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AJNR Am J Neuroradiol 1987, 8 (2) 185-192
http://www.ajnr.org/content/8/2/185

The Role of Dural Anomalies in Vein of Galen Aneurysms: Report of Six Cases and Review of the Literature

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It is proposed that the vein of Galen aneurysm represents a venous ectasia secondary to an increased flow (usually caused by a deep-seated arteriovenous shunt draining either directly into the vein of Galen aneurysm or into a tributary of the vein of Galen) associated with obstruction of a dural sinus distal to the aneurysm. The closer the venous obstruction is to the vein of Galen, the better the chances are of developing obstructive (noncommunicating) hydrocephalus and the more likely it is that the venous drainage from the rest of the brain will be unaffected. The farther the venous obstruction is from the vein of Galen aneurysm, the better the chances are of developing a communicating type of hydrocephalus. The development of cardiac failure is related to the magnitude of the arteriovenous shunt. Brain damage, seizures, and hemorrhage may be related to the retrograde venous engorgement, causing impaired drainage of the healthy brain. Careful attention should be paid to the venous drainage characteristics of the lesion because the types of dural venous obstructions and anomalies vary from case to case. The term "vein of Galen aneurysm" should be abandoned in favor of the term "vein of Galen ectasia."

Since concluding our research in miniaturization of balloon devices for use with children [1], we have been involved in the treatment of patients with vein of Galen aneurysm (VGA). We report six cases of VGA, with special attention to the associated dural anomalies. Careful analysis of these cases and comparison with deep-seated arteriovenous malformations (AVMs) without VGA raises several questions.

The purpose of the present paper is to contribute to the angioanatomic analysis of the VGA, to discuss what may be the primary developmental defect, and to consider the relationship between the anomalies encountered and the clinical outcome of the disease.

Case Reports

Case 1

A 4-kg newborn girl presented with cardiac failure. Vertebral angiography demonstrated a VGA with agenesis of the straight sinus and high-flow venous collateral circulation from the VGA to the torcular (Fig. 1). There was angiographic evidence of arterial steal, but there was no retrograde venous drainage into the cortical veins or sinuses.

Case 2

A 7-kg infant boy presented with an increase in head circumference. A vertebral angiogram showed a VGA with agenesis of the straight sinus and moderately high-flow collateral circulation from the VGA to the lateral and cavernous sinuses (Fig. 2). Angiographically, no arterial steal was demonstrated, but retrograde venous drainage into the cortical veins was noted.

Received March 3, 1986; accepted after revision September 11, 1986.

Presented at the 24th annual meeting of the American Society of Neuroradiology, San Diego, January 1986.

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AJNR 8:185–192, March/April 1987
0195–6108/87/0802-0185

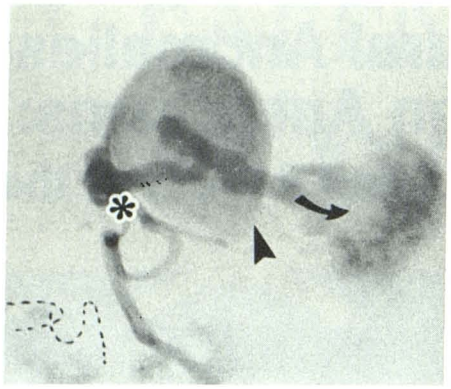


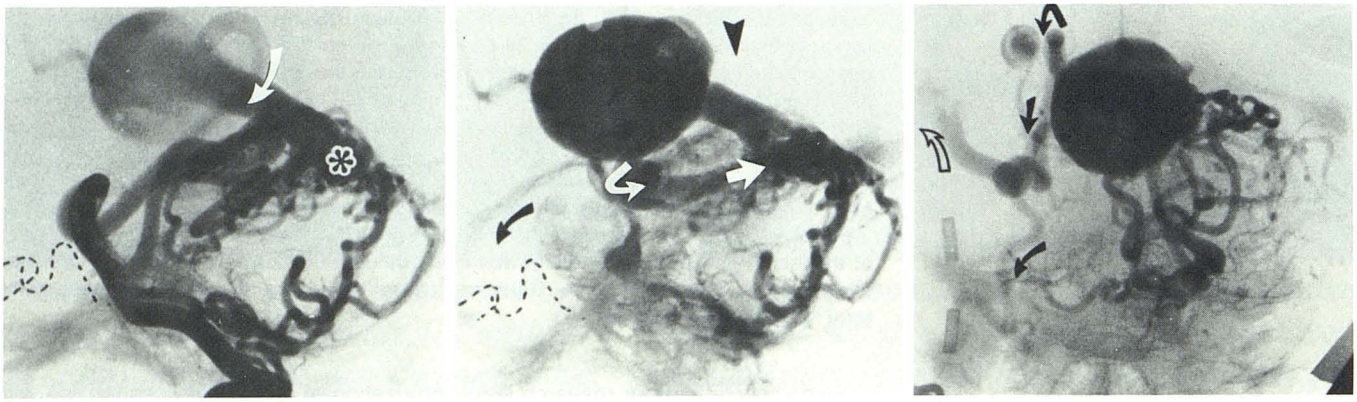
Fig. 1.—Vertebral angiogram (lateral view). Note mesencephalotectal arteriovenous shunt (*asterisk*) and absence of straight sinus (*arrowhead*); a tentorial vein bridges vein of Galen to the torcular (*curved arrow*).

