AJNR

Treatment of dural fistulas involving the deep cerebral venous system.

V V Halbach, R T Higashida, G B Hieshima, C B Wilson, C W Hardin and E Kwan

AJNR Am J Neuroradiol 1989, 10 (2) 393-399
http://www.ajnr.org/content/10/2/393

Treatment of Dural Fistulas Involving the Deep Cerebral Venous System

Van V. Halbach[1,2]
Randall T. Higashida[1,2]
Grant B. Hieshima[1,2]
Charles B. Wilson[2]
Carl W. Hardin[1]
Eddie Kwan[3]

Eight patients with dural arteriovenous fistulas involving the deep cerebral venous system were treated by a combination of preoperative embolization, intraoperative embolization, and/or surgical excision. All eight patients were men 30–71 years old (mean age, 48.5). The presenting symptoms were hemorrhage in four patients (two subarachnoid, one intraventricular, and one parenchymal), stroke in two patients, and severe chronic headaches in two patients. Four patients were treated and cured by preoperative embolization of external carotid feeding vessels followed by direct intraoperative placement of liquid adhesives into the fistula site. Two patients underwent preoperative embolization followed by surgical interruption of feeding vessels to the fistula. Both patients had persistent fistulas and were subsequently treated by intraoperative embolization with liquid adhesives. One patient was cured and the second had 95% reduction in fistula size. The remaining two patients had surgical excision of the fistula, one in combination with preoperative embolization. Both were completely cured. Two patients developed hydrocephalus after placement of liquid adhesive into the involved vein of Galen and were successfully treated with placement of ventriculoperitoneal shunts. Follow-up periods ranged from 7 to 21 months (mean, 14).

We found that patients with dural arteriovenous fistulas could be treated effectively through a combination of neuroradiologic and surgical intervention.

Dural arteriovenous fistulas (AVFs) account for 10–15% of all intracranial vascular malformations [1]. The majority involve a dural sinus, usually the transverse [2], sigmoid [3], or cavernous [4]. Treatment techniques include surgical isolation or excision [5], compression therapy [6], and transarterial [7–9] or transvenous embolization [10, 11]. Only a few scattered reports of dural AVFs involving the deep cerebral venous system have been published. We have treated 97 patients with symptomatic dural AVFs in the past 9 years. In eight (8.3%) of these patients, dural fistulas involved the deep cerebral venous system. The severity of presenting symptoms, deep midline location, and complex vascular supply complicate the treatment of these lesions. Our experience with eight patients treated by a combination of embolization and/or surgery is summarized in this report.

Received April 12, 1988; accepted after revision September 28, 1988.

[1] Department of Radiology, Neurointerventional Section, University of California, San Francisco, Hospitals, 505 Parnassus Ave., San Francisco, CA 94143. Address reprint requests to V. V. Halbach.

[2] Department of Neurological Surgery, University of California, San Francisco, Hospitals, San Francisco, CA 94143.

[3] Department of Radiology, New England Medical Center, Boston, MA 02111.

AJNR **10:393–399, March/April 1989**
0195–6108/89/1002–0393

Materials and Methods

The patients' age, gender, symptoms, presentation, fistula location, feeding arteries, treatments, outcome, follow-up period, and complications are given in Table 1. The patients were 30–71 years old (mean, 48.5 years), and all eight were men. None had a history of severe head trauma or previous CNS infection. The most common presenting symptom was headache (five patients, three secondary to hemorrhage). Four patients had hemorrhage, with two (cases 1 and 2) presenting with subarachnoid hemorrhage, one (case 3) an intraventricular hemorrhage, and another (case 5) a cerebellar hematoma. Three of the four patients with hemorrhage had severe venous occlusive disease involving the draining veins. Two patients had strokes related to their dural fistulas. One (case 4) developed vertigo and decreased sensation in the left fifth nerve distribution secondary to a brainstem stroke. The second (case 7) developed ataxia and writing difficulties secondary to a large cerebellar stroke in the

TABLE 1: Summary of Dural Arteriovenous Fistulas Involving the Deep Venous System

