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High-Risk Dural Arteriovenous Fistulae of the Transverse and Sigmoid Sinuses

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Dural arteriovenous fistulae of the transverse and sigmoid sinuses are highly variable in symptomatology and prognosis. However, we have identified a subgroup of patients who have a high risk of hemorrhage and dementia due to severe venous overload caused by high arterial flow into the fistulae and by occlusive changes of the transverse and sigmoid sinuses. Three representative cases selected from 31 patients with dural arteriovenous fistulae of the transverse and sigmoid sinuses are presented, and 45 reported similar cases are reviewed to discuss pathophysiology and problems encountered during treatment.

Dural arteriovenous fistulae (AVF) often disappear spontaneously [1, 2] and tend to be regarded as benign. However, we have reviewed our cases of dural AVF of the transverse and sigmoid sinuses and have found a subgroup of patients who have a high risk of hemorrhage and dementia. These serious complications were related to severe venous overload caused by high arterial flow into the fistulae and occlusive changes involving the transverse and sigmoid sinuses. Early diagnosis and treatment is mandatory in these dural AVF to improve the prognosis; however, the choice of treatment is still controversial [3–16]. We present three representative cases selected from 31 patients with dural AVFs of the transverse and sigmoid sinuses to demonstrate various types of intracranial hemorrhage and/or dementia and to describe both the pathophysiological processes involved and the prognosis after treatment.

Representative Case Reports

Case 1

A 75-year-old woman was admitted to our hospital after having a convulsion, followed by right hemiparesis. CT showed a small subcortical hemorrhage in the left parietal region (Fig. 1B). Also noted were numerous vermiform contrast-enhancing structures over the surface of the brain and diffuse decrease in deep-white-matter density (Figs. 1A and 1B). Common carotid angiography showed a dural AVF involving the left transverse and sigmoid sinuses (Figs. 1C–1F). The left occipital artery was the main feeding artery, but there was also a considerable contribution from the posterior branch of the middle meningeal artery. The left sigmoid sinus was occluded just above the jugular foramen, and the medial part of the right transverse sinus was severely narrowed. This resulted in prominent retrograde drainage into the superior sagittal sinus and then into the engorged cortical veins, which drained inferiorly to the sylvian veins and then to the cavernous sinus bilaterally. Progressive dementia followed the hemorrhage. One month later, the patient had a second seizure. CT showed a subcortical hematoma in the left frontal lobe.

Treatment was refused by the patient's family, and 5 months later she became comatose after a seizure. CT showed multiple subcortical hematomas in both cerebral hemispheres (Figs. 1G and 1H). Decrease in deep-white-matter density became more prominent. The patient went into a vegetative state and died 6 months later.

Fig. 1.—Case 1.