Case 3

A 6-kg infant boy presented with hydrocephalus and moderate cardiac insufficiency. The vertebral angiogram showed a VGA with agenesis of the straight sinus. The lesion drained into the superior sagittal sinus through an accessory straight sinus (falcial sinus) (Fig. 3). There was a moderate arterial steal but no retrograde venous drainage into the cortical veins or sinuses was noted.

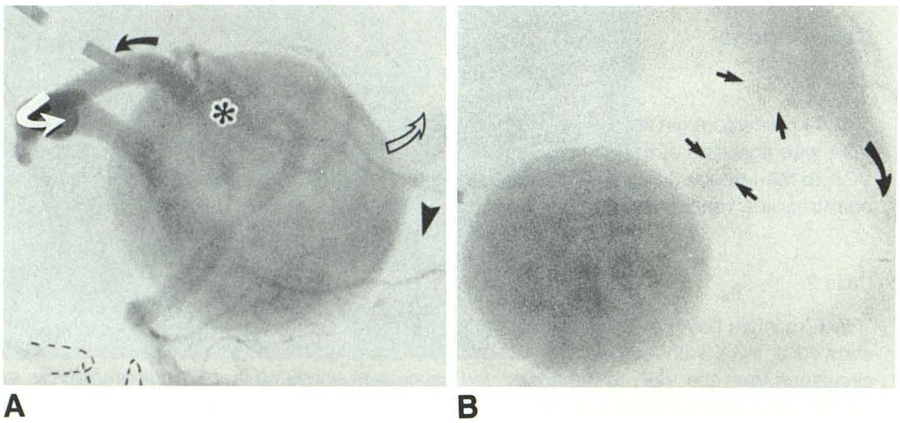
Case 4

A 45-year-old man presented with subarachnoid hemorrhage; a VGA was demonstrated at angiography. Although a straight sinus was visible, the falcial sinus was also opacified. Flow into the lesion was moderately high (Fig. 4). Angiographically, no arterial steal was evident, but retrograde venous drainage into the sinuses and cortical venous stasis were demonstrated.



A **B** **C**

Fig. 2.—Vertebral angiogram. Lateral projection, early (*A*) and late (*B*) phases, and frontal projection, late phase (*C*). Note vermian arteriovenous shunt (*asterisk* in *A*) draining via superior vermian vein (*arrow* in *A*) into vein of Galen aneurysm. Straight sinus is not seen (*arrowhead* in *B*). Venous collateral drainage (*bent arrow* in *C*) opacifies basal vein of Rosenthal and a hippocampal vein anastomoses (*curved arrow* in *B* and *C*) with an infratemporal vein (*straight arrow* in *B* and *C*), which later opens into lateral sinus (*curved open arrow* in *C*).



A **B**

Fig. 3.—Vertebral angiogram. Early (*A*) and late (*B*) phases. Arteriovenous shunt (*asterisk* in *A*) drains into thalamostriate vein and joins at foramen of Monro (*bent arrow* in *A*) with internal cerebral vein. Due to agenesis of the straight sinus (*arrowhead* in *A*), ectatic vein of Galen opens into superior sagittal sinus (*curved open arrow* in *A*) via an accessory straight sinus (*small arrows* in *B*).

Case 5

An 18-month-old girl presented with a cranial bruit, failure to thrive, and nonpulsatile vessels near the medial canthus of the left eye. Angiography demonstrated a VGA with a normal straight sinus. A left jugular foramen agenesis as well as hypoplasia of the jugular foramen on the opposite side were noted. There was no arterial steal, but retrograde venous opacification of cortical veins and dural sinuses was noted (Fig. 5).

Case 6

A 20-year-old man presented with a 2-year history of headaches associated with visual symptoms, thought to be related to migraine. Two months before admission he developed ascending paresthesias first on his left side and then on his right, which spontaneously regressed. Although no abnormality was detected on neurologic examination, he developed nausea and vomiting, which led to admission and CT study. Results of the CT were thought to be compatible with VGA (Figs. 6A and 6B). Angiography showed the entire venous drainage of the left hemisphere to be converging on the vein of Galen. No arteriovenous shunt could be demonstrated. Narrowing of the vein of Galen/straight sinus junction was noted, with pre- and poststenotic ectasia (Fig. 6). The unusual venous drainage pattern was apparently caused by a venous anomaly (obstacle) preventing venous drainage toward the superior sagittal sinus and the cavernous sinus systems.

