagulation was not done owing to the risk of bleeding from the neck puncture site.

A 5- or 7-French catheter was placed in the distal internal carotid artery and a smaller catheter was advanced coaxially into the cavernous internal carotid artery. In our earliest case a 2-French polyethylene catheter was used. In the remaining cases a 3.0-French Tracker* catheter with a 0.014-in. platinum steerable guidewire was used. The space between the guidewire and the Tracker catheter as well as the space between the two catheters was perfused with heparinized saline by means of sidearm adapters. Appropriate curves were formed in the distal catheter tips to assist in catheterization. Most often, a tight radius C-shape was adequate to engage the orifices, but in two instances an S-shape was required for stability. Selective angiograms were obtained in all cases to watch for reflux and to document the supply from the pedicle. Ten branches were catheterized (seven MHTs and three ILTs) and eight were embolized. In the two pedicles that were not embolized, spontaneous thrombosis of the dural fistula was noted during the catheterization procedure. In both cases the flow to the fistula was extremely slow because of successful embolization from the contralateral MHT supplying the same fistula site. The embolic agents used in the eight pedicles were as follows: polyvinyl alcohol (PVA) particles in five, hypertonic glucose in two, and IBCA[†] in one. The PVA particles[‡] ranged from 200 to 300 μm in size. In case 6, particles from 500 to 700 μm were used because of large shunts within the nidus of the fistula. The PVA particles were suspended in a mixture of iopamidol[§] and normal saline. These injections were monitored under real-time digital subtraction technique. Dilute mixtures were used to prevent clumping and occlusion of the catheter. Injections were given during real-time digital subtraction, and reflux was watched for carefully. When occlusion of the pedicle was observed, the catheter was removed. We saw no instances of reflux and had no embolic complications. In two patients, hypertonic glucose was used as a sclerosing agent. Hypertonic glucose (dextrose 50%) and hypertonic contrast (Hypaque 76%) were mixed in equal amounts. This mixture was also injected under real-time digital subtraction. In the remaining patient (case 2), 0.10 ml of a mixture of 50% IBCA and 50% iophendylate [‖] was injected into the ILT by the "sandwich push" technique. To ensure the stability of the catheter, a nondetachable balloon was transiently inflated across the origin of this vessel.

Several techniques were employed to reduce the risk of distal embolization, including systemic anticoagulation and perfusion of the spaces between the guidewire and catheter and between the two catheters with heparinized saline. The guidewire was removed frequently and cleaned to prevent the formation of platelet thrombi on the guidewire tip. The poor stability of the catheter within the origin of the cavernous branches required the injection of small amounts of contrast material mixed with emboli. The use of high-quality digital subtraction angiography as well as real-time subtraction for the embolization was used to discern any small amount of reflux during the procedure. Any reflux into the carotid artery would be immediately diluted and would not be appreciated without the use of high-quality real-time subtracted images. Dilute mixtures of PVA were embolized to ensure that clumping or blockage of the catheter did not occur.

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[§] Squibb Diagnostics, New Brunswick, NJ.
[‖] Pantopaque, Lafayette Pharmacal, Inc., Lafayette, IN.

TABLE 1: Summary of Cavernous Internal Carotid Catheterizations

Case No.	Age	Gender	Disease	Branches	Embolic Agent	Outcome/Follow-up in Months	Complications
1	56	F	R cavernous DAVF	R MHT	D_{50}	Cure/48	Embolic stroke
2	36	F	L cavernous DAVF	L ILT	0.2 ml IBCA	Cure/16	None
3	66	M	R cavernous DAVF	L and R MHT and R ILT	PVA	Cure/6	None
4	47	M	R transverse DAVF	R MHT	D_{50}	MHT supply occluded/16	None
5	32	F	L facial AVM	L ILT	PVA	ILT supply occluded/10	None
6	63	F	L and R cavernous DAVF	L and R MHT	PVA	Cure/5	None
7	27	F	L transverse DAVF	L MHT	PVA	MHT supply occluded/16	None

R = right, L = left, DAVF = dural arteriovenous fistula, MHT = meningohypophyseal trunk, D_{50} = hypertonic glucose, ILT = inferolateral trunk, IBCA = isobutyl cyanoacrylate, PVA = polyvinyl alcohol sponge particles.

During the embolization procedure, we frequently cleared the catheter of emboli and performed a repeat angiogram to ensure that reflux or perfusion of unwanted vascular territories was not occurring. After embolization, the circulating anticoagulants were reversed with IV protamine sulfate (1 mg reverses 100 units of circulating heparin) given slowly over 15 min. Angiograms were performed from all potential vascular pedicles.

Results

In four patients (cases 1, 2, 3, 6), the vascular pathology was a dural cavernous fistula supplied primarily by branches of the internal carotid artery. Two patients had embolization of the trace external supply without improvement. In the remaining two patients the supply was too small to embolize. In all four patients the fistula was completely closed and the symptoms were resolved after the cavernous dural supply was embolized.

