Neurosurg. Rev.
9 (1986) 233–244

Developmental venous anomalies (DVA): the so-called venous angioma

Pierre Lasjaunias[1], Patricia Burrows[2], and Chantal Planet[3]

[1] Unit of Neuroradiology, Hôpital Bicêtre, France
[2] Department of Radiology, Sick Children Hospital, Toronto, Canada, and
[3] Service de Neurologie, Hôpital du Val de Grâce, France

Summary

Following a review of the literature it is possible to demonstrate the "normality" of the so called venous angiomas. They should be named Developmental Venous Anomaly (DVA). They illustrate in their two extreme types (superfical and deep) the hemodynamic equilibrium of the transcortical venous drainage in the periependymal zones. Venous ectasias and varices which can be encountered, associated with DVA constitute an acquired feature in relation to a venous outlet obstacle.

The sinus pericranii represents an extracerebral DVA, but also corresponds to a normal variation. As any extreme anatomical variant, each DVA corresponds to a weak situation which may express itself clinically; only rare situations justify a radical treatment.

Keywords: Brain venous drainage, transcerebral veins, varices, venous aneurysms, venous angiomas.

1 Introduction

One hundred and ten venous angiomas have been reported in the literature [1, 2, 4–7, 9, 11, 14–19, 24, 26–29, 31, 33–36, 39–47, 49, 51, 52, 54, 55, 57, 58, 61, 65, 66] since 1967. However, in only a few papers have the authors analyzed in detail the morphological [58] or clinical problems [52]. It is surprising that although it has been recognized that these lesions could represent a normal state of affairs [36, 52, 58], there has been no real attempt to analyze the rest of the venous system [52]. In the presence of venous angioma, normal nervous tissue is found between the venous channels, which are structurally normal [1, 5, 8–10, 16, 30, 40, 47, 52, 65, 66]; the lesions can be an incidental finding [18, 52]. Surgical removal [26, 29, 39, 42, 45, 46, 52] or clipping of the vein can have terrible results [52]. Before discussing the morphologic, clinical and therapeutic aspects of these so-called venous angiomas, it is appropriate to review a few points regarding the hemodynamic behavior of the cerebral veins.

2 Functional aspects of the venous system [12, 13, 20–23, 25, 38, 60, 62–64]

2.1 Venous drainage balance

One can consider that, at least supratentorially there are two easily identifiable fundamental venous systems: superficial and deep. Together they form an equilibrium.

Trolard, Labbé, superficial sylvian, and medial hemispheric veins in various forms of dominance can share or drain part or most of the venous blood of a given cerebral hemisphere. No one of these veins (or venous collector) is constant in a given territory. The internal cerebral and the basal vein and their tributaries constitute the prominent components of the deep system. The deep sylvian, lateral mesencephalic, and the hippocampal veins constitute at the same time both a source of equilibrium and an "exit door" for the vein of Galen confluence in case of congestion. As occurs in the superfical territory, a variety of anatomical arrangements can be seen within the deep system with dominance of one or more of the above-mentioned "venous collectors" (Figure 1).

Junctional or anastomotic channels exist between the deep and superficial systems. Normally, they occur at the level of these collectors, in the choroïdal fissure anteriorly or posteriorly. They respectively involve the junction between the deep sylvian-uncate vein and the superficial vein anteriorly and the vein of Galen posteriorly. For completeness, the lateral mesencephalic vein and the petrous vein also constitute junctional areas. These channels are re-

cruited in several well known situations such as tumoral or thrombotic occlusion of a vein or sinus with retrograde collateral circulation, and high flow lesions like arteriovenous malformations. In the former situation the acquired occlusion recruits from an already existing venous pattern, the most efficient channel to drain the cerebral blood extracranially. In high flow lesions, the increased venous pressure related to the arteriovenous shunt is applied to an apparently normal venous system in most cases. Although the vessel enlargment reflects an abnormal situation, the venous arrangement is usually anatomically normal. Additional acquired venous changes may further modify this situation (venous thrombosis, stenotic venous kinking, iatrogenic occlusion, bleeding. etc.).