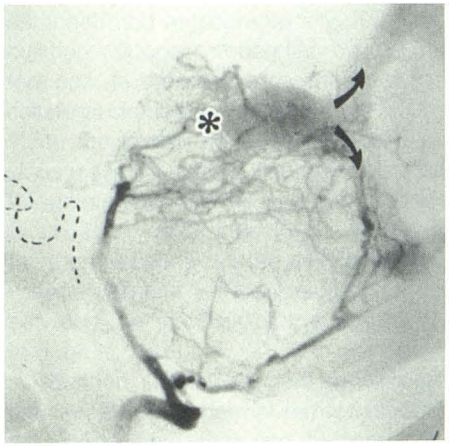


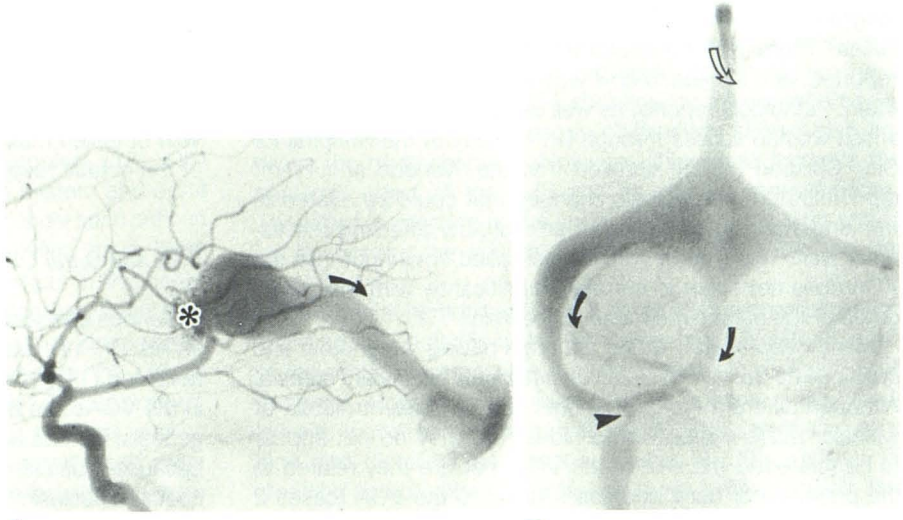
Fig. 4.—Vertebral angiogram in lateral projection. Thalamic arteriovenous shunt (*asterisk*) drains into both an accessory and a common straight sinus (*curved arrows*). There is no angiographic evidence of arterial steal.

Discussion

Analysis of these six patients raised several questions and initiated our review of the literature on the so-called VGA. The normal and abnormal development of the vein of Galen and dural sinuses has been previously reviewed [2–5]. Three items, however, need further discussion and elucidation: (1) VGA and abnormal arteriovenous shunt (AVS); (2) VGA and dural obstruction; and (3) the clinical consequences of the anatomic features encountered.

VGA and Abnormal AVS

In the literature, as well as in five of our six cases, a parenchymatous AVS (with or without dural participation) is associated with VGA. However, in case 6, vein of Galen ectasia developed secondary to an increased input to the vein of Galen caused by an absence of patent superior sagittal and cavernous sinus openings. Collateral venous drainage in this case was noted to converge on the vein of Galen, as it represented the only patent channel available for drainage from the brain. The common feature of patients with VGA is the preferential venous drainage of the proximal AVS into the vein of Galen tributaries [6].



A B

Fig. 5.—Carotid angiogram. Lateral projection, early phase (*A*); frontal projection, late phase (*B*). Note thalamic arteriovenous shunt (*asterisk* in *A*) draining into vein of Galen aneurysm and straight sinus (*curved arrow* in *A*). The enlarged torcula opens into a lateral sinus and an occipital sinus (*curved arrows* in *B*). The only patent jugular foramen (the contralateral one is absent) has a narrow portion (*arrowhead* in *B*) before opening into internal jugular vein. Retrograde opacification of superior sagittal sinus reveals large left frontal vein (*curved open arrow* in *B*), which later drains into superior ophthalmic vein via ipsilateral cavernous sinus (not shown).

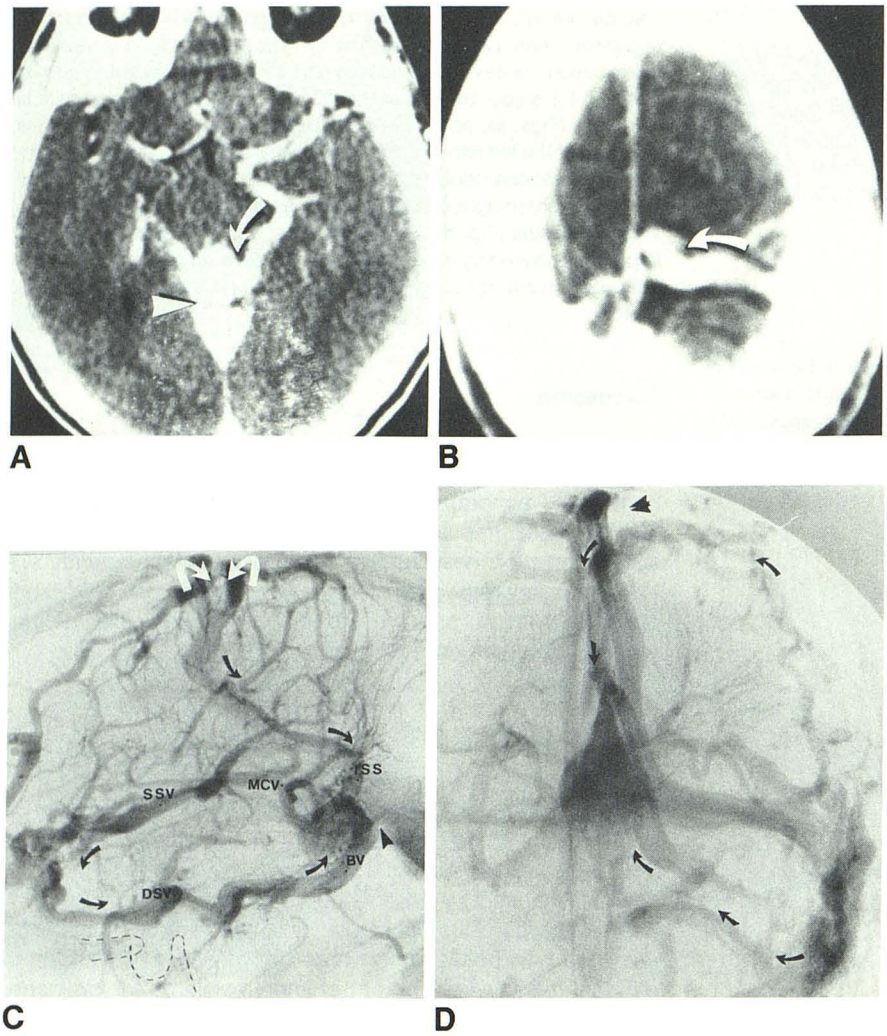


Fig. 6.—*A* and *B*, Contrast-enhanced CT scans. Two enlarged vascular structures corresponding to basal vein of Rosenthal (*curved arrow* in *A*) and to a vein of Trollard (*curved arrow* in *B*) are shown. Vein of Galen aneurysm is visualized with a "weblike" appearance distally (*arrowhead* in *A*) followed by moderate ectasia of straight sinus.