A and B, Contrast CT scans on admission showed small subcortical hematoma in left parietal region, vermiform contrast enhancement over both cerebral hemispheres, and diffuse decrease in density of cerebral white matter with obliteration of cortical sulci.
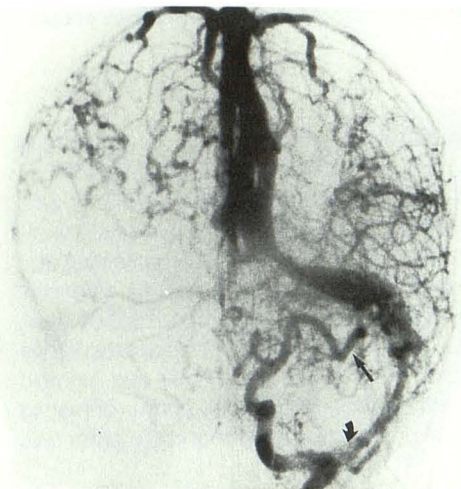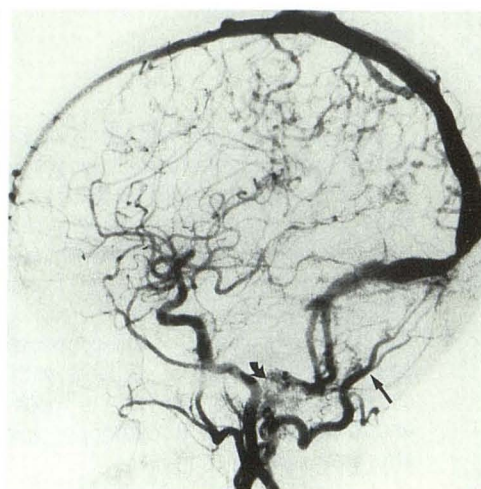
C and D, Left common carotid angiography, anteroposterior/lateral mid arterial phase. Dural arteriovenous fistula of left transverse and sigmoid sinuses fed by the occipital artery (straight arrow). Distal transverse sinus was stenotic and sigmoid sinus was occluded just above jugular foramen on left (curved arrow).
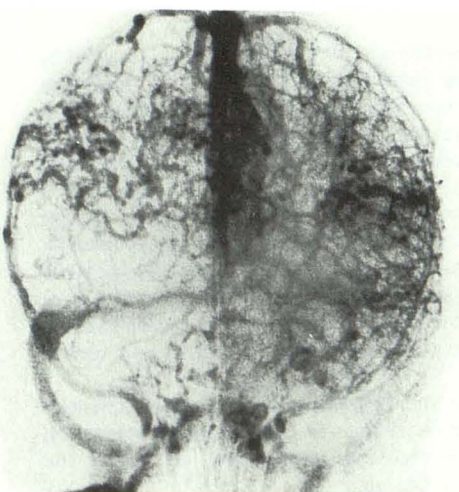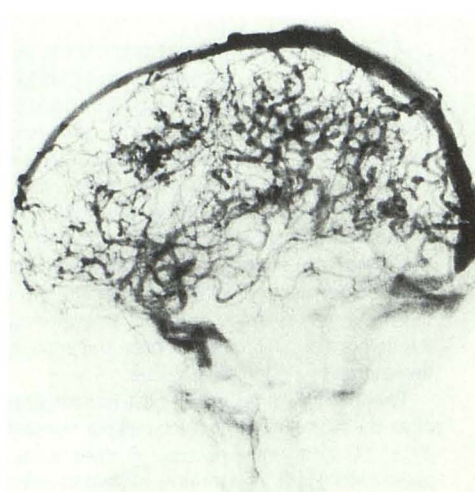
E and F, Anteroposterior late arterial, early venous phase. Medial part of right transverse sinus was almost occluded. Retrograde drainage into superior sagittal sinus and engorged cerebral veins of both cerebral hemispheres became prominent.

G and *H*, Follow-up CT scan 5 months later revealed multiple subcortical hematomas in both cerebral hemispheres and further decrease in white-matter density.

I, Schematic drawing of hemodynamic abnormalities of case 1 (*star* shows site of arteriovenous fistula; *arrows* show directions of venous drainage; *tear drops* show site of hemorrhage).

Case 2

A 62-year-old woman had complained of bilateral tinnitus for a year, and progressive dementia was first noted 3 months prior to admission. The patient was admitted to our hospital when she devel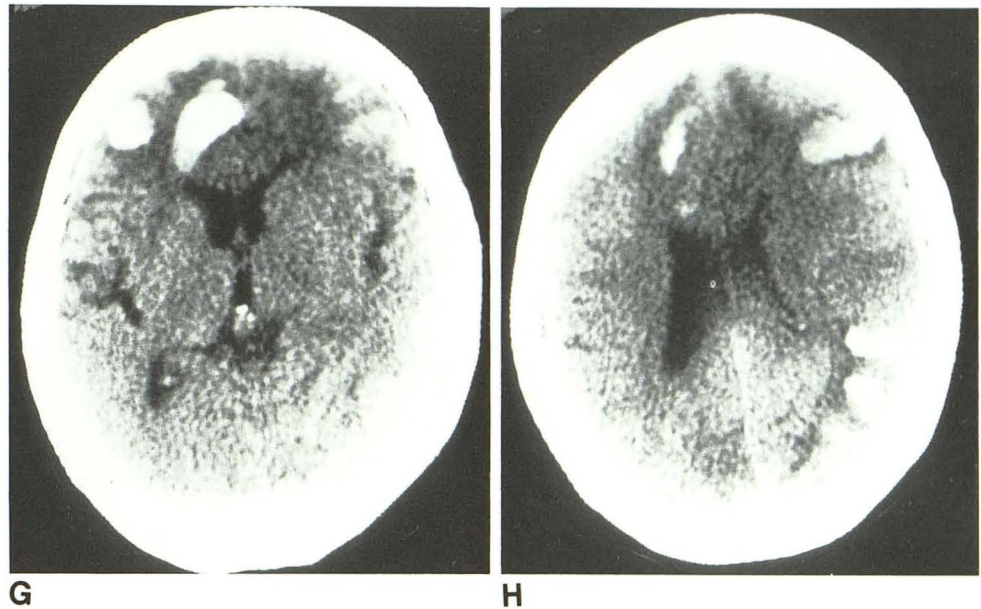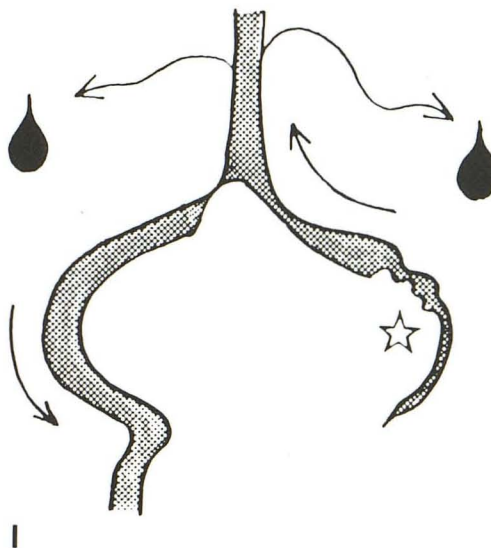oped severe headache and vomiting. CT showed subarachnoid hemorrhage most prominent in the right sylvian fissure (Fig. 2A). Angiography disclosed a dural AVF involving both transverse sinuses and confluence of the sinuses (Figs. 2B–2D). There was occlusion of the left transverse and sigmoid sinuses and irregular stenosis of the right transverse sinus. The main feeding arteries were the dural branches of both occipital arteries. An additional blood supply came from both middle meningeal arteries, the posterior meningeal artery of the left vertebral artery, and the marginal tentorial branches of both internal carotid arteries. There was prominent retrograde drainage into the superior sagittal sinus, straight sinus, engorged cortical veins bilaterally, and the basal vein.