2.2 Venous ectasias

The vein of Galen aneurysms (VGA) reflect a primary venous abnormality (developmental malformation of the midline sinuses or early thrombosis of the posterior dural draining system), with or without an associated arteriovenous shunt. The venous obstruction leads to an increased pressure upstream and subsequently an ectasia. This mechanism is not related to the increased flow but to the venous obstruction downstream to the ectasia. The location of the obstruction varies from the proximal straight sinus to the jugular foramen, and its severity may range from absence to stenosis. The point here is that venous ectasia [18, 32] (or varix) does not exist without an obstruction in the cerebral venous system. Therefore they do not correspond to a DVA but reflect a secondary response of the venous system, however both DVA and a varix may be encountered. Some key areas favor the development of secondary obstruction and upstream ectasia: the anterior choroidal vein basal vein junction, the foramen of Monro confluence and the atrial vein opening. These veins, when involved in the drainage of a high flow lesion, are prone to the development of "varices" or to becoming "dolichomegalic" due to

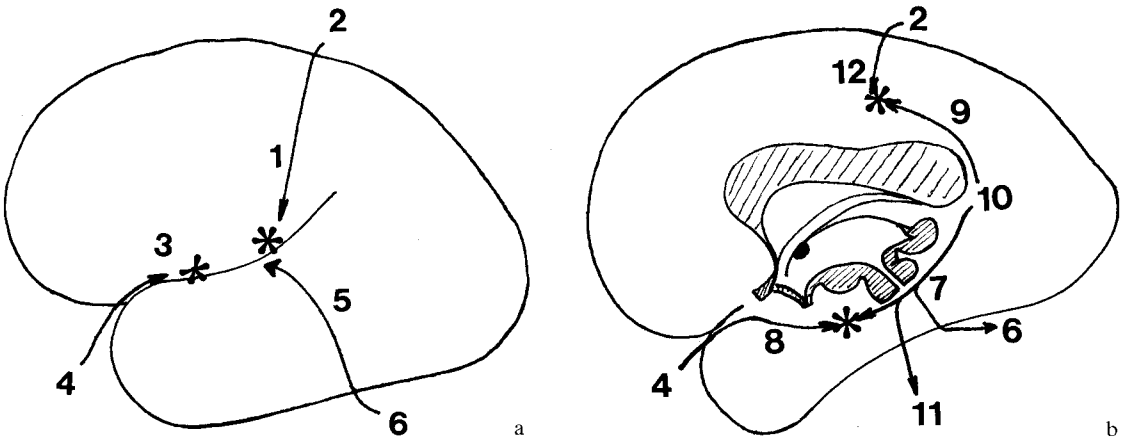

Figure 1. Schematic representation of the superficial (A) and deep (B) venous system, with the areas of intrinsic hemodynamic equilibrium (asterisks).

1. Vein of Trolard draining into the superior sagittal sinus (2)
3. Superficial sylvian vein draining into the cavernous sinus (4)
5. Vein of Labbé draining into the lateral sinus (6)
7. Basal vein draining into the straight sinus (10) anastomosed with the posterior venous system (11) and the lateral sinus (6)
8. Deep sylvian vein draining into the cavernous sinus (4)
9. Inferior sagittal sinus draining into the straight sinus (10)
12. Medial parietal vein draining into the superior sagittal sinus (2)

kinking which occurs at specific points of right angle change of direction of blood flow, plus or minus mechanical fixation.

One of our cases, thought initially to be a vein of Galen malformation and later a venous angioma, is particularly illustrative. This anomaly corresponds to increased flow in the vein of Galen system, following early thrombosis of the basal sinuses, associated with stenosis of the straight sinus. The collateral circulation, the appearance of the veins and the moderate ectasia of the vein of Galen emphasize the role played by both the extraparenchymatous anastomoses and the venous obstructions, in the patterns of venous drainage. In comparison, the congenital venous dysplasia seen in Sturge-Weber disease and the associated venous arrangement is very different, with its rarefaction of cortical venous collectors (Figures 2 and 3).

3 Developmental venous anomalies

3.1 Cerebral (parenchymatous)

The DVA reported in the literature and those which we have studied, consistently demonstrate the following characteristics:

- They drain normal cerebral tissue within a functionally normal arterial territory.
- They are associated with absence of a venous pathway which would normally drain the territory.
- They drain into a normal extraparenchymatous collector.
- At angiography, they are opacified in the same time sequence as the normal veins. DVAs in the frontal lobes may appear to be opacified earlier than normal and may have an associated "capillary blush". However, this blush does not appear pathological, and these features of frontal venous angiomas are not well understood.