***C* and *D*, Venous phase of left internal carotid angiogram in frontal (*C*) and lateral (*D*) projections. All veins of the cortex converge into two systems: (1) Medial, draining upper frontoparietal cortex; it is unable to join superior sagittal sinus (*double arrowhead* in *D*) and courses with falx (*bent arrows* in *C*) to open into inferior sagittal sinus (ISS). (2) Lateral, remaining cortical territories open into superficial sylvian vein (SSV), which then courses into sylvian fissure anteriorly (*curved arrows* in *C*) and anastomoses with deep sylvian vein (DSV). Posteriorly, this channel opens into vein of Galen via the basal vein (BV). No other venous sinus at base of skull could be seen, particularly at level of middle cranial fossa; no arteriovenous shunt could be demonstrated either in brain parenchyma or in dura. Jugular foramen was patent bilaterally.**

The AVS location can therefore be either within, close to, or remote from the VGA [7] and can even be infratentorial as long as the drainage occurs into a tributary of the vein of Galen. There was no anatomic similarity among the cases reported, and increased flow was a feature common to all of them. Pathologic reports, as well as nonpublished material to which we had access through Dr. Halliday of the Hospital for Sick Children (1985), showed that the "venous aneurysm" demonstrated morphologic changes that could be related to the long-standing presence of arterial flow affecting the venous wall. The type of AVS or increased flow input into the VGA does not seem to correlate significantly with the occurrence of the ectatic response of the vein.

Many deep-seated AVMs although having a high flow and draining into the vein of Galen do not create significant ectasia. Although different types of shunts can be present (fistula or vascular malformation with a nidus [8]), they do not appear to be related to the size of the VGA, nor are they related to the presence of dural anomalies distal to the VGA (cases 2 and 3). Conditions in which rerouting of venous blood is apparently caused by venous obstacles located far from the VGA (case 6) can be associated with a VGA of a size similar to the retrothalamic high-flow AVS draining directly into the aneurysm (case 5). Therefore, the VGA does not seem to be the disease, but a consequence of a hemodynamic phenomenon [7]. Therefore, the existence of an obstacle distal to the vein of Galen must be postulated for a better understanding of the ectatic response of the vein to the increased input.

VGA and Dural Obstacles

Venous agenesis or thrombosis has been described in brain AVMs [9, 10], and their relation to hemorrhage has been reviewed [11]. The high frequency of straight sinus agenesis in the VGAs has been recently reported [4, 12]. As to pathogenesis, venous agenesis seems more likely than thrombosis because multiple reports in the literature fail to confirm that acquired occlusions (even acute) of the straight sinus produce a VGA [13–16]. In all our personal cases and in the published

data, whenever careful anatomic analysis has been carried out a dural venous obstacle could always be demonstrated. Various types and locations of the dural venous obstructions have been noted, the most common type being the agenesis of the straight sinus. The junction of the vein of Galen and the dural sinus seems to be a critical area. If this junction does not enlarge with the increased flow, it becomes a mechanical obstacle leading to proximal ectasia (case 3).

When a patent straight sinus is demonstrated in a patient with VGA (cases 4 and 5), an obstruction can be seen further distally, such as at the jugular foramen level (case 5). Sometimes the anatomic obstacle is difficult to demonstrate due to the superimposition of vessels, and only indirect angiographic signs will demonstrate the hemodynamic constraints, such as intense opacification of emissary veins filling the suboccipital plexuses via the mastoid vein. It is our impression that the closer to the VGA the venous obstruction is located, the larger the ectasia will be.

The development of venous collateral circulation to bypass the constraint will create a variety of pathways that can be demonstrated angiographically. Two types of collateral circulation will occur depending on the patency of the jugular foramen. In the case of agenesis of the straight sinus, the collateral circulation will bridge the VGA to the torcular (case 1), to the lateral sinus (case 2), or to the superior sagittal sinus (cases 3 and 4). The latter disposition is the one most frequently encountered and corresponds to the persistence of an embryonic sinus [3–5], which later disappears and therefore emphasizes the developmental etiology. Additional venous channels will be recruited depending on the balance between the stenosis of the venous channel and the flow through it.

Attention should be paid to retrograde flow into veins draining adjacent normal territories, such as the basal vein, the hippocampal veins, and the posterior fossa veins. In cases of dural venous obstruction along the skull base (case 5), the collateral circulation pattern will vary, as the cavernous sinus bilaterally and the emissary veins of the superior longitudinal sinus and sigmoid sinus represent the only possible drainage for both the AVS and the normal brain. Enlarged facial veins are sometimes noted in patients with VGA, and the presence of pulsations may allow one to determine clinically if they drain the normal brain parenchyma or the AVS.