Embolization of the external carotid branches was performed with polyvinyl alcohol particles (150–249 μm). The patient's mental state and EEG findings improved markedly within a couple of days after embolization. However, progressive dementia and deterioration of the EEG pattern became prominent again 3 weeks after embolization, necessitating neuroradiologic reevaluation. CT revealed a prominent, diffuse decrease in deep-white-matter density and numerous enhancing superficial vermiform structures representing engorged cortical veins (Fig. 2E). The patient underwent repeat embolization followed by craniotomy and dural incision to isolate the dural AVF.

The patient showed a gradual and persistent improvement of dementia. Pathologic examination showed that the lumen of the excised left transverse sinus was almost completely replaced by dural AVF. There was abnormal low density of the deep white matter, which was considered to be white-matter edema caused by venous congestion. This became less prominent 19 months after embolization and surgery (Fig. 2F).
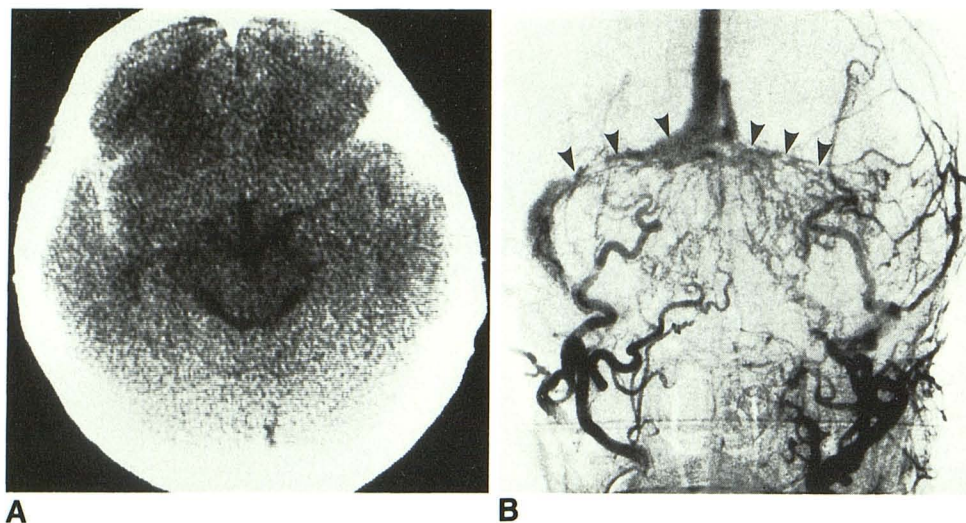
A

B

Fig. 2.—Case 2.

A, CT scan on admission showed subarachnoid hemorrhage in right sylvian fissure.