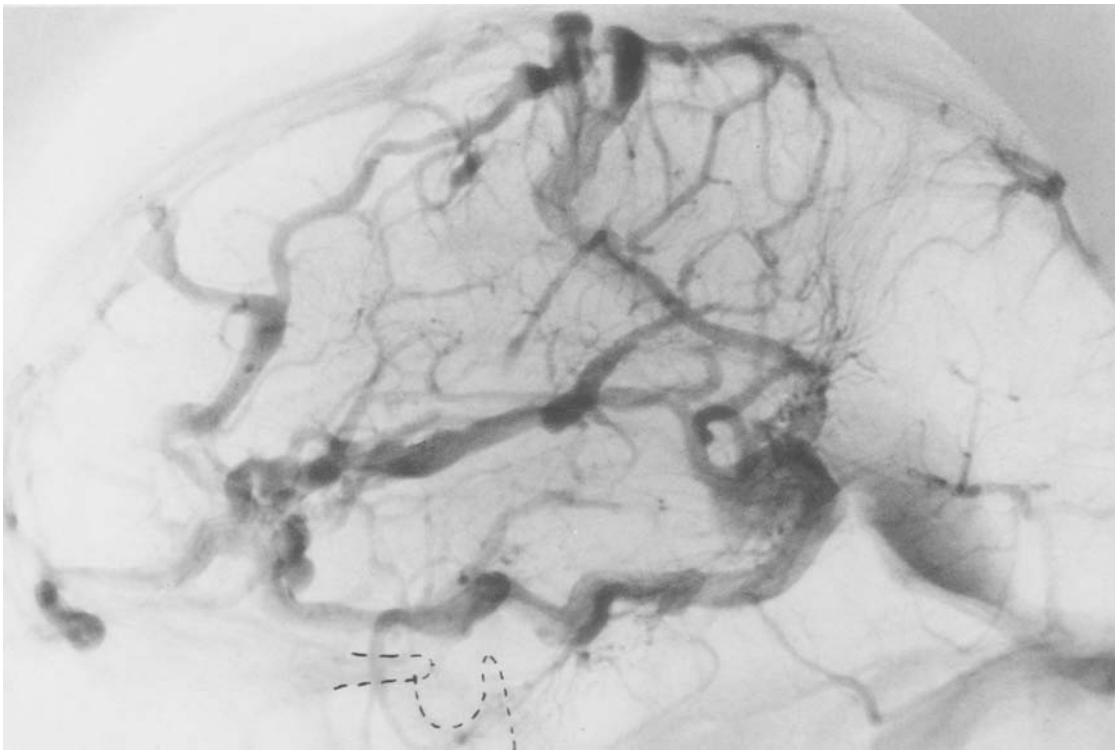

Figure 2. Collateral circulation of the venous system of the right cerebral hemisphere of a young adult. Developmental or early acquired occlusion of the sinuses at the skull base and superior sagittal sinus opening of the vein of the cerebral convexity, has to led to a venous convergence into the vein of Galen. Note the moderate ectasia of the vein of Galen related to a straight sinus stenosis. The arterial and capillary phases were normal (see Figure 6, D and E).