Enlarged facial veins, which we encountered in one of our cases (case 5), may cause a cosmetic problem [17]. Obviously, knowledge of the cerebral venous system and dural sinuses is mandatory in order to properly analyze such difficult situations [2, 3, 6]. These venous anomalies have been previously correlated with postoperative results in the deep-seated AVMs [18]. Recently, some attention has been paid to the individual characteristics of the venous drainage of dural and brain AVMs and their relationship to the clinical presentation and complications [11, 19–21]. Proper pretherapeutic evaluation of the arterial and venous patterns of VGA in relation to the presenting symptoms is important in order to ascertain the least aggressive treatment with the best clinical results.

Type of Dural Obstacle and Clinical Outcome

The modes of clinical presentation of patients with VGA as related to age have been described in the literature [17, 22–26]. From the neonatal period on, VGA most frequently occurs with cardiac failure, hydrocephalus, and subarachnoid hemorrhage. Failure to thrive, intracranial bruit, cerebral atrophy, epilepsy, and mental retardation are additional symptoms that are frequently noted as the child gets older. In the cases that we were able to review, cardiac failure was only related to the size of the AVS, not to its type (fistula or nidus) or its topography [23, 27, 28].

The dural obstruction encountered in VGA with cardiac failure was either at the level of the straight sinus or at the skull base. Although the obstruction reduced the flow, it was not sufficient to avoid the cardiac failure. However, it is conceivable that the presence of dural obstruction partially protects the cardiac function. The reduced coronary blood flow secondary to the low diastolic pressure, leading to myocardial insufficiency, represents an additional factor conducive to cardiac failure [25]. Since the presenting symptom of VGA in newborns with cardiac failure is not related to the venous ectasia but to the high output, only the correction of the shunt can improve the clinical situation.

The development of ventricular enlargement and its effect on the brain tissue (atrophy, mental retardation, etc.) has been studied and related to the site of the dural anomaly. When the venous obstruction was located at the level of the straight sinus, the collateral venous outflow channels were usually functioning adequately with a relatively moderate increase in pressure. The ventricular enlargement in this situation is due to a direct compression of the sylvian aqueduct. This obstructive (noncommunicating) type of hydrocephalus has been recognized and its mechanism has been well established [24, 29–35].

If the venous obstruction is located at the skull base, the pressure is higher in the adjacent sinuses, which causes a hydrocephalus of the communicating type with diminished reabsorption of CSF due to increased venous pressure in the superior saggital sinus [36]. This may be in addition to obstruction at the level of the sylvian aqueduct caused by the VGA compression. A lumboperitoneal shunt as well as a ventricular shunt have been proposed as therapy in these cases [37]. In addition to CSF shunting, on a theoretical basis, correction of the dural venous obstruction should also improve the hydrocephalus. However, this would increase the systemic effect of the AVS and its cardiac consequences. Therefore, correction of the shunt represents the best option for decreasing the venous pressure into the VGA and the dural sinuses.

Intracranial hemorrhage is the most frequent presentation in the adult population with VGA (cases 4 and 5) [38]. In none of the reported cases was rupture of the vein of Galen itself apparent. Most of the published data suggest that the bleeding site was located far from the AVS, likely on the venous side and distal to the venous ectasia [7, 39]. Analysis of bleeding complications of brain and dural AVMs reinforce this concept [21, 40]. Epistaxis in VGA has also been reported.

The chronic arterial steal phenomenon may not be the only, or even the most important, mechanism for functional brain damage [29, 41–43]. Particularly with a distal dural venous obstruction, the retrograde engorgement of the venous channels that drain the normal brain may produce the same type of neurologic manifestations. Seizures encountered in dural AVMs with cortical venous drainage further support this hypothesis [20, 21]. Failure to thrive, observed in cases without cardiac insufficiency, is a difficult symptom to understand. In case 5, the drainage of both the lesion and the normal brain involved the cavernous sinus and the superior ophthalmic vein. Although still speculative, a hypothalamic hypophyseal dysfunction due to retrograde venous congestion could be postulated.

The prognosis for a child presenting with a VGA remains guarded. Some reports have emphasized the spontaneous calcification of the venous ectasias and its subsequent thrombosis [44–47]. Unfortunately, the venous drainage of the lesion was not carefully studied and no criteria can be drawn from the literature to predict which VGA will calcify and thrombose. This evolution previously thought to be favorable may in fact lead to mental retardation [47, 48].

Therefore, careful attention should be paid to the patterns of the collateral venous circulation (Fig. 7) and the characteristics of the venous drainage of the normal brain. Pretherapeutic evaluation of each venous channel must be achieved especially if the surgical approach to the region may require sacrifice of some of the cortical veins [49–51]. The venous system appears to be the key factor in causing hemodynamic equilibrium or disequilibrium in patients with brain AVMs [10].