B, Towne's view of compound right and left external carotid angiography. Dural arteriovenous fistula in bilateral transverse sinuses (*arrowheads*) and confluence of the sinuses fed by bilateral occipital arteries and middle meningeal arteries. Severe stenotic change of right transverse sinus and occlusion of left transverse and sigmoid sinuses caused retrograde filling of superior sagittal and straight sinuses.

C

D
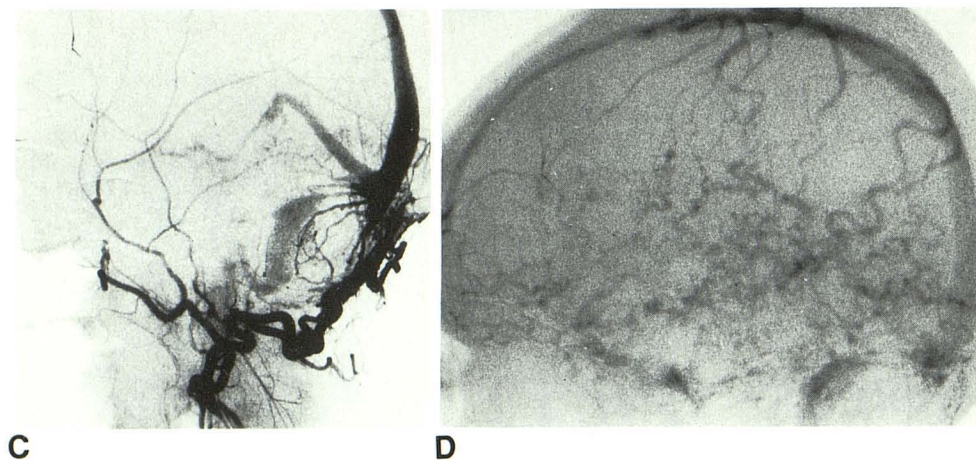
C, Lateral view of right external carotid angiography. Dural arteriovenous fistula was fed by middle meningeal and occipital arteries, which were markedly dilated. Retrograde drainage into superior sagittal and straight sinuses and basal vein of Rosenthal was noted.

D, Lateral view of the late phase of left external carotid angiography. Note marked diffuse engorgement of cerebral veins, which drained anteroinferiorly into cavernous sinus and pterygoid plexus.

Case 3

A 75-year-old woman was admitted to our hospital for recurrent vomiting and vertigo. She had had a subarachnoid hemorrhage 4 years earlier. CT taken on the day of admission showed a subarachnoid hemorrhage in the left sylvian fissure and the posterior fossa, as well as cerebellar hemorrhage (Fig. 3A). Her level of consciousness deteriorated to a semicomatose state 4 days after admission. A repeat CT showed an increase in the amount of cerebellar hemorrhage (Fig. 3B) with mass effect and associated obstructive hydrocephalus. External carotid angiography (Figs. 3C and 3D) disclosed a dural AVF involving the left transverse sinus. The left transverse sinus was isolated because the sigmoid sinus was occluded and the medial part of the transverse sinus was absent. This resulted in prominent retrograde drainage into the engorged cortical veins over the posterior temporal lobe and the left cerebellar hemisphere. These cortical veins then drained into the superior sagittal, occipital, and sphenoparietal sinuses. Surgical evacuation of the cerebellar hematoma was done immediately after embolization of the feeding arteries with polyvinyl alcohol particles. The patient did not need a blood transfusion, and there was a marked improvement in her state of consciousness. Curative surgery for the dural AVF could not be done owing to cardiopulmonary disease.

Three months later the patient became comatose again. CT showed subcortical hematomas in the left posterior temporal lobe, the putamen (Fig. 3E), and the left cerebellar hemisphere. These were most likely caused by recanalization of the dural AVF.

Discussion

Dural AVF tends to be regarded as a benign disease; however, those fistulae occurring in the transverse and sigmoid sinuses are highly variable in symptomatology and prognosis [3, 10, 11]. Some disappear spontaneously [1, 2], while others cause severe neurologic deficits or even death. We reviewed our file of 31 cases of dural AVFs of the sigmoid and transverse sinuses and identified a subgroup of patients who have a high risk of hemorrhage and dementia. Details of those 31 cases, with emphasis on treatment results [16], were reported elsewhere by one of the authors.

Eleven of our cases had intracranial hemorrhage. Some regard drainage of the dural AVF into the cortical vein as the major cause of intracranial hemorrhage [3, 7–11, 17]. We

E, Follow-up CT scan with contrast 3 months later showed prominent decrease in white-matter density and vermiform contrast enhancement.