In cases 4 and 7, the goal of embolization of the MHT was to eliminate the supply to a large transverse sinus dural fistula in preparation for surgical exposure. This goal was achieved with obliteration of the tentorial supply.

In case 5, the patient with a large facial AVM, previous surgical ligation of both external carotid arteries had increased the supply from the cavernous branches of the internal carotid. The patient presented with recurrent epistaxis. Embolization of the inferolateral trunk with PVA caused the bleeding episodes to stop. The follow-up period ranged from 5 to 48 months (mean, 16 months).

The only complication was in our earliest case (case 1, Fig. 2). A transfemoral catheterization was unsuccessful and so a direct puncture of the carotid artery was performed. Because of the risk of bleeding from the neck puncture site, systemic anticoagulation was not done. The embolization procedure was completed without deficit. When the neck catheter was removed the patient developed a hemiparesis, presumably from a clot dislodged from outside the 5-French catheter.

Representative Case Reports

Case 3 is a 66-year-old man with progressive proptosis, chemosis, and visual loss in both eyes over a 2-week period. Angiography revealed a right cavernous dural fistula with primary supply from the right ILT and the left and right MHT (Figs. 3A and 3C). The right ILT and MHT were selectively catheterized and embolized with PVA, resulting in obliteration of this supply (Fig. 3B). During attempted catheterization of the left MHT, spontaneous thrombosis was noted (Fig. 3D).
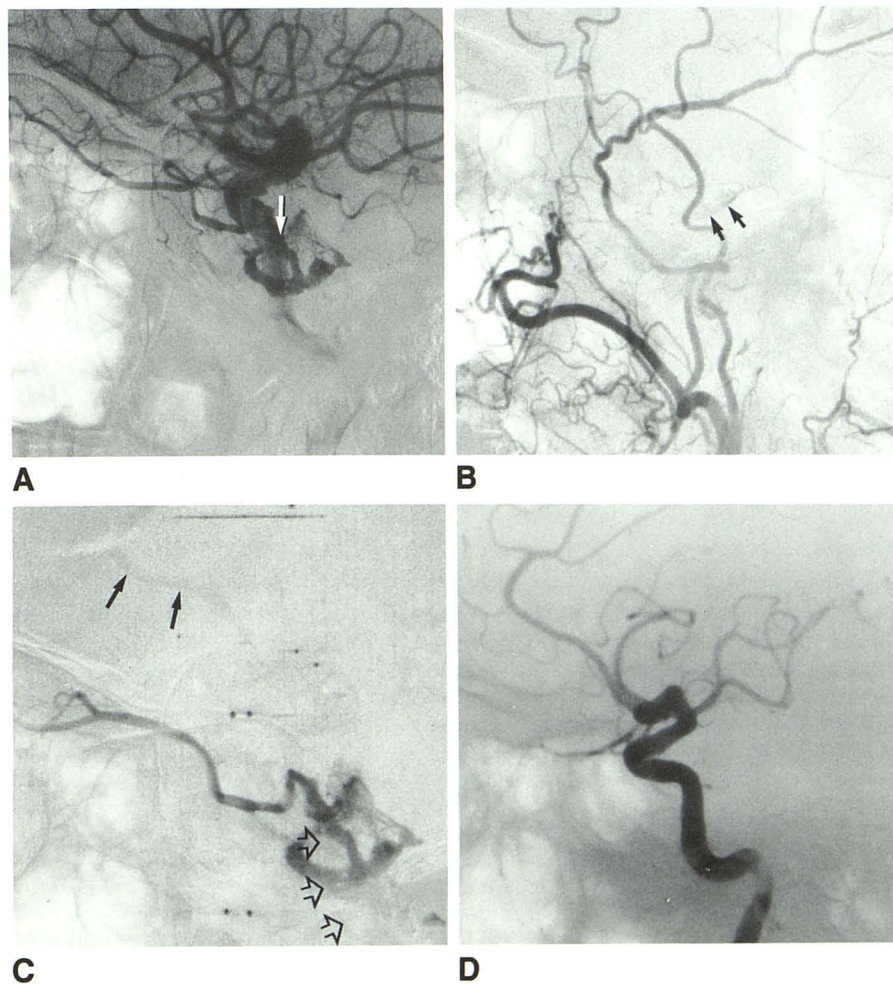

Fig. 2.—Case 1.

A, Right internal carotid angiogram shows cavernous dural fistula supplied by meningohypophyseal trunk (MHT) (*arrow*).

B, Right external carotid angiogram shows faint supply to fistula (*arrows*) arising from artery of foramen rotundum.

C, Selective injection of MHT through a 2-French catheter (*open arrows*). Note small amount of reflux into anterior cerebral artery (*closed arrows*).