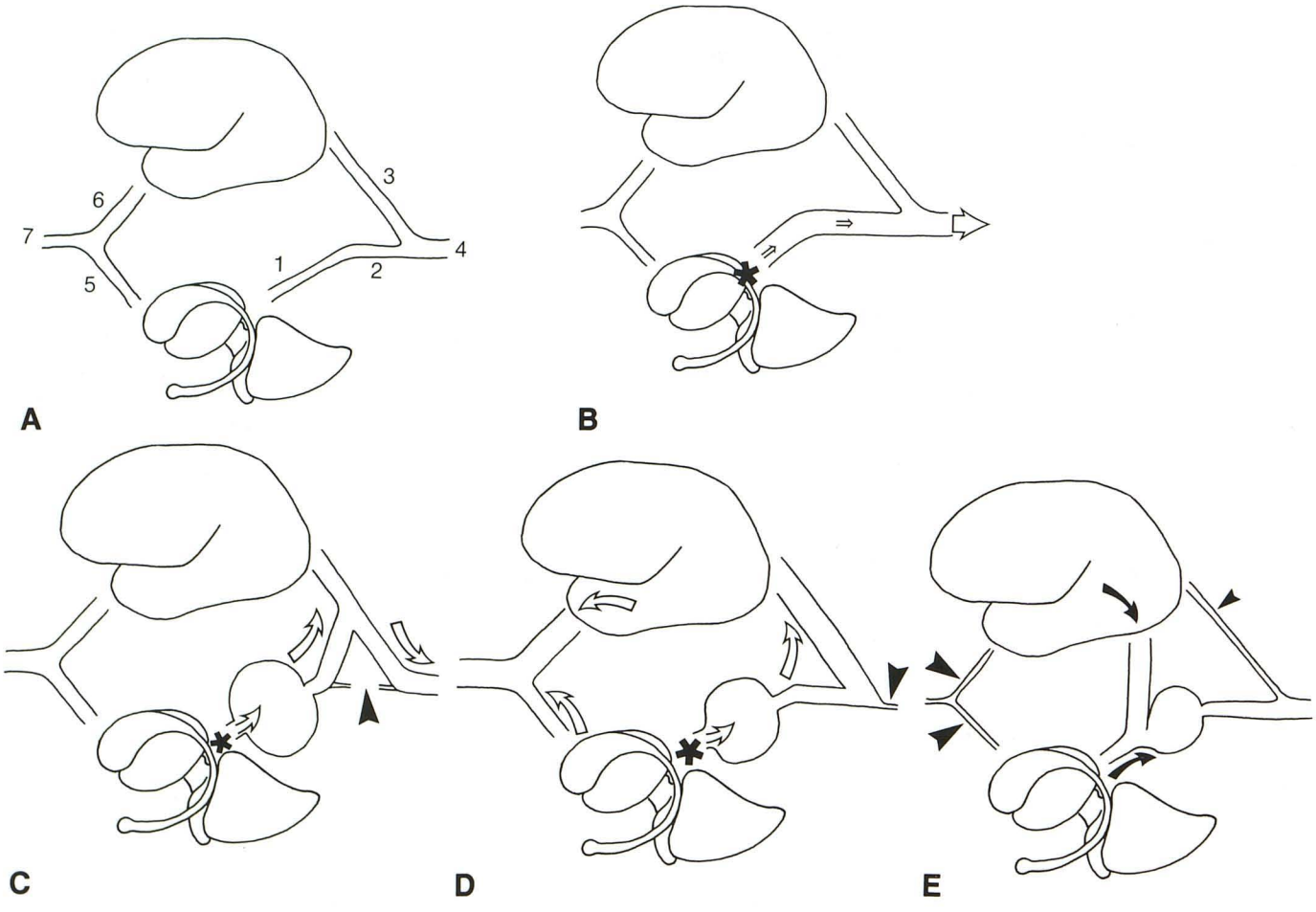


Fig. 7.—*A*, Schematic representation of venous drainage of cortical and deep structures of brain. 1 = vein of Galen; 2 = straight sinus; 3 = superior sagittal sinus; 4 = transverse jugular system; 5 = deep sylvian vein; 6 = superficial sylvian vein; 7 = cavernous sinus. Depending on the variant, the anterior or posterior drainage may be dominant. All intermediate arrangement stages and combinations may be encountered.

***B*, Deep-seated arteriovenous shunt (*asterisk*) draining into an enlarged vein of Galen and straight sinus (*open arrow*) without dural obstruction.**

***C*, Proximal dural obstruction at straight sinus level (*arrowhead*) in a case of deep-seated arteriovenous shunt (*asterisk*). The drainage (*curved open arrow*) bypasses obstacle and drains into transverse sinus and jugular system. Minimal venous constraint will affect remaining brain, but vein of Galen shows marked enlargement.**

***D*, Distal dural obstruction (*arrowhead*) at transverse sinus level in a case of deep-seated arteriovenous shunt (*asterisk*). The venous drainage of lesion will use anteriovenous system (*curved open arrows*) either toward deep sylvian vein or retrogradely via superior sagittal sinus, and the vein of Trollard toward superficial sylvian vein.**

***E*, Cavernous and superior longitudinal venous outlet of deep and superficial system (*arrowhead*) are thrombosed or hypoplastic. The only available venous drainage is vein of Galen, which will develop moderate enlargement due to narrowing at junction of vein at dural sinus.**

Conclusions

Since it has become evident that both the AVS and the dural obstruction are present in every patient with VGA, one could question whether the dural obstruction itself could produce both the ectasia and the shunt. The embryological development of vascular malformations is unknown. There is, however, definite evidence that it does not correspond to a proliferative disease [52]. A purely hemodynamic phenomenon seems possible on a chronologic basis, as the venous system matures before the arterial system and it might possibly have some retrograde influence [3]. It has been suggested that a venous obstruction could maintain the capillary network at a plexiform stage [10]. This developmental dural anomaly would then induce the persistence of a proximal AVS, which would later become a nonreversible fistula or a nidus type of lesion. A similar mechanism has already been proposed to explain parenchymatous AV shunting proximal to aseptic acquired cerebral venous occlusive disease in an adult [53]. In our case 6, the dural obstruction came after the maturation of the capillary bed and must be related to an acquired occlusion of the dural sinuses with a moderate vein of Galen straight sinus anomaly. Therefore, all VGAs could be the consequence of a dural disease [4]. Although interesting, this hypothesis remains speculative and requires further experimental evidence to support it.