F, Follow-up CT scan with contrast 19 months after embolization and surgery. Note decreased prominence of low-density white-matter lesion and vermiform contrast enhancement, and increased prominence of cerebral sulci.
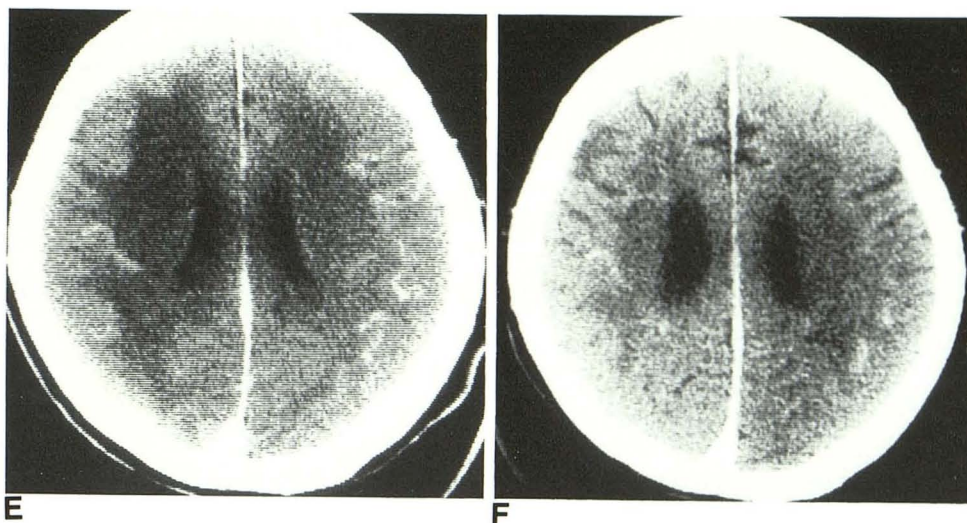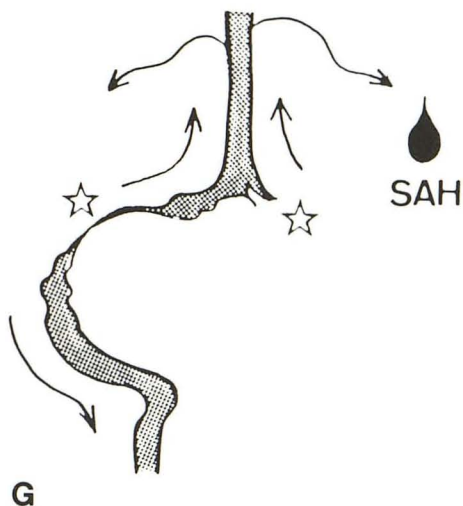

G, Schematic drawing of case 2 (SAH = subarachnoid hemorrhage).

believe that the degree of cerebral venous overload is of primary importance in understanding the signs and symptoms of dural AVF of the transverse and sigmoid sinuses. Figures 1I, 2G, and 3F are schematic drawings showing the dural AVF sites, the direction of venous drainage, and the hemorrhage sites in our representative cases.

In cases 1 and 2, the most outstanding angiographic findings were prominent retrograde filling of the superior sagittal sinus with diffuse engorgement of cortical veins. This was secondary to severe occlusive changes of the transverse and sigmoid sinuses and of high arterial flow into the dural AVF (Figs. 1I and 2G). Twelve of our 31 cases showed total occlusion of the affected dural sinuses. Diffuse decrease in deep-white-matter density seen on CT was considered brain edema [18, 19], which became less prominent after embolization and surgery in case 2. Diffuse venous overload with brain edema is regarded as the cause of dementia. Subcortical and subarachnoid hemorrhage, remote from the site of the dural AVF, probably resulted from the rupture of dilated pial

or medullary veins in these cases. Similar neurologic and angiographic changes were seen in eight more cases in our series.

In case 3, characteristic angiographic findings were localized venous overload of the temporal lobe and posterior fossa due to high-flow dural AVF of the isolated transverse sinus (Fig. 3F). There was drainage into the superior sagittal and straight sinuses via the engorged pial veins. Subcortical and subarachnoid hemorrhage near the lesions in these cases were thought to be due to rupture of pial or medullary veins by localized venous overload.