Case No.	Age	Gender	Symptoms/ Presentation	Location	Feeding Arteries	Treatment		Follow-up (months)
						Preop	Postop	
1	48	M	Headache/SAH	Straight sinus	L & R middle meningeal & vertebral	IBCA	IBCA	16
2	71	M	Headache/SAH	Straight sinus	L & R occipital & vertebral	IBCA	IBCA	21
3	46	M	Headache/IVH	Vein of Galen	L & R middle meningeal & vertebral	PVA	Ligation & IBCA	18
4	42	M	Vertigo/brainstem stroke	Vein of Galen	L & R occipital, ascending pharyngeal, middle meningeal, meningohypophyseal trunk, anterior cerebral	PVA	IBCA	16
5	40	M	Ataxia/cerebellar hematoma	Straight sinus	L & R occipital	IBCA	Two ligations & IBCA	16[a]
6	55	M	Headache/bruit	Vein of Galen	L & R middle meningeal, occipital, vertebral, meningohypophyseal trunk, posterior cerebral	PVA	IBCA	8
7	63	M	Ataxia/cerebellar stroke	Straight sinus	R vertebral, R meningohypophyseal trunk	None	Surgical excision	9
8	30	M	Headache/ venous hypertension	Posterior falx	L & R occipital, L & R middle meningeal, L & R superficial temporal	PVA	Surgical excision	7

Note.—Complications occurred in only two patients (cases 3 and 4); both had hydrocephalus. Preop = preoperative; postop = postoperative; SAH = subarachnoid hemorrhage; IVH = intraventricular hemorrhage; L = left; R = right; IBCA = isobutyl 2-cyanoacrylate; PVA = polyvinyl alcohol (sponge particles).

[a] A 95% reduction occurred in this patient; all other patients were cured.

distribution of arterialized draining veins. Two patients (cases 6 and 8) had severe chronic headaches related to their fistulas. Case 6 also had a loud bruit.

Seven patients underwent preoperative embolization of the external carotid feeding vessels. After angiographic delineation of all the feeding pedicles, a 3.2-French Tracker catheter* was advanced into the distal feeding vessels. Digital subtraction angiography (DSA) was performed to confirm the supply to the fistula. Isobutyl 2-cyanoacrylate (IBCA, bucrylate)† mixed with iophendylate (Pantopaque)‡ was injected by the push technique in three patients (cases 1, 2, and 5). IBCA is no longer commercially supplied by Ethicon. Other cyanoacrylates with similar properties are commercially available or are being studied as investigational agents. In the push technique, the catheter is perfused with a sterile glucose (D_5H_2O) solution, and the IBCA/Pantopaque is then placed into the catheter and "pushed" into the desired location by a syringe filled with D_5H_2O. When the desired location of the mixture is noted on real-time DSA, the injection is terminated. Polyvinyl alcohol sponge particles, ranging from 200 to 700 μm, were used in the remaining cases (cases 3, 4, 6, and 8). In six of seven cases embolized, there was persistent supply from internal carotid or vertebral dural branches, which could not be selectively catheterized. After preoperative embolization, six patients subsequently underwent direct intraoperative placement of IBCA into the fistula site in the following fashion: The involved dural structure was surgically exposed and the major arterial feeders were ligated or coagulated. The straight sinus (three cases) or draining vein (three cases) was surgically clipped or ligated. A 19- or 21-gauge needle was then placed into the involved dural sinus proximal to the surgical occlusion. An intraoperative DSA image was then obtained to delineate the remaining flow. A mixture of IBCA and Pantopaque was injected into the fistula by the push technique monitored by real-time DSA. An angiogram was then obtained through the same needle, if the needle was patent. Additional injections were done until the fistula was closed or the needle occluded by IBCA. If persistent arterialized veins were observed, another needle puncture was performed and the sequence repeated until obliteration of the fistula was accomplished. When arterialized venous flow was no longer observed, the needles were removed. The clips and/or ligatures were left in place. Follow-up angiograms were obtained 3–14 days after the procedure.

The remaining two patients had surgical interruption of their fistulas without intraoperative placement of IBCA. Preoperative embolization of the external carotid supply with polyvinyl alcohol foam was performed in one patient; the remaining patient (case 7) had no external supply.

In two patients (cases 3 and 5) treated initially with surgical interruption of the feeding arteries, follow-up angiograms showed residual fistula flow. These patients subsequently underwent intraoperative embolization procedures, two in case 5 and one in case 3. Follow-up angiograms were obtained 5 days to 8 months after embolization.

Results

Four patients (cases 1, 2, 4, and 6) underwent preoperative embolization of the external carotid feeders, followed by intraoperative placement of liquid adhesives into the fistula site. Complete cure was documented in all four patients on follow-up angiograms.