D, Right internal carotid angiogram, lateral view, after embolization, shows obliteration of fistula.
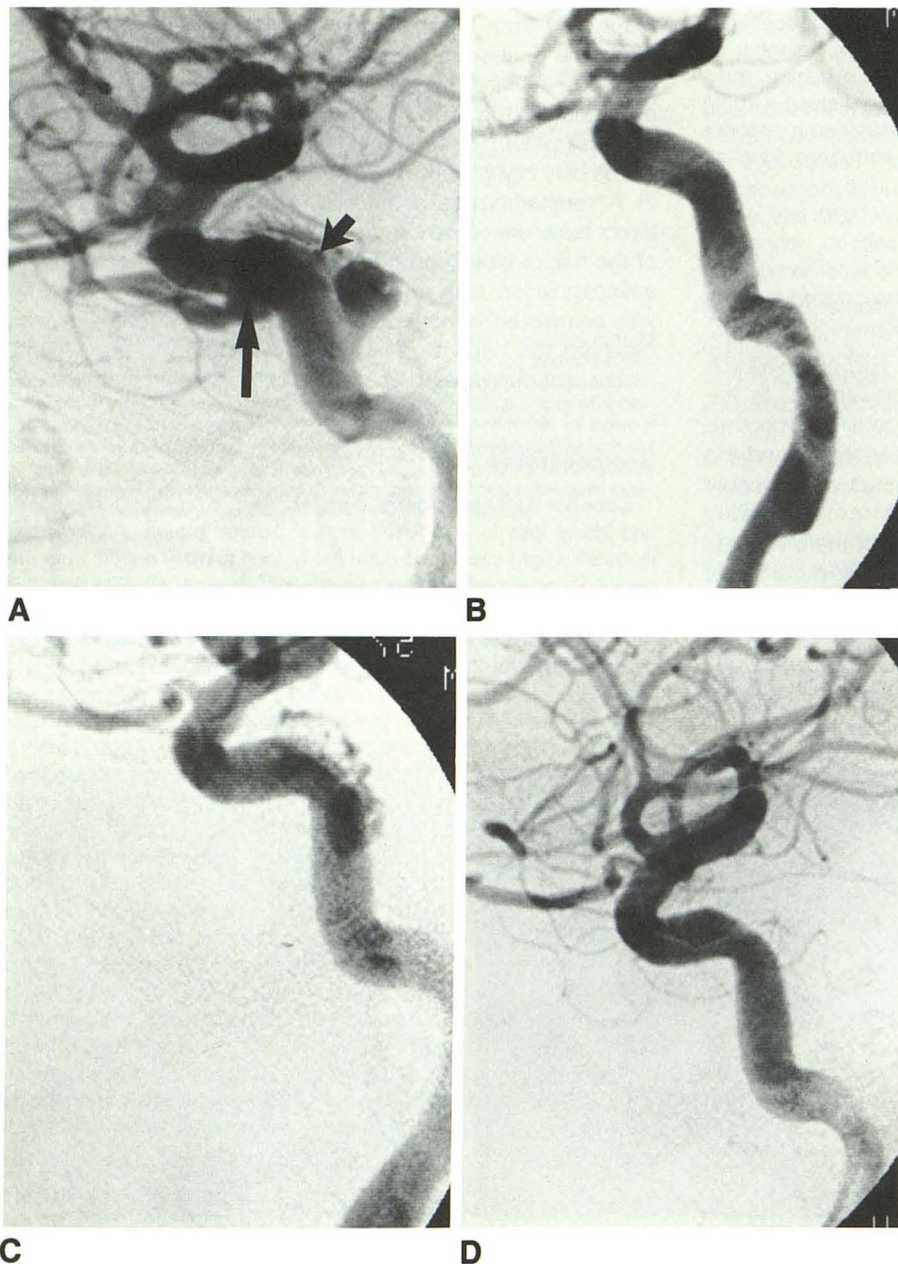
A

B

C

D

Fig. 3.—Case 3.
A, Right internal carotid artery injection, lateral view, shows cavernous dural fistula supplied by meningohypophyseal trunk (MHT) (*short arrow*) and inferolateral trunk (ILT) (*long arrow*).
B, Same vessel after embolization of MHT and ILT with polyvinyl alcohol sponge particles.
C, Left internal carotid angiogram, lateral view, shows supply from MHT.
D, Same vessel after attempted embolization shows occlusion of fistula.

Case 5 (Fig. 4) is a 32-year-old woman with a large facial AVM. Previous ligation of the external carotid arteries had recruited a supply from the inferolateral trunk. The patient presented with recurrent epistaxis. The ILT was selectively catheterized (Fig. 4B) and embolized with PVA particles. A follow-up angiogram revealed complete obliteration of the supply from this pedicle, and the patient remains free of hemorrhage.