We reviewed 45 previously reported cases with good angiographic documentation of the dural AVF of the transverse and sigmoid sinuses that had caused relatively severe neurologic signs and symptoms [3, 5–8, 12, 17–18, 20–31]. The signs and symptoms are summarized in Table 1. There was a high frequency of focal neurologic signs, increased intracranial pressure, mental deterioration, and intracranial hemorrhage remote from the AVF in those 30 cases that had
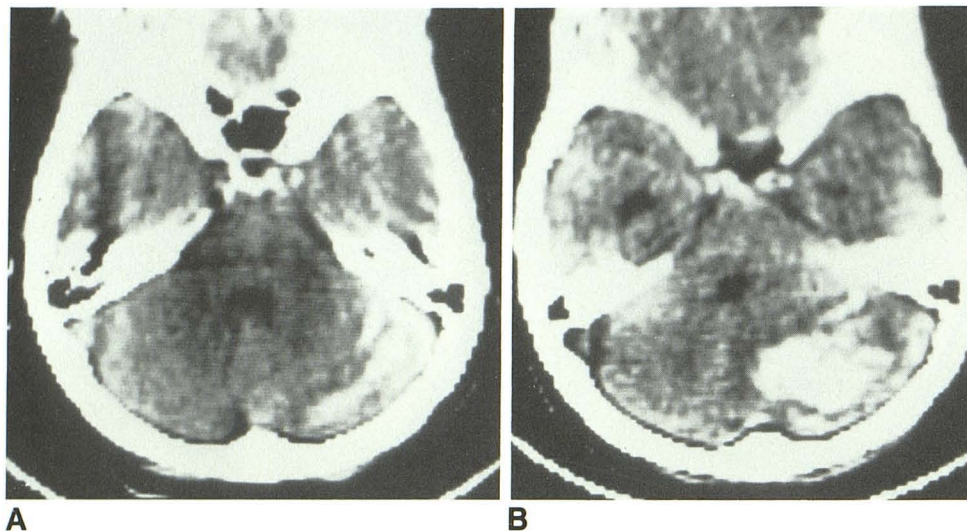
Fig. 3.—Case 3.
A, CT scan on admission showed cerebellar and subarachnoid hemorrhage in left posterior fossa.

B, CT scan 5 days later showed increase in amount of cerebellar hemorrhage.

C, Lateral arterial phase. Dural arteriovenous fistula in left transverse and sigmoid sinuses fed by occipital and middle meningeal arteries. Occlusion of sigmoid sinus (arrow) was noted.
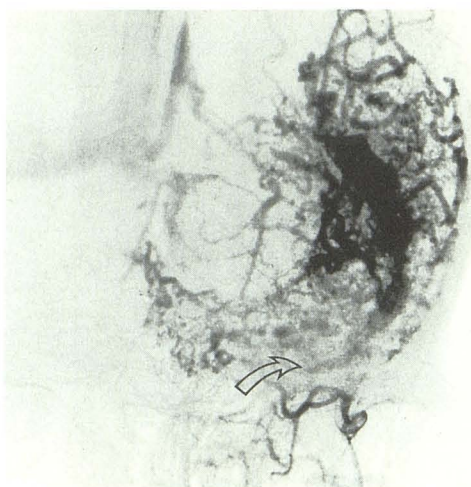
D, Anteroposterior venous phase. Left transverse sinus was isolated by occlusion of sigmoid sinus (arrow) and absence of medial part of transverse sinus. This resulted in retrograde filling of markedly engorged pial veins over cerebellar hemisphere and temporal and occipital lobes on left.
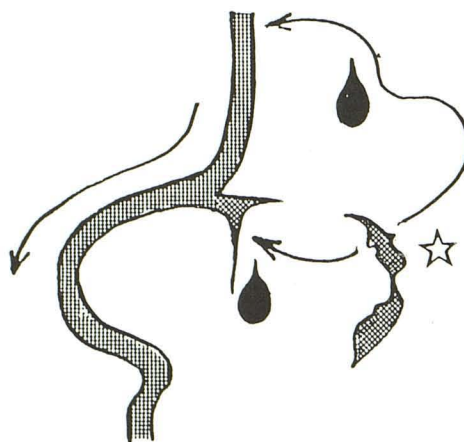
E, Follow-up CT scan 3 months later disclosed large subcortical hematoma with blood-fluid level in left temporal lobe and putaminal hemorrhage. Also shown was decrease in deep-white-matter density due to venous edema.
F, Schematic drawing of case 3.