* Target Therapeutics, Inc., Mountain View, CA.
† Ethicon, Inc., Somerville, NJ.
‡ Lafayette Pharmacal, Inc., Lafayette, IN.

Four patients underwent surgical excision (cases 7 and 8) or isolation (cases 3 and 5) of the fistula site. Surgical interruption of the fistula feeders was performed by direct visualization of the fistula site and interruption of all visible feeders by bipolar coagulation or ligation. Complete angiographic obliteration was achieved in the two patients (cases 7 and 8) treated by surgical excision, and subtotal occlusion in the two treated by surgical isolation. These two patients (cases 3 and 5) underwent subsequent intraoperative embolization procedures, with complete cure in one and a 95% decrease in fistula flow in the other. Subsequent to the procedures, none of the eight patients had symptoms related to the fistula. The clinical follow-up period ranged from 7 to 21 months (mean, 14).

Follow-up angiograms were obtained in all eight patients 3 days to 8 months after the procedure. In seven patients complete angiographic obliteration was observed; in the remaining patient (case 5) 95% obliteration was noted.

Two complications were related to the procedures. In cases 3 and 4, both patients had fistulas involving the vein of Galen. After intraoperative placement of liquid adhesives into the fistula site, both developed decreased mentation and hydrocephalus. Both patients were treated successfully with ventriculoperitoneal shunting, with improvement in their symptoms. Case 3 developed cognitive dysfunction related to a frontal hemorrhage after surgical interruption of feeding vessels and intraoperative embolization.

Representative Case Reports

Case 2

A 71-year-old man presented with subarachnoid hemorrhage. Angiography (Figs. 1A and 1B) demonstrated a dural AVF at the junction of the superior vermian vein and the straight sinus (Fig. 1A). The connection between the fistula and the straight sinus was thrombosed, and venous drainage was to an inferior vermian vein and then to cerebellar hemispheric collaterals. The occipital arterial supply was embolized preoperatively. Surgical exposure confirmed the fistula location and revealed several freshly thrombosed hemispheric veins. The draining vein was ligated and punctured proximal to the ligature. A small injection of liquid adhesive was refluxed into the fistula site. A follow-up angiogram showed complete obliteration of the fistula (Fig. 1D).

Case 4

A 42-year-old man had vertigo and decreased sensation in the left fifth nerve distribution. MR revealed a brainstem stroke and a fistula involving the vein of Galen (Fig. 2A). Angiograms showed supply from both internal and external carotid and vertebral arteries (Figs. 2B–2E). The external carotid supply was preoperatively embolized with polyvinyl alcohol particles. The vein of Galen was surgically exposed and large feeding vessels were coagulated. A surgical clip was placed across the straight sinus and a 21-gauge needle placed into the vein of Galen. After angiographic confirmation of the location, a 0.5-ml mixture of 50% IBCA and 50% Pantopaque was injected into the sinus. Follow-up angiography (Figs. 2F–2H) demonstrated complete obliteration of the fistula.

Case 6

A 55-year-old man had severe headaches. Angiography disclosed dural supply from the vertebral (Fig. 3A), internal carotid (Figs. 3B and 3C), and external carotid (Figs. 3D and 3E) arteries. The occipital and middle meningeal arteries were embolized preoperatively with polyvinyl alcohol particles. Intraoperatively, the involved superior vermian vein was exposed, isolated, punctured, and filled with liquid adhesives. Follow-up angiography (Figs. 3F–3H) demonstrated complete cure.

Discussion

Dural AVFs involving the deep cerebral venous system are rare and pose difficult diagnostic and therapeutic problems. Symptoms may be life-threatening. The most common presenting symptoms in our series were headaches (63%), hemorrhage (50%), and stroke (25%). It is possible that the disease is more prevalent than has been reported but that only cases with severe symptoms are recognized clinically. Most of the patients in our series had deep venous outflow obstruction with diversion of flow into cortical veins. Several authors have noted a strong association between cortical venous drainage and severity of clinical symptoms [12, 13].

Dural fistulas are categorized most often by their location. They usually occur in the transverse and sigmoid sinuses, followed by the cavernous sinus, anterior cranial fossa, superior sagittal sinus, and tentorium [1, 3, 4, 12–14]. Venous drainage occasionally is diverted into the deep venous system. This diversion is recognized most often in fistulas of the transverse and sigmoid sinuses. Few reports have demonstrated fistulas in the walls of the deep venous system with diversion to superficial cortical veins [15, 16].