Case 2 (Fig. 5) is a 34-year-old woman with progressive proptosis, chemosis, and headaches. An angiogram demonstrated a dural cavernous fistula supplied by the inferolateral trunk and some external supply from the artery of the foramen rotundum and an accessory meningeal artery (Figs. 5A and 5B). Selective catheterization of the external branches could not be performed because of their small caliber. A Tracker catheter was advanced into the ILT and a nondetachable balloon inflated transiently across the origin to prevent reflux

(Fig. 5C). A small injection of IBCA caused complete obliteration of the fistula without deficit (Fig. 5D).

Case 7 (Fig. 6) is a 28-year-old woman with a dural fistula involving the transverse sinus that was partially treated by transvenous coil embolization. The patient had progressive visual loss secondary to the remaining fistula. The MHT was selectively catheterized and embolized with PVA particles, resulting in complete obliteration of the supply from this pedicle (Fig. 6B).

Discussion

Vascular disease involving the cavernous sinus region derives its blood supply from either the internal carotid artery, the external carotid artery, or both. For example, Barrows et
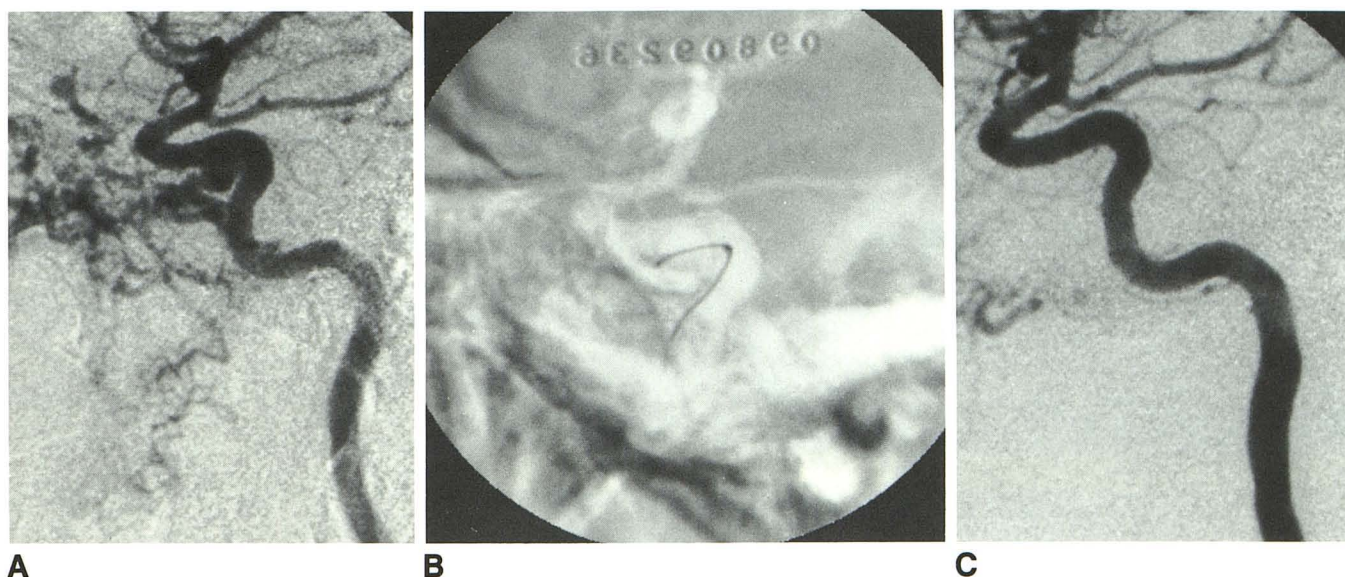
A   **B**   **C**