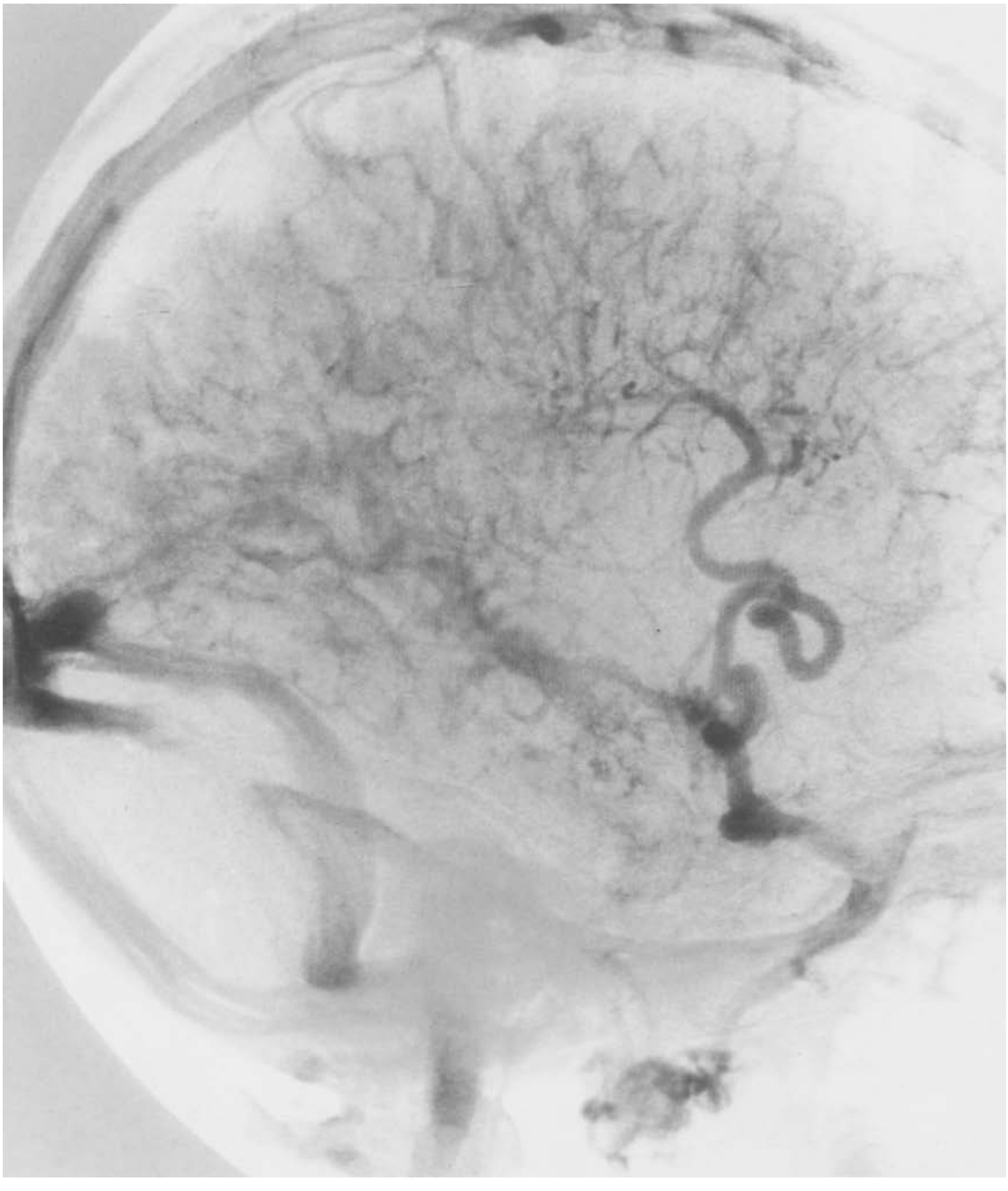

Figure 3. Late phase of internal carotid angiogram in Sturge-Weber disease (arterial and capillary phases were normal).