In the vein of Galen location, differentiation between arteriovenous malformations of the posterior thalamus and quadrigeminal plate and acquired dural AVFs can be made on the basis of location of the nidus. Vein of Galen aneurysms and dural AVFs may be distinguished by the fact that the primary blood supply to dural AVFs is dural and the primary blood supply to vein of Galen aneurysms is via parenchymal branches [17, 18]. At times, distinction can be difficult. Both can have severe venous occlusive changes; some investigators have suggested that vein of Galen aneurysms develop secondary to intrauterine straight sinus thrombosis and represent a type of dural AVF [19]. Vein of Galen aneurysms generally present in the newborn or neonatal age group with congestive failure [17, 18]. Only rarely does an adult present with hydrocephalus [17]. The older age of presentation in our series, common for dural AVFs, suggests an acquired etiology.

The deep location and multiplicity of arterial feeders complicate treatment. This is reflected in the fact that transvascular embolization resulted in fistula obliteration in only one of seven patients in our series. Preoperative embolization is a useful adjunct before surgical exposure and embolization to reduce the vascularity of the overlying structures and reduce blood loss during exposure. Dural branches arising from the posterior circulation (posterior meningeal branch of the vertebral artery, artery of Davidoff and Schecter arising

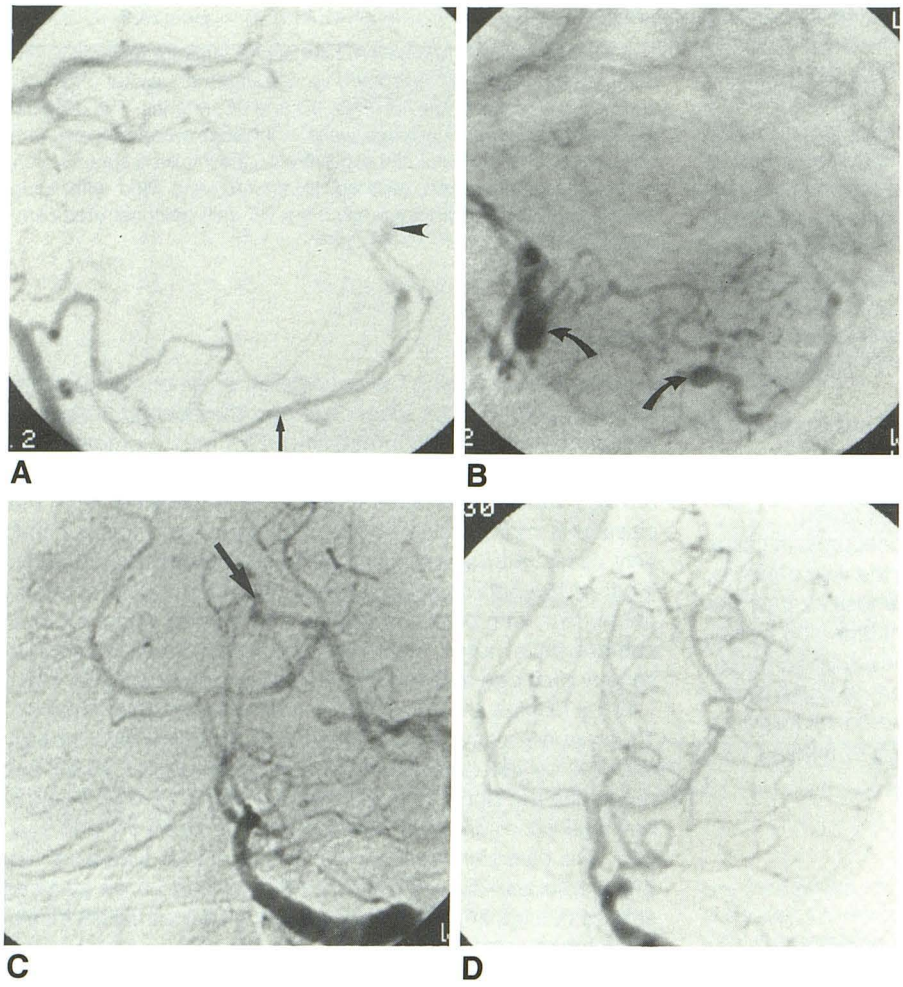

Fig. 1.—Case 2.