Fig. 4.—Case 5.
A, Left internal carotid angiogram shows supply to a facial AVM from enlarged inferolateral trunk (ILT).
B, Roadmap during catheterization shows platinum wire in origin of ILT.
C, Left internal artery injection after embolization shows obliteration of supply from ILT. The remaining supply from vidian artery was not treated.
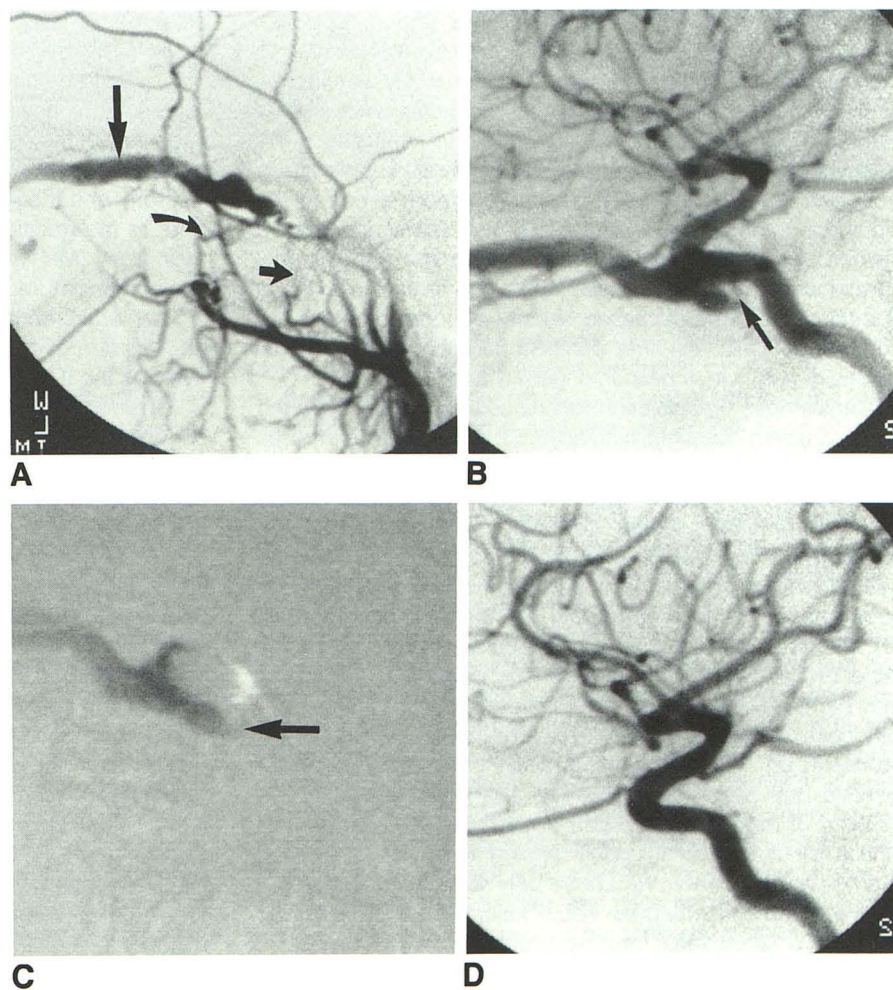

A   **B**

C   **D**

Fig. 5.—Case 2.
A, Left external carotid angiogram, lateral view, shows dural fistula supplied by artery of foramen rotundum (*curved arrow*) and accessory meningeal (*short straight arrow*) with drainage to superior ophthalmic vein (*long straight arrow*).
B, Left internal carotid angiogram, lateral view, shows supply to same fistula from enlarged inferolateral trunk (ILT) (*arrow*).
C, A small catheter has been placed into ILT. A balloon has been positioned across the origin to prevent reflux. The catheter tip is indicated by *arrow*.
D, Left carotid angiogram, after embolization, shows complete obliteration of fistula.
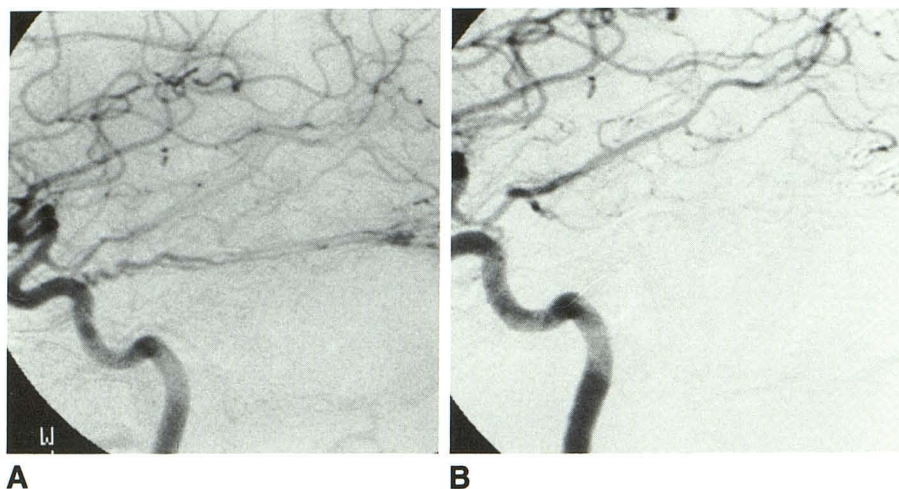
A   **B**

Fig. 6.—Case 7.
A, Left internal carotid angiogram, lateral projection, shows supply to a transverse sinus dural arteriovenous fistula from meningohypophyseal trunk.
B, Same vessel, after embolization, shows occlusion of MHT supply to fistula.

al. [4] subdivided dural cavernous carotid fistulas according to their vascular supply: internal carotid supply (type B), external carotid supply (type C), and mixed supply (type D). Traditional transvascular embolization of the external carotid supply can result in cure in type-C fistulas and in some type-D fistulas. Debrun et al. [5] have reported the difficulty in achieving anatomic cures in type-D fistulas by embolizing the external carotid alone. We have found that temporary occlusion of the internal carotid supply can increase permeation of the nidus during external embolization. Figure 7 is an example of a patient with a dural fistula involving the cavernous sinus with dural supply. The primary supply is from branches of the external carotid artery; however, an additional supply arises from both the ILT and MHT. To improve permeation of the nidus during embolization of the external carotid branches, a balloon was placed transiently across the origin of the internal carotid artery, which increased the supply from the external branches. Note the increased density of contrast in the superior ophthalmic vein after transient occlusion of the internal carotid supply with a balloon (Figs. 7C and 7D).