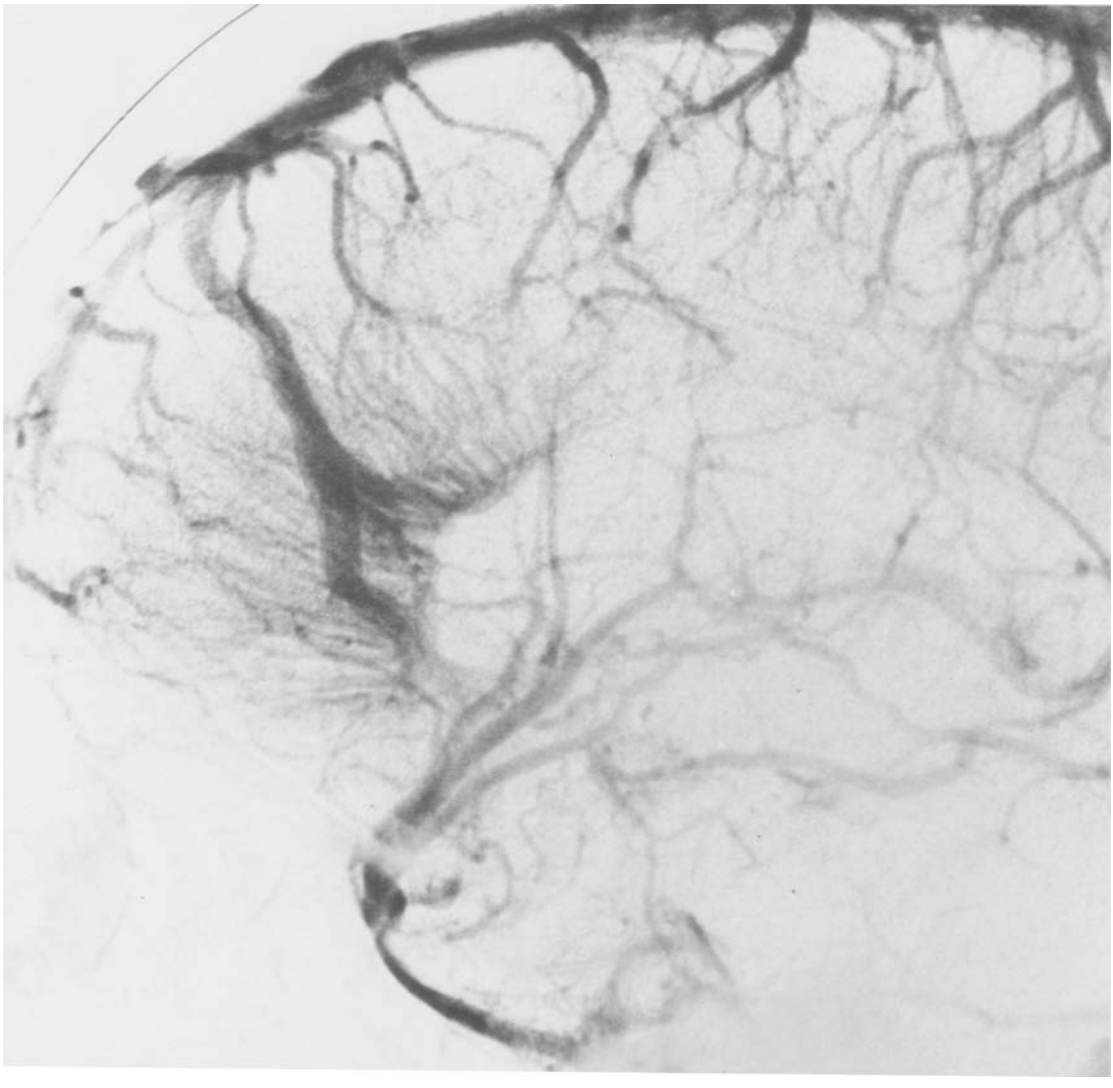

Figure 4. Typical frontal DVA (superficial type) draining the genu of the corpus callosum. Note the usual capillary venous stain in this position (see Figure 6 C).

- The angiographic appearance of these anomalies is that of a series of small deep (parenchymatous) veins which converge towards a larger collector. One or more of these veins follow a transhemispheric course before draining into a normal deep or superficial vein. **It is this image of the capillary-venous convergence and trans-hemispheric course which constitutes the DVA.**

DVAs, in fact, illustrate well-known trans-hemispheric anastomotic pathways which have developed in response to an hemodynamic need. Each one of them can, in a given region, be the dominant drainage of an unusual territory. Depending upon the individual anatomical arrangement present, the DVA may drain a deep territory into cortical collector veins (paraventricular type of Valavanis) or a cortical territory into a deep venous channel (subcortical type of Valavanis) (Figures 4 and 5). We do not think that mixed drainage exists as a given different type; it reflects in fact a patent anastomosis within the network. Supratentorial DVA most often occur in the corpus callosum, although they may involve the temporal or thalamo-capsular regions.