TABLE 1: Signs and Symptoms of 45 Reported Cases of High-Flow Dural AVF[a] of the Transverse and Sigmoid Sinuses

	Group No.		
	1 (No. of Cases)	2A (No. of Cases)	2B (No. of Cases)
Signs and symptoms	30	2	13
Focal neurologic deficit	16	2	4
Papilledema	14	0	3
Bruit	11	0	10
Mental deterioration	8	0	2
Headache	8	1	4
Hemorrhage	5	0	4
Subarachnoid hemorrhage	3	0	2
Intracerebral hemorrhage	2	0	1
Subdural hematoma	0	0	1
Visual disturbance	7	1	1
Epilepsy	4	2	0

Note.—Group 1 = Cases with occlusion of the affected dural venous sinuses, reflux into the sagittal sinuses and Galenic system, and diffuse engorgement of cortical veins. Group 2 = Cases without occlusive changes in the sinuses and either (group 2A) with cortical venous drainage due to direct shunting to cortical veins or (group 2B) without cortical venous drainage but with signs and symptoms related to diffuse venous overload due to high arterial flow into the fistulae.

[a] AVF = arteriovenous fistulae.

occlusion of the dural venous sinuses, reflux into the sagittal sinuses and Galenic system, and diffuse engorgement of cortical veins (group 1 in Table 1).

On the other hand, cases without occlusive changes in the sinuses and with cortical venous drainage due to direct shunting to cortical veins (group 2A), had a tendency to have focal neurologic deficit close to the lesion. All five of the similar cases from our file had subcortical and subdural hemorrhages close to the lesion in the transverse sinus. Other cases without occlusive changes in the sinuses presented with signs and symptoms related to diffuse venous overload secondary to a very high arteriovenous shunt (group 2B).

Regarding treatment, we formerly used polyvinyl alcohol particles (150–249 μm) to embolize dural AVFs. However, as shown in case 2, early recanalization tends to occur shortly after embolization in cases treated with polyvinyl alcohol particles. A second embolization had to be performed shortly before excision of the dural AVF in that case. Blood transfusion was unnecessary during surgery in that case and no angiographic evidence of recurrence was seen postoperatively in any of those cases treated by a combination of embolization and surgery. Recently, we have been using cyanoacrylate to treat high-flow dural AVF, which can result in complete embolization, eliminating the supply from relatively minor feeding arteries. For superselective cannulation into the main feeding arteries, we have been using a combination of a steerable guidewire and a 3-French Teflon catheter or a 2.5-French Tracker catheter.* In cases where severe arteriosclerotic changes prevent superselective cannulation from a transfemoral or transcarotid approach, we have been doing intraoperative embolization guided by digital subtraction fluoroscopy. Further improvement in catheters, guidewires,

and embolic materials is needed to improve the success rate of treatment and to diminish complications.

Based on our experience with patients and our review of 45 cases from the literature, we believe that high-flow dural arteriovenous fistulae of the transverse and sigmoid sinuses accompanied by occlusive changes in these sinuses merit consideration as a separate group, since, if left untreated, they can cause severe venous overload. To improve the prognosis in these cases, such patients should be diagnosed early and have either curative embolization or embolization followed by surgery.

REFERENCES

1. Bitoh S, Sasaki S. Spontaneous cure of dural arteriovenous malformations in the posterior fossa. *Surg Neurol* **1979**;12:111–114
2. Magidson MA, Weinberg DE. Spontaneous closure of dural arteriovenous malformation. *Surg Neurol* **1976**;6:107–110
3. Obrador J, Suto M, Silvela J. Clinical symptoms of arteriovenous malformation of the transverse sigmoid sinus. *J Neurol Neurosurg Psychiatry* **1975**;38:436–451
4. Kosnik EJ, Hunt WE, Miller CA. Dural arteriovenous malformations. *J Neurosurg* **1974**;40:322–329
5. Ishijima T, Iwasa H, Miyagi K, Sato F. A case of a tentorial dural arteriovenous malformation. *Proceedings of the 4th conference of surgical treatment of stroke* **1979**:97–98
6. Shimizu K, Oku K, Go J, Hayakawa T, Ushio Y. Dural arteriovenous malformation—a case of total removal. *No Shinkei Geka* **1979**;7:257–263
7. Lasjaunias P, Halimi PH, Lopez-Ibor L, Sichez J, Hurth M, Tribolet N. Traitement endovasculaire des malformation vasculares durales (MVD) pures "spontanées": revue de 23 cas explorés et traités entre mai 1980 et octobre 1983. *Neurochirurgie* **1983**;30:207–223
8. Malik GM, Pearce JE, Ausman JI, Mehta B. Dural arteriovenous malformations and intracranial hemorrhage. *Neurosurgery* **1984**;15:332–339
9. Enker SH. Progression of a dural arteriovenous malformation resulting in an intracerebral hematoma: a case report. *Angiology* **1979**;30:198–204
10. Hunt WE. Dural arteriovenous malformations. In: Wilson CB, Stein BM, eds. *Intracranial arteriovenous malformations*. Baltimore: Williams & Wilkins, **1984**:222–233

* Tracker catheter is manufactured by Target Therapeutics, Los Angeles, CA 90025.