Transvenous embolization techniques have been reported for dural cavernous sinus fistulas [6, 7]; however, these routes are not always available. When the inferior petrosal vein is not present, venous drainage is rerouted through cortical or superior ophthalmic venous pathways, which can aggravate ocular symptoms [8]. It is important to recognize that the natural history of cavernous dural fistulas is diverse. While spontaneous closure can occur in some of these fistulas [9], others, particularly those with primary drainage to the superior ophthalmic vein and venous occlusive changes in that pathway, can cause severe proptosis, visual loss, and blindness [8]. The decision to treat an individual patient should be based on an understanding of the natural history of the disease, the patient's symptoms, and projected risks and benefits of treatment. Because of the potential risks of embolization of the dural supply arising from the cavernous internal carotid artery, we chose only to treat patients with severe symptoms (marked visual loss, life-threatening epistaxis) in whom a safer treatment route was not available.

An understanding of the vascular anatomy and anastomoses with other vascular pedicles (see Fig. 1) is essential for safe embolization. The classical description of the first branch originating from the cavernous segment of the internal carotid artery is the MHT. It projects posteriorly and laterally, immediately trifurcating into three branches: the tentorial branch (of Bernasconi and Cassinari), the dorsal meningeal, and the inferior hypophyseal arteries [10–14]. Anatomic dissections by Lasjaunias et al. [15, 16] have shown that considerable variation exists in the origin of these vessels, and more commonly these arise separately from the C5 portion (ascending segment) of the cavernous carotid artery. These investigators described the separate origins and anatomic variations of the posteroinferior hypophyseal artery, the lateral artery of the clivus, the recurrent artery of the foramen lacerum, and the lateral artery of the trigeminal ganglion. We refer to the traditional description of these branches as one main trunk (MHT), while recognizing the oversimplification of this nomenclature.

The dural branches arising from the C4 (horizontal cavernous) portion of the internal carotid artery are known collectively as the ILT. Lasjaunias et al. [17, 18] have shown there is considerable variation in the supply to this region. Most commonly, the trunk passes over the sixth nerve giving off four branches: a superior branch anastomosing with the superficial recurrent ophthalmic artery; an anteromedial branch that enters the superior orbital fissure to supply the third, fourth, and sixth nerves terminating in the deep recurrent ophthalmic artery; an anterolateral branch that anastomoses with the artery of the foramen rotundum; and a posterior branch that supplies both the fifth and seventh nerves, finally anastomosing with the accessory meningeal artery.

Important anastomoses exist between the external carotid artery and the cavernous branches of the internal carotid artery [19]. The neuromeningeal branches of the ascending pharyngeal artery have clival connections with both the dorsal meningeal (lateral clival artery) and the inferior hypophyseal arteries [20, 21]. The internal maxillary and accessory meningeal arteries have anastomoses with the ILT through the foramen rotundum and foramen ovale, respectively [18]. The middle meningeal artery has a petrous communication with the ILT and MHT [22]. Anatomic variations of the dural supply are common; for example, the accessory meningeal artery

Fig. 7.—A, Right external carotid arteriogram, lateral view, shows cavernous dural sinus fistula, supplied by arteries through foramen rotundum (*curved arrow*) and ovale (*straight arrow*) with drainage to superior ophthalmic vein (SOV).

B, Right internal carotid arteriogram, lateral view, shows inferolateral trunk (ILT) (*straight arrow*) and meningohypophyseal trunk (MHT) (*curved arrow*) supply to same fistula.

C, Right internal maxillary injection shows opacification of fistula. Washout of unopacified blood from internal supply accounts for faint density in SOV.