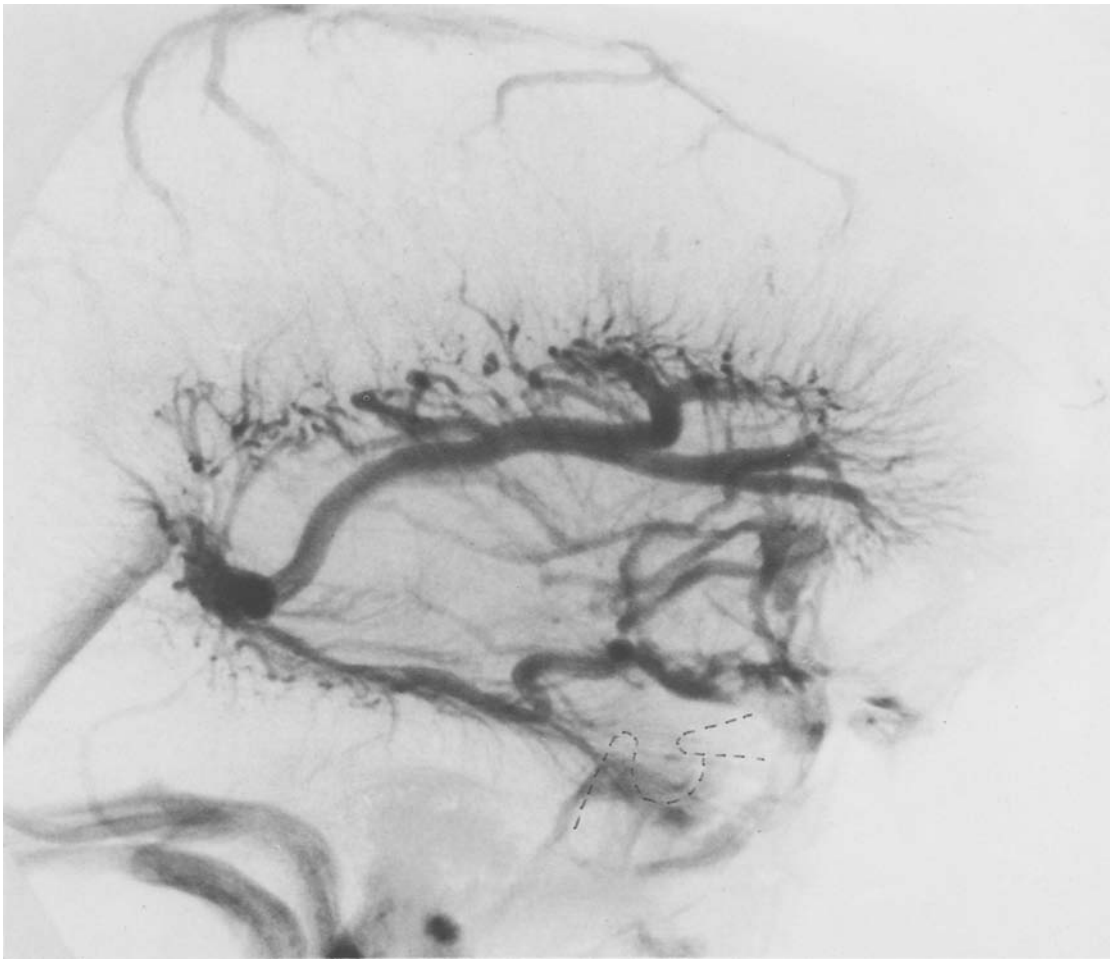

Figure 5. Typical deep DVA, the medial cerebral vein drains the entire subcortical territory via transcallosal veins. Opposite cerebral hemisphere (not shown) drains into a sinus pericranii. These findings were incidental. The angiogram was performed for "pre-operative study of a forehead varix" (see Figure 6 B and 7).

The same functional anatomical approach is valid in the infratentorial so-called DVA; however, due to imaging limitations the missing normal or usual venous channel is more difficult to recognize. Nevertheless, the angiographic pattern is so well – known that the diagnosis is unequivocal (Figure 6).