***A*, Left vertebral artery injection, lateral view, shows dural fistula (*arrowhead*) supplied by posterior meningeal branch (*arrow*).**

***B*, Capillary phase shows venous drainage to inferior vermian vein and then to hemispheric veins with multiple varices (*arrows*).**

***C*, Left vertebral artery injection, Towne's view, shows midline site of fistula (*arrow*) with cortical drainage to left cerebellar hemispheric vein.**

***D*, Left vertebral artery injection, Towne's view, after intraoperative obliteration of dural arteriovenous fistula with liquid adhesives.**

from the peduncular segment of the posterior cerebral artery, and superior cerebellar tentorial branches) and the cavernous internal carotid artery (meningohypophyseal trunk branches) are difficult to catheterize and, because of the risk of reflux of emboli, dangerous to embolize. Because of the extreme tortuosity and length of the external carotid arteries feeding the nidus, accurate deposition of embolic material into the nidus is difficult. Surgical management of dural AVFs in other locations has included isolation of the fistula, placement of embolic material into the feeding artery [7–9] or the involved sinus or vein [10, 11], or direct surgical excision [5]. Grisoli et al. [20] have recently reported good results with surgical excision and/or ligation of the draining vein in four patients with dural arteriovenous malformations involving the tentorium. Owing to the plethora of venous drainage pathways arising from the vein of Galen, it was believed that occlusion of venous drainage pathways alone would not suffice as a sole treatment and could increase the risk of hemorrhage. Surgical isolation can be difficult in this deep location, and persistence of the fistula occurred in two patients initially treated in this fashion. If the draining veins are interrupted without obliteration of the arterial supply, increased pressure can result, predisposing to hemorrhage.

Surgical excision of the involved dural sinus can be tolerated if arterial pressure from the dural AVF has resulted in the development of adequate venous collaterals. Similarly, occlusion of the venous sinus involved with the dural AVF by embolic agents can produce closure of the fistula. Mickle and Quisling [21] have used this principle to treat vein of Galen aneurysms by placing coils into involved venous aneurysms. We, too, have used this technique to treat dural fistulas involving the cavernous, transverse, and sigmoid sinuses [10, 11]. Because of the difficulties in surgical resection of the involved dural sinus in this location, we have chosen to place liquid adhesives directly into the involved sinus intraoperatively to cause fistula obliteration. Before this, the venous drainage is occluded to promote retrograde flow of the embolic material from the venous sinus or draining vein into the fistula nidus. The deep midline location of fistulas involving the vein of Galen potentially allows draining arterialized blood or embolic material into its major tributaries including the internal cerebral veins, internal occipital veins, posterior pericallosal veins, inferior sagittal sinus, and veins draining the posterior mesencephalon and superior cerebellum. If embolic material were to flow into draining veins rather than the fistula alone, then aggravation of venous hypertension, increased