D, Same injection after temporarily inflating balloon in the internal carotid artery across origin of MHT. Note denser opacification of SOV.
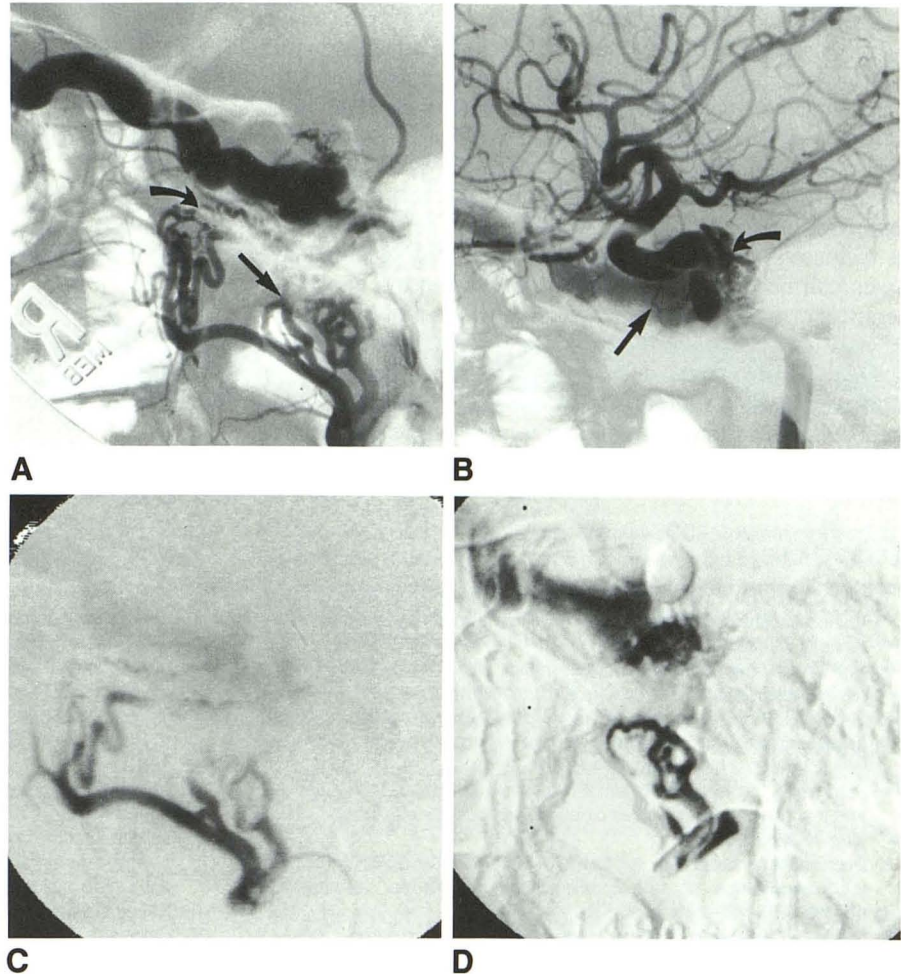
can supply the entire vascular territory of the ILT in 20% of cases [17, 18].

Because of the acute angle of the origins of the ILT and MHT from the internal carotid artery, selective catheterization is difficult. The MHT immediately branches into a multitude of small vessels, which makes it difficult to seat the catheter; and the risk of reflux into the cerebral circulation makes the embolization hazardous. In our earliest case (case 1, Fig. 2), reflux into the internal carotid artery was noted during the selective angiogram. We chose a mixture of hypertonic glucose and contrast as a sclerosing agent. In the slowly flowing fistula it promotes thrombosis, but if refluxed into the cerebral circulation it would immediately become diluted, passing harmlessly through the cerebral circulation. While affording some degree of safety, this agent has several drawbacks. The sclerosis results in thrombosis of all venous pathways perfused. If the venous outflow pathways are occluded without closure of the fistula, then the symptoms may become aggravated. The intense thrombosis and inflammation that occur result in intense pain during and immediately after the infusion. Both patients we treated had persistent thrombosis of the pedicles infused; however, recanalization is a possibility. When greater stability of the catheter could be achieved, we injected either PVA particles (five cases) or IBCA (one case), which completely closed the fistula.

One of our major concerns about embolization of the dural branches of the cavernous internal carotid has to do with the risk of reflux of embolic material and the resultant neurologic event. All seven patients in this series had either failed or were not candidates for more traditional routes of embolization. There were no embolic events related to the embolization procedure. One complication arose from clot formation on the outside of the guiding catheter.

As discussed by Lapresle et al. [23, 24], much of our knowledge of the vascular supply to the cranial nerves has been discovered on the heels of diagnostic and therapeutic angiography. The role of the blood supply to the fifth and seventh nerves was discovered during embolization of the middle meningeal arterial system [25, 26]. Similarly, knowledge of the supply to the ninth through the 12th cranial nerves came after embolization of the ascending pharyngeal artery [24]. Anatomically, the ILT and MHT have been shown to supply the third, fourth, fifth, and sixth nerves, but functional confirmation has been lacking primarily because of the rarity of embolization in this territory. Fortunately, in our small series, we have had no instances of cranial nerve dysfunction

related to the embolization procedure. In the one case in which we used IBCA, the catheter could be advanced close to the nidus. In addition, by wedging the catheter into the origin of the vessel to be embolized and injecting small amounts of embolic material, the preferential flow was to the low-resistance nidus. Although provocative tests for cranial nerves have been devised by using Xylocaine in the external carotid arteries [27], the risk of reflux and resultant seizures is excessive. In addition, we have noted that false-negative tests can occur with provocative tests when the anesthetic agent is diverted into the low-resistance shunt of the nidus.

In conclusion, advances in angiographic techniques and microcatheter delivery systems have allowed access to the cavernous internal carotid arterial branches. Our initial experience suggests that this vascular territory can be embolized with success.