On a theoretical basis, treatment of the venous obstruction does not appear to be a desirable goal. Treatment of the shunt, in view of the collected results [25], will best be achieved by endovascular approach either for a complete (although staged) occlusion of the shunt or a partial occlusion to gain time and to control the acute situation [54]. Miniaturization of endovascular devices will allow for repeated percutaneous transfemoral approaches while preserving the femoral artery [1]. This would represent significant progress in the treatment of a frequently devastating diseases process.

The term "vein of Galen aneurysm" is improper and should be changed to "vein of Galen ectasia" whenever the AVS is into the territory of its tributaries, a situation that reflects the presence of a brain AVM. The term "vein of Galen malformation" should be reserved for those cases in which the AVS is located within the vein of Galen wall. "Vein of Galen varix" would indicate a pure venous ectasia of this vessel in the absence of any AVS.

REFERENCES

1. Lasjaunias P, TerBrugge K, Chiu M. A coaxial balloon catheter device for endovascular treatment of newborns and infants. *Radiology* **1986**; 159:269–271
2. Dora F, Zileti T. Common variations of the lateral and occipital sinuses at the confluens sinuum. *Neuroradiology* **1980**;20:23–27
3. Padget DH. The development of the cranial venous system in man from the viewpoint of comparative anatomy. *Contrib Embryol* **1957**;36:79–140
4. Raybaud CH, Hald JK, Strother CHM. Vein of Galen aneurysms. *AJNR* **1985**;6:457 (abstr)
5. Yokota A, Oota T, Matsukado T, Okudera T. Structures and development of the venous system in congenital malformations of the brain. *Neuroradiology* **1978**;16:26–30
6. Huang YP, Wolf BS. The veins of the posterior fossa, superior or galenic draining group. *AJR* **1965**;95:808–821
7. Massey CE, Carson LV, Beveridge WD, Allen MB Jr, Brooks B, Yaghmai F. Aneurysms of the great vein of Galen: report of two cases and review of the literature. In Smith RR, Haerer A, Russell WF, eds. *Vascular malformations and fistulas of the brain.* New York: Raven Press, **1982**
8. Litvak J, Yahr M, Ransohoff J. Aneurysm of the great vein of Galen and midline cerebral arteriovenous anomalies. *J Neurosurg* **1960**;17:945–954
9. Agee OF, Greer M. Anomalous cephalic venous drainage in association with aneurysm of the great vein of Galen. *Radiology* **1967**;88:725–729
10. Dobbelaere P, Jomin M, Clarisse J, et al. Interet pronostique de l'etude du drainage veineux des aneurysmes arterioveineux cerebraux. *Neurochirurgie* **1979**;25:178–184
11. Vinuela F, Nombela L, Roach MR, Fox AJ, Pelz DM. Stenotic and occlusive disease of the venous drainage system of deep brain AVMs. *J Neurosurg* **1985**;63:180–184
12. Lasjaunias P, Manelfe C, Lopez Ibor L, et al. Vascular malformations involving the mesencephalic region. In: Samii M, ed. *Surgery in and around the brainstem and the third ventricle.* Berlin, New York: Walter de Gruyter, **1986**
13. Bedford TBH. The venous system of the velum interpositum of the rhesus monkey and the effect of the experimental occlusion of the great vein of Galen. *Brain* **1934**;57:255–265
14. Bots GAM. Thrombosis of the galenic system veins in the adult. *Acta Neuropathol* **1971**;17:227–233
15. Hammock MK, Milhorat TH, Earle K, Dichiro G. Vein of Galen ligation in primate. Angiographic, gross and light microscopic evaluation. *J Neurosurg* **1971**;34:77–83
16. Yoshii N, Seiki Y, Samejima H, Shibata K, Awazu S. Occlusion of the deep cerebral veins: case report. *Neuroradiology* **1978**;16:287–288
17. Agee OF, Musella R, Tweed CG. Aneurysm of the great vein of Galen: report of two cases. *J Neurosurg* **1969**;31:346–351
18. Laine E, Jomin M, Clarisse J, Combelles G. Les malformations arterioveineuses cerebrales profondes. Classification topographique. Possibilites et resultats therapeutiques a propos de 46 observations. *Neurochirurgie* **1981**;27:147–160
19. Albright AL, Latchaw RE, Price RA. Posterior dural arteriovenous malformations in infancy. *Neurosurgery* **1983**;31:129–135
20. Lasjaunias P, Halimi PH, Lopez Ibor L, Sichez JP, Hurth M, De Tribolet N. Traitement endovasculaire des malformations vasculaires durales (MVD) pures spontanees. Revue de 23 cas explores et traites entre Mai 1980 et Octobre 1983. *Neurochirurgie* **1983**;30:207–223
21. Lasjaunias P, Chiu M, Terbrugge K, et al. Spontaneous intracranial dural arteriovenous malformations (DAVM) neurological manifestations. Report of four cases and review of the literature. *J Neurosurg* **1986**;64:724–730
22. Amacher AL, Shillito J Jr. The syndromes and surgical treatment of aneurysms of the great vein of Galen. *J Neurosurg* **1973**;39:89–98
23. Glatt BS, Rowe RD. Cerebral arteriovenous fistula associated with congestive heart failure in the newborn: report of two cases. *Pediatrics* **1960**;26:596–603
24. Gold AP, Ransohoff J, Carter S. Vein of Galen malformation. *Acta Neurol Scand [Suppl]* **1964**;40:5–31
25. Hoffman HJ, Chuang S, Hendrick B, Humphreys RP. Aneurysms of the vein of Galen. Experience at the Hospital for Sick Children, Toronto. *J Neurosurg* **1982**;57:316–322
26. Levine OR, Jameson AG, Nellhaus G, Gold AP. Cardiac complications of cerebral arteriovenous fistula in infancy. *Pediatrics* **1962**;30:563–575
27. Gomez MR, Whitten CF, Nolke A, Bernstein J, Meyer JS. Aneurysmal malformation of the great vein of Galen, causing heart failure in early infancy. Report of five cases. *Pediatrics* **1963**;31:400–411
28. Long DM, Seljeskog EL, Chou SN, French LA. Giant arteriovenous malformations of infancy and childhood. *J Neurosurg* **1974**;40:304–312
29. Askensay HM, Herzberger EE, Wijsenbekk HS. Hydrocephalus with vascular malformation at the brain. A preliminary report. *Neurology* **1953**;3:213–220
30. Bedford TBH. The great vein of Galen and the syndrome of increased intracranial pressure. *Brain* **1934**;57:1–24
31. French LA, Peyton WT. Vascular malformation of the great vein of Galen. *J Neurosurg* **1954**;11:488–498
32. Gagnon J, Boileau G. Anatomical study of an arteriovenous malformation drained by the system of Galen. *J Neurosurg* **1960**;17:75–80
33. Pribil S, Boon SC, Waley R. Obstructive hydrocephalus at the anterior third ventricle caused by dilated vein from an arteriovenous malformation.