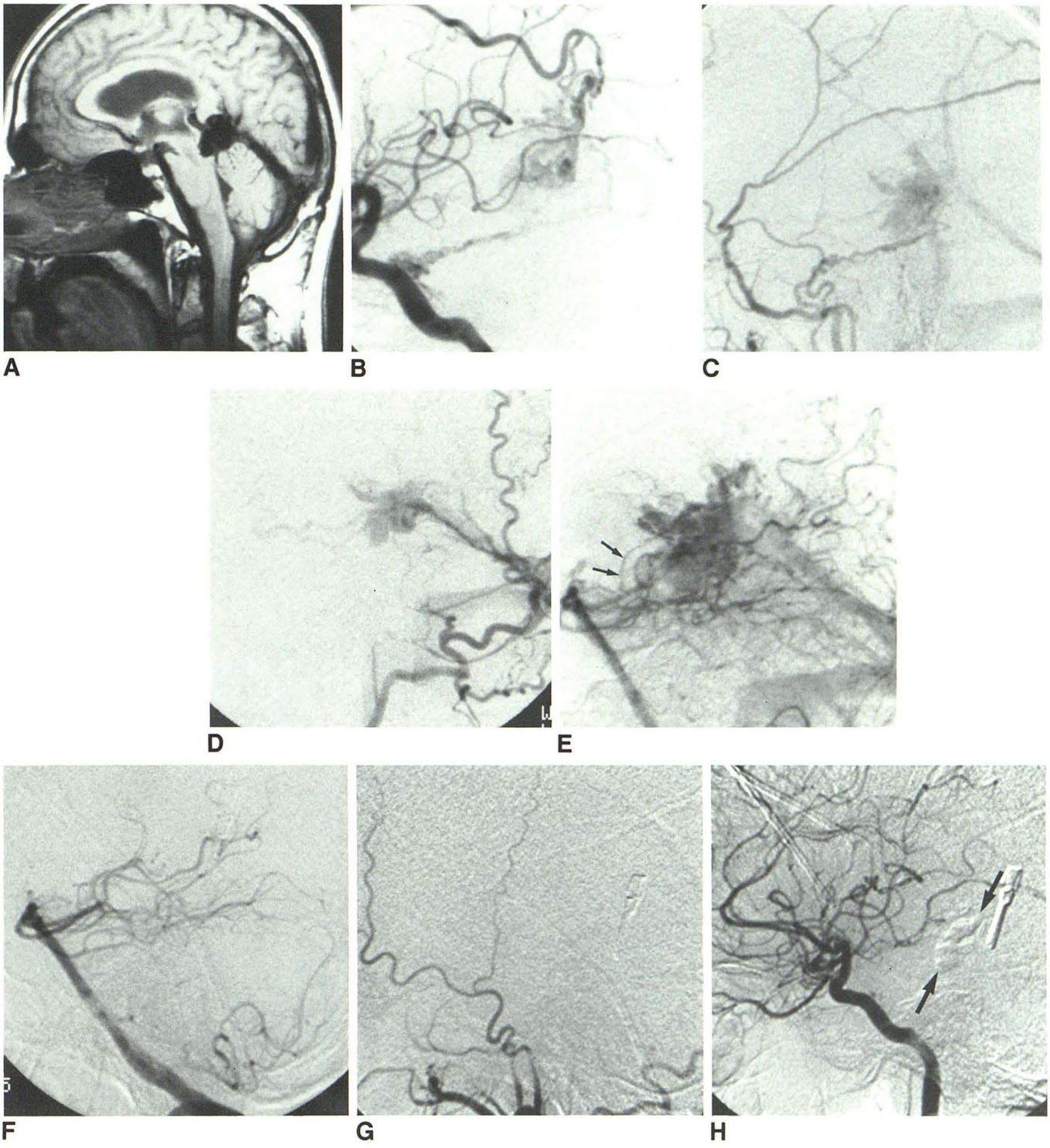

Fig. 2.—Case 4.

A, Sagittal midline T1-weighted MR scan shows prominent signal void behind tectum.

B, Right internal carotid injection, lateral view, shows dural fistula involving vein of Galen supplied by meningohypophyseal trunk and distal pericallosal arteries. Similar supply was noted from left internal carotid.

C and *D*, Left internal maxillary (*C*) and occipital (*D*) injections show supply from middle meningeal and transmastoid perforator branches from occipital artery.

E, Left vertebral injection, lateral view, shows supply to fistula by artery of Davidoff and Schecter (*arrows*) (dural branches from peduncular segment of posterior cerebral artery).

F, Left vertebral artery injection, lateral view, after intraoperative embolization, shows obliteration of supply.

G, Left external carotid angiogram, lateral view, shows obliteration of fistula.

H, Right internal carotid injection after embolization. Liquid adhesive is located within thrombosed fistula (*arrows*).