11. Djindjan R, Merland JJ. Superselective arteriography of the external carotid artery. New York: Springer Verlag, 1978

12. Bitoh S, Arita N, Fujiwara M, Oku Y, Tanada M. Dural arteriovenous malformations in the region of the transverse sigmoid sinus. Neurol Med Chir (Tokyo) 1979;19:1203–1211

13. Sano H, Kanno T, Katada K, Nagata J, Ishiyama N. Treatment of the dural AVM: embolization using Aron Alpha. Neurol Med Chir (Tokyo) 1980;20:845–851

14. Sundt TM, Piepgras DG. The surgical approach to arteriovenous malformations of the lateral and sigmoid sinuses. J Neurosurg 1983;59:32–39

15. Kühner A, Krastel A, Stoll W. Arteriovenous malformations of the transverse dural sinus. J Neurosurg 1976;45:12–19

16. Halbach VV, Higashida RT, Hieshima GB, Goto K, Norman D, Newton TH. Dural fistulas involving the transverse and sigmoid sinuses: results of treatment in 28 patients. Radiology 1987;163:443–447

17. Castaigne P, Bories J, Brunet P, Merland JJ, Meninger V. Le fistules artérioveineuses méningées pures a drainage veineux cortical. Rev Neurol (Paris) 1976;132:169–181

18. Miyasaka K, Takei H, Nomura M, et al. Computerized tomography findings in dural arteriovenous malformations. Report of three cases. J Neurosurg 1980;53:698–702

19. Chiras J, Bories J, Leger JM, Gaston A, Launay M. CT scan of dural arteriovenous fistulas. Neuroradiology 1982;23:185–194

20. Dichgans J, Gottshaldt M, Voigt K. Arteriovenöse Dura-Angiome am Sinus Transversus: klinische Symptome, characteristische arterielle Versorgung und häufige venöse Abflussstörungen. Zentralbl Neurochir 1972;33:1–18

21. Urdanibia JF, Silvela J, Suto M. Occipital dural arteriovenous malforma-

tions. Neuroradiology 1974;7:57–64

22. Debrun G, Chartres A. Infra and supratentorial arteriovenous malformations. A general review about 2 cases of spontaneous supratentorial malformations of the dura. Neuroradiology 1972;3:184–192

23. Handa J, Yoneda S, Handa H. Venous sinus occlusion with a dural arteriovenous malformation of the posterior fossa. Surg Neurol 1975;4:433–437

24. Takaku A, Sato T, Sakamoto T, Suzuki J. Dural arteriovenous malformations of the posterior fossa—clinical and angiographic analysis of six cases. No Shinkei Geka 1976;4:489–501

25. Houser OW, Cambell JK, Cambell RJ, Sundt TM. Arteriovenous malformation affecting the transverse dural venous sinus—an acquired lesion. Mayo Clin Proc 1979;54:651–661

26. Senarclens BD, Schar J, Wüthrich R. Von der arteria carotis externa ausgehende arteriovenöse Missbildungen der hinteren Schädelgrube. Schweiz Arch Neurol Neurochir Psychiatr 1972;110:69–82

27. Nakamoto K, Suzuki S. Occipital artery–transverse sinus communication. Nippon Igaku Hoshasen Gakkai Zasshi 1980;40:105–113

28. Mabuchi S, Nakagawa Y, Abe H, Kinomoto H, Tsuru M. Surgical treatment for three cases of dural arteriovenous malformation in the posterior fossa and its problems. No Shinkei Geka 1983;11:883–889

29. Amico G, Macchi L, Permeggiani A. Malformazioni vascolari epicraniche drenantisi nei seni durali postreriori. Minerva Neurochir 1970;14:324–329

30. Lamas E, Lobato RD, Esparza J, Escudero L. Dural posterior fossa AVM producing raised sagittal sinus pressure. J Neurosurg 1977;46:804–810

31. Nicola GC, Nizzoli V. Dural arteriovenous malformations of the posterior fossa. J Neurol Neurosurg Psychiatry 1968;31:514–519