The term "angioma" should be abandoned for several reasons. There is no proliferation of vascular lumens but instead some sort of developmental venous convergence and concentration, perhaps due to failure of, or abnormal development of the homologous venous channel with which the balance exists. Considering the arteriographic visualization of vascular malformations DVA cannot be considered to be venous malformations since the latter are never filled during conventional angiographic studies. The so-called venous angiomas are then only "venous deviation". They are stable, they should be well tolerated, they do not create any structural damage in the growing brain. However, they constitute, as with every extreme anatomical variation, a weak situation, less flexible than the "normal" one. A given raised pressure may occur in the territory drained by the vein and this may produce ischaemic symptoms, such as a focal deficit, or epilepsy, or may lead to hemorrhage [5, 26, 36, 39, 42, 56]. It is interesting to point out that there is good correlation between the territories drained by the DVA and the symptoms. This is similar to the situation with cere-

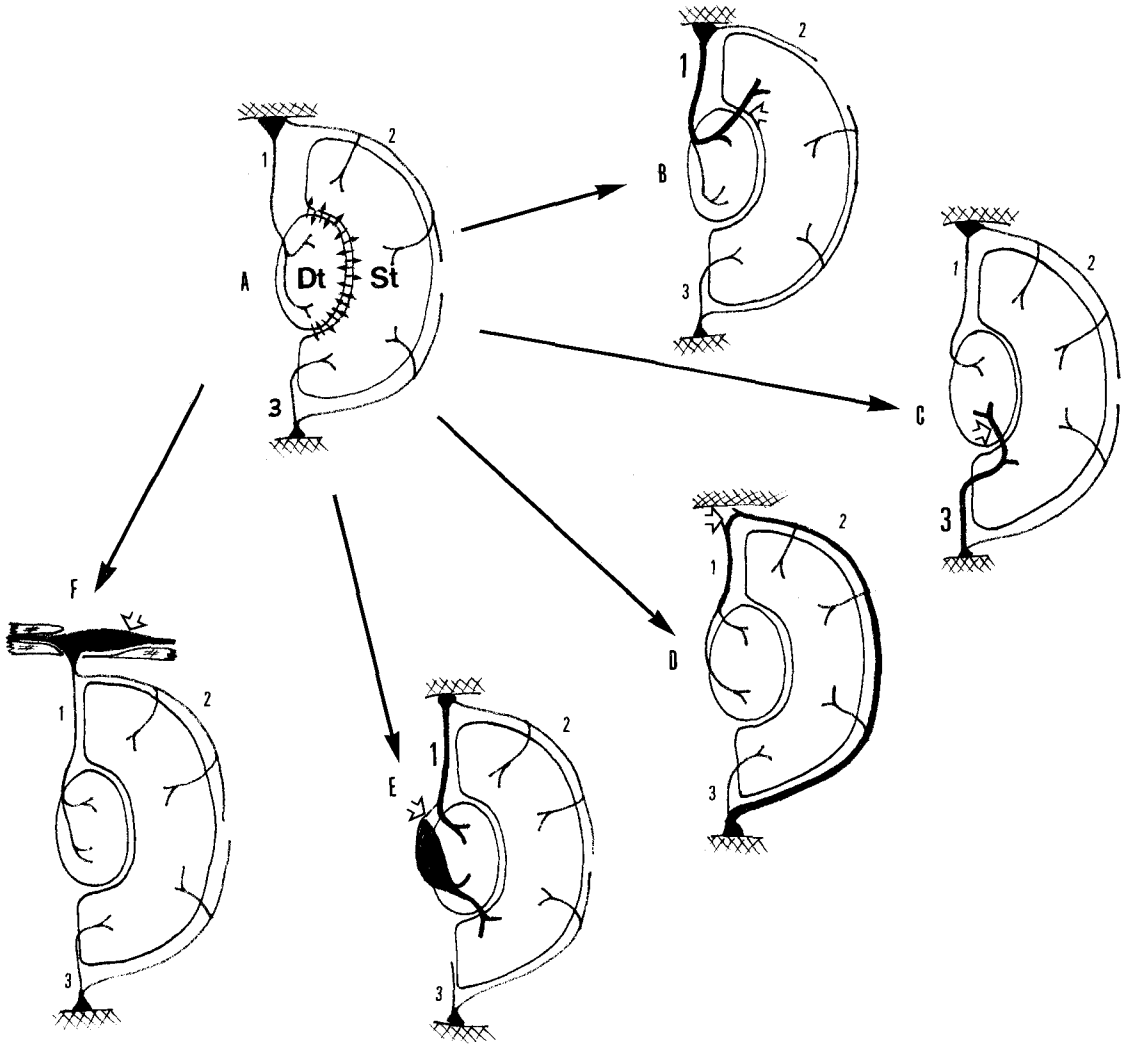

Figure 6. Schematic representation of DVAs.