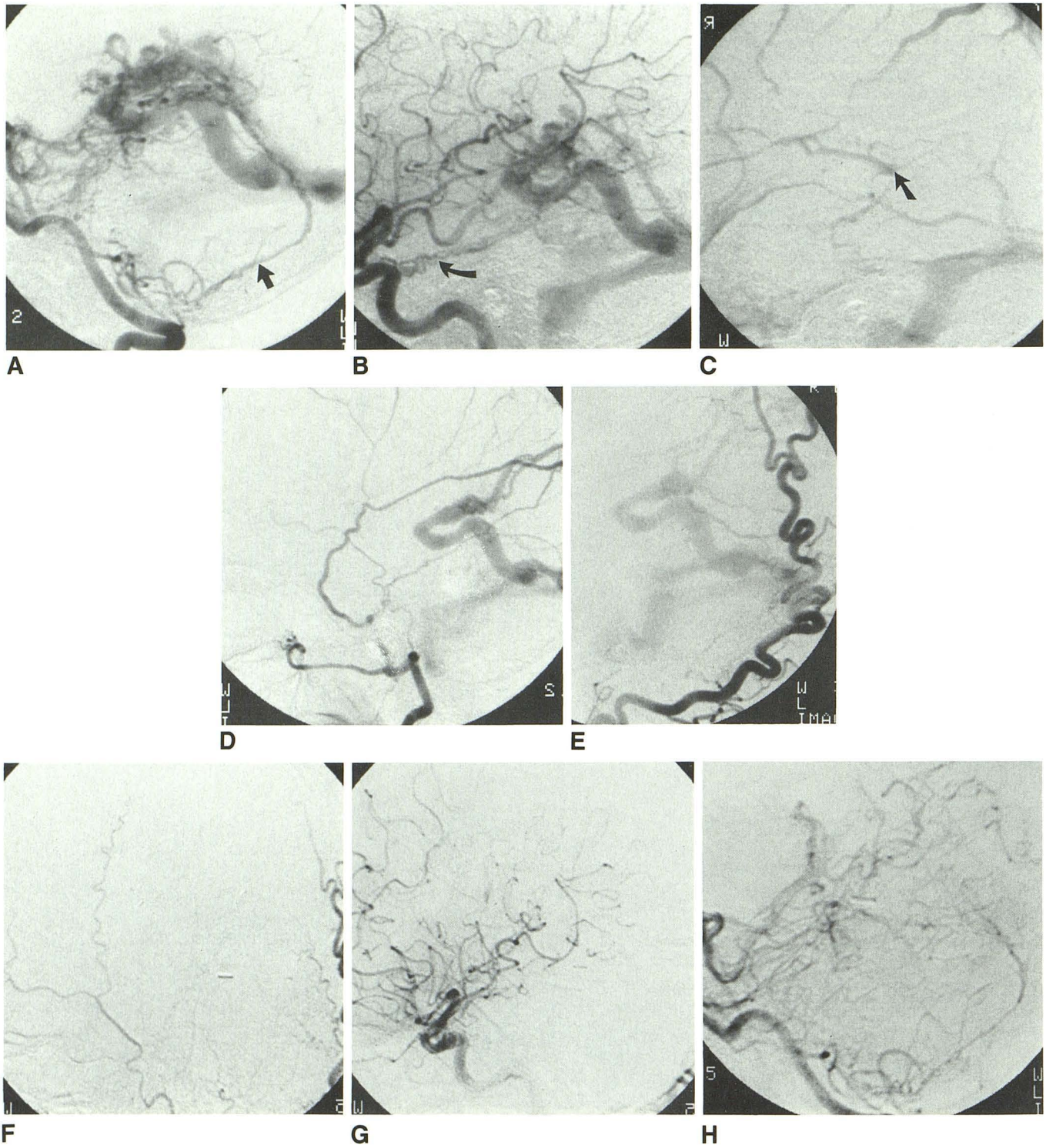

Fig. 3.—Case 6.

A, Left vertebral artery injection, lateral view, shows midline dural fistula supplied by posterior meningeal artery (*arrow*) and posterior cerebral branches.

B and *C*, Right internal carotid angiograms, lateral views, early (*B*) and late (*C*) phases, show supply from meningohypophyseal trunk (*curved arrow*) and posterior cerebral arteries. Late phase shows occlusion of internal cerebral vein (*straight arrow*) at junction with vein of Galen.

D, Right internal maxillary injection, lateral view, shows supply from middle meningeal arteries. Similar supply arose from opposite side. This was embolized with polyvinyl alcohol particles before intraoperative embolization.

E, Right occipital artery injection, lateral view, shows supply from transmastoid perforators to the same fistula.

F, Right external carotid angiogram after embolization shows occlusion of supply to fistula.

G, Right internal carotid injection, lateral view, shows obliteration of supply.

H, Left vertebral artery injection, lateral projection, confirms fistula obliteration.

intracranial pressure, or hemorrhage could occur. High-quality real-time DSA is essential for accurate deposition of the embolic material.

By using preoperative embolization to reduce the supply and pressure within the nidus, followed by direct intraoperative placement of liquid adhesives, we have achieved complete cures in five of six patients treated and a marked decrease in flow in the remaining patient. Hydrocephalus developed after intraoperative embolization of the two fistulas in the vein of Galen region. This was probably related to mechanical compression or inflammation obstructing the nearby aqueduct. Hydrocephalus can result from a dural fistula because of impairment of CSF absorption; however, it was not present in our two patients before treatment. Both patients were treated effectively with ventriculoperitoneal shunting.