Surg Neurol **1983**;20:487–492
34. Russel DS, Nevin S. Aneurysm of the great vein of Galen causing internal hydrocephalus. Report of two cases. *J Pathol* **1940**;51:375–383
35. Sang H, Kerber C. Ventricular obstruction secondary to vascular malformations. *Neurosurgery* **1983**;12:572–574
36. Gibson JB, Taylor AR, Richardson AE. Congenital arteriovenous fistula with an aneurysm of the great cerebral vein and hydrocephalus treated surgically. *J Neurol Neurosurg Psychiatry* **1959**;22:224–228
37. Rosenfeld JV, Fabinvi GCA. Acute hydrocephalus in an elderly woman with an aneurysm of the vein of Galen. *Neurosurgery* **1984**;15:852–854
38. Castaigne P, Rondot P, Escourolle P, Brunet P, Ribadeau Dumas JL. Malformation de la veine de Galien a revelation tardive. *Etude Anatomoclinique Rev Neurol* **1972**;12:304–312
39. Hirano A, Solomon S. Arteriovenous aneurysm of the vein of Galen. *Arch Neurol* **1960**;5:589–593
40. Yamada S. Arteriovenous malformations in the functional area: surgical treatment and regional cerebral blood flow. *Neurol Res* **1982**;(4)34:283–322
41. Grossman R, Bruce DA, Zimmerman RA, Goldberg HI, Bilaniuk LT. Vascular steal associated with vein of Galen aneurysm. *Neuroradiology* **1984**;26:381–386
42. Norman MG, Becker LE. Cerebral damage in neonates resulting from arteriovenous malformation of the vein of Galen. *J Neurol Neurosurg Psychiatry* **1974**;37:252–258
43. Olivercrona H, Rives J. Arteriovenous aneurysm of the brain: their diagnosis and treatment. *Arch Neurol Psychiatry* **1948**;59:569–602
44. Harwood-Nash DC, Fitz CR. *Neuroradiology in infants and children,* vol. 1. St. Louis: Mosby, **1976**
45. Martelli A, Scotti G, Harwood-Nash DC, Fitz CR, Chuang SH. Aneurysm of the vein of Galen in children: CT and angiographic correlations. *Neuroradiology* **1980**;20:123–133
46. Oscherwitz D, Davidoff IM. Midline calcified intracranial aneurysm between occipital lobes. Report of a case. *J Neurosurg* **1947**;4:539–541
47. Weir BKZ, Allen PBR, Miller JDR. Excision of thrombosed vein of Galen aneurysm in an infant. Case report. *J Neurosurg* **1968**;29:619–622
48. Diebler C, Dulac O, Renier D, Ernest C, Lalande G. Aneurysms of the vein of Galen in infants aged 2 to 15 months. Diagnostic and natural evolution. *Neuroradiology* **1981**;21:185–197
49. Menezes AH, Graf CJ, Jacoby CG, Cornell SH. Management of vein of Galen aneurysms. Report of two cases. *J Neurosurg* **1981**;55:457–462
50. Smith DR, Donat JF. Giant arteriovenous malformation of the vein of Galen: total surgical removal. *Neurosurgery* **1981**;8:378–382
51. Yasargil MG, Antic J, Laciga R, Jain KK, Boone SC. Arteriovenous malformations of the vein of Galen: microsurgical treatment. *Surg Neurol* **1976**;6:195–200
52. Mulliken JB, Zeter BR, Folkman J. In vitro characteristics of endothelium from hemangiomas and vascular malformations. *Surgery* **1982;**92:348–353
53. Gabrielsen TO, Heinz ER. Spontaneous aseptic thrombosis of the superior sagittal sinus and cerebral veins. *AJR* **1969**;107:579–588
54. Berenstein A, Epstein F, Choi IS. The role of embolization in the management of vein of Galen malformation. *AJNR* **1985**;6:457 (abstr)