REFERENCES

1. Newton TH, Hoyt WF. Spontaneous arteriovenous fistula between dural branches of the internal maxillary artery and the posterior cavernous sinus. *Radiology* **1968**;91:1147–1150
2. Newton TH, Hoyt WF. Dural arteriovenous shunts in the region of the cavernous sinus. *Neuroradiology* **1970**;1:71–81
3. Vinuela F, Fox AJ, Debrun GM, et al. Spontaneous carotid cavernous fistulae: clinical, radiological, and therapeutic considerations. Experience with 20 cases. *J Neurosurg* **1984**;60:976–984
4. Barrows PL, Spector RH, Braun IF, Landman J, Tindall GT. Classification and treatment of spontaneous carotid-cavernous sinus fistulas. *J Neurosurg* **1984**;62:248–256
5. Debrun GM, Vinuela F, Fox AJ, Davis RR, Ahn HS. Indications for treatment and classification of 132 carotid cavernous fistulas. *Neurosurgery* **1988**;22:285–289
6. Labbe D, Courtheoux P, Rigot-Jolivet M, Compere JF, Theron J. Bilateral dural carotid-cavernous fistula. Its treatment by way of the superior ophthalmic vein. *Rev Stomatol Chir Maxillofac* **1987**;88(2):120–124
7. Halbach VV, Higashida RT, Hieshima GB, Hardin CW, Pribram H. Transvenous embolization of dural fistulas involving the cavernous sinus (in press)
8. Halbach VV, Hieshima GB, Higashida RT, Reicher M. Carotid cavernous fistulas: indications for urgent therapy. *AJNR* **1987**;8:627–633, *AJR* **1987**;149:587–593
9. Picard L, Bracard S, Moret J, Per A, Giacobbe HL, Roland J. Semin Interventional Radiol **1987**;4:219–241
10. Parkinson D. Collateral circulation of cavernous carotid artery anatomy. *Can J Surg* **1964**;7:251–268
11. Parkinson D. A surgical approach to the cavernous portion of the carotid artery. Anatomical studies and case reports. *J Neurosurg* **1965**;23:474–483
12. Pribram HFW, Boulter TR, McCormick WF. Roentgenology of meningo-hypophyseal trunk. *AJR* **1966**;98:583–594
13. Cortes O, Chase NE, Leeds N. Visualization of tentorial branches of the internal carotid artery in intracranial lesions other than meningiomas. *Radiology* **1964**;82:1024–1028
14. Palacios E, Behrooz A, Williams V. The significance of the dural supply from the carotid siphon. *AJR* **1974**;125:816–822
15. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso AN, Seeger JE. Arterial anomalies at the base of the skull. *Neuroradiology* **1977**;13:267–272
16. Lasjaunias P, Moret J, Doyon D, Vignaud J. C5 collaterals at the internal carotid siphon. *Neuroradiology* **1978**;16:304–305
17. Lasjaunias P, Moret J, Mink J. The anatomy of the inferolateral trunk (ILT) of the internal carotid artery. *Neuroradiology* **1977**;215–220
18. Lasjaunias P, Theron J. Radioanatomy of the accessory meningeal artery. *Radiology* **1976**;121:99–104
19. Margolis MT, Newton TH. Collateral pathways between the cavernous portion of internal carotid and external carotid arteries. *Radiology* **1969**;93:834–836
20. Lasjaunias P, Moret J. The ascending pharyngeal artery: normal and pathological radioanatomy. *Neuroradiology* **1976**;11:77–82
21. Lasjaunias P, Doyon D. The ascending pharyngeal artery and the blood supply of the lower cranial nerves. *Neuroradiology* **1978**;5:287–301
22. Lasjaunias PL, Berenstein A. Craniofacial and upper cervical arteries: functional, clinical and angiographic aspects. Baltimore: Williams & Wilkins, **1981**
23. Lapresle J, Lasjaunias P. Cranial nerve ischaemic arterial syndromes. *Brain* **1986**;109:207–215
24. Lapresle J, Lasjaunias P, Thevenier D. Atteinte transitoire des IX, X et XII ainsi que du VII gauches au decours d'une angiographie: contribution a la pathologic ischemique des nerfs craniens. *Rev Neurologique* **1980**;136:787–791
25. Calcaterra TC, Rand RW, Bentson JR. Ischemic paralysis of the facial nerve: a possible etiologic factor in Bell's palsy. *Laryngoscope* **1976**;86:92–97
26. Lasjaunias P, Doyon D, Edouard A. Les paralysies faciales peripheriques post-embolisation. Rapport sur un cas, discussion, prevention. *Ann Otolaryngol Chir Cervicofac* **1978**;95:595–602
27. Horton JA, Kerber CW. Lidocaine injection into external carotid branches: provocative test to preserve cranial nerve function in therapeutic embolization. *AJNR* **1986**;7:105–108