A. General arrangement of the deep territory (Dt) draining into a dural sinus through its specific vein (1). The superficial territory (St) shows the same draining arrangement (2–3). A paraventricular area is outlined (arrows).

B. Parenchymatous DVA (deep type). Note the subcortical territory (open arrow) collected by the deep system (1)

C. Parenchymatous DVA (superficial type). Note the deep territory (open arrow) collected by the superficial system (3).

D. Following absence (or occlusion) of the dural sinuses (open arrow) the deep venous system (1) drains into the superficial one (2 and 3). However the developmental character of the defect cannot be assessed easily.

E. Association of a deep parenchymatous DVA and varix following the narrowing (developmental or acquired) of the venous collector (open arrow)

F. Sinus pericranii (open arrow), the deep and superficial venous systems drain into an extradural venous pouch. This arrangement can be associated with any of the previously illustrated DVA.

bral AVMs, in which, as seen in another chapter, the clinical findings correlate with the venous drainage, and not with the topography of the arteries or the arterial nidus.

It becomes obvious that these DVA should only be approached when the symptoms do not regress or in the presence of life-threatening complications. Even in these situations the potential adverse effects of iatrogenic occlusion of the venous drainage of a normal territory should be carefully considered. For completeness let us recall that several anatomical variants can be symptomatic and can be surgically treated although respected as a structure: intratympanic internal carotid artery, vascular loops or duplications, ectopic jugular bulb. None of these dispositions is a disease nor a malformation but rather a marginal, extreme variant, which can be explained embryologically which has a normal functional activity. However, they share with the venous variants a tendency to a "weak" behavior brought on, not by retrograde increased pressure but by acquired morphological changes following variations in arterial blood pressure. Treatment usually involves separation of the preserved symptomatic tissue (cranial nerves, spinal cord, cochlea) from the irritating vessels.

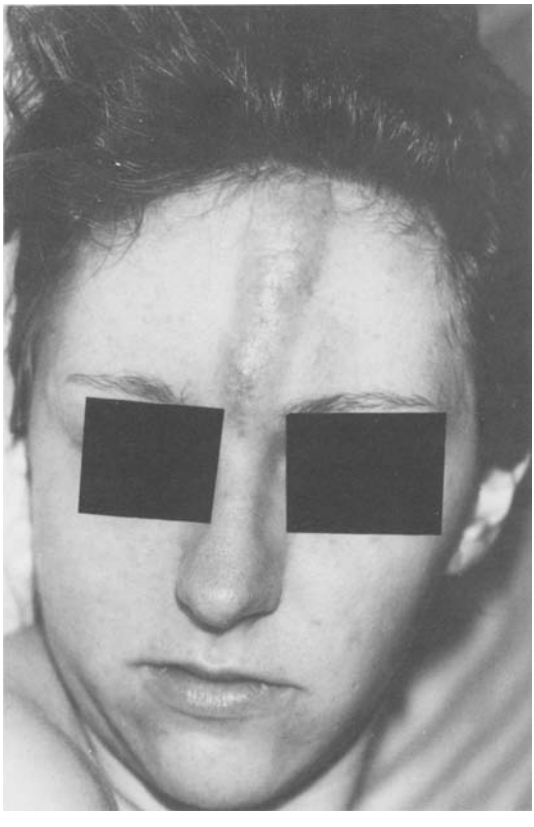

Figure 7. Same patient as Figure 5. The forehead varix drains the normal brain and corresponds to the subcutaneous visualization of a sinus pericranii (see Figure 6 F).

3.2 Osteodural (extraparenchymatous) [3, 12, 35, 37, 53, 59]

The sinus pericranii should be also considered as a third type of DVA in which the venous collectors joins a midline dural sinus at the calvarium before draining into either a diploïc lake and later a subcutaneous vein of the scalp or into subcutaneous venous channel which will present as a cutaneous venous varix (Figure 7). Missing normal venous collectors should be found. These arrangement must be differentiated from:

- intracranial collateral circulation through the ophthalmo-facial venous system in brain AVM (vein of Galen or others).