In conclusion, dural AVFs involving the deep venous system are rare, usually present with headaches or hemorrhage, and can be treated effectively by a combination of neuroradiologic and surgical intervention.

REFERENCES

1. Newton TH, Cronqvist S. Involvement of the dural arteries in intracranial arteriovenous malformations. *Radiology* **1969**;93:1071–1078
2. Kuhner A, Krastel A, Stoll W. Arteriovenous malformations of the transverse dural sinus. *J Neurosurg* **1976**;45:12–19
3. Kosnik EJ, Hunt WE, Miller CA. Dural arteriovenous malformations. *J Neurosurg* **1974**;40:322–329
4. Newton TH, Hoyt WF. Dural arteriovenous shunts in the region of the cavernous sinus. *Neuroradiology* **1970**;1:71–81
5. Sundt TM, Piepgras DG. The surgical approach to arteriovenous malformations of the lateral and sigmoid dural sinuses. *J Neurosurg* **1983**;59:32–39
6. Higashida RT, Hieshima GB, Halbach V, Bentson JR, Goto K. Closure of carotid cavernous sinus fistulae by external compression of the carotid artery and jugular vein. *Acta Radiol [Suppl] (Stockh)* **1986**;369:580–583
7. Lasjaunias P, Halimi P, Lepez-Ibor L, Sichez JP, Hurth M, Tribolet ND. Traitement endovasculaire des malformation vasculares durales (MVD) pures spontanees. *Neurochirurgie* **1983**;30:207–223
8. Halbach VV, Higashida RT, Hieshima GB, Reicher M, Norman D, Newton TH. Dural fistulas involving the cavernous sinus: results of treatment in 30 patients. *Radiology* **1987**;163:437–443
9. Halbach VV, Higashida RT, Hieshima GB, Goto KS, Norman D, Newton TH. Dural fistulas involving the transverse and sigmoid sinuses. Results of treatment in 28 patients. *Radiology* **1987**;163:443–447
10. Halbach VV, Higashida RT, Hieshima GB, Hardin CW, Pribram H. Transvenous embolization of dural fistulas involving the cavernous sinus. *AJNR* **1988**;9(7):741–749
11. Halbach VV, Higashida RT, Hieshima GB, Mehringer CM, Hardin CW. Transvenous embolization of dural fistulas involving the transverse and sigmoid sinuses. *AJNR* **1989**;10:385–392
12. Houser OW, Cambell JK, Cambell RJ, Sundt TM. Arteriovenous malformations affecting the transverse dural venous sinus: an acquired lesion. *Mayo Clinic Proc* **1979**;54:651–661
13. Lasjaunias P, Chiu M, Ter Brugge K, Tolia A, Hurth M, Bernstein M. Neurological manifestations of intracranial dural arteriovenous malformations. *J Neurosurg* **1986**;64:724–730
14. Vinuela F, Fox AJ, Pelz DM, Drake CG. Unusual clinical manifestations of dural arteriovenous malformations. *J Neurosurg* **1986**;64:554–558
15. O'Reilly GV, Hammerschlag SB, Ronthal M. Aneurysmal dilation of galenic venous system caused by a dural arteriovenous malformation. *J Comput Assist Tomogr* **1981**;5:899–903
16. Mayberg MR, Zimmerman C. Vein of Galen aneurysm associated with dural AVM and straight sinus thrombosis. *J Neurosurg* **1988**;68:288–291
17. Hoffman HJ, Chuang S, Hendrick EB, Humphreys NP. Aneurysms of the vein of Galen. Experience at the Hospital for Sick Children, Toronto. *J Neurosurg* **1982**;57:316–322
18. Martelli A, Scotti G, Harwood-Nash DL, Fitz CR, Chuang SH. Aneurysms of the vein of Galen in children: CT and angiographic correlations. *Neuroradiology* **1980**;20:123–133
19. Lasjaunias P, Ter Brugge K, Lopez-Ibor L, et al. The role of dural anomalies in vein of Galen aneurysms: report of six cases and review of literature. *AJNR* **1987**;8:185–192
20. Grisoli F, Vincentelli F, Fuchs S, et al. Surgical treatment of tentorial arteriovenous malformations draining into the subarachnoid space. *J Neurosurg* **1984**;60:1059–1066
21. Mickle JP, Quisling RG. The transtorcular embolization of vein of Galen aneurysms. *J Neurosurg* **1986**;64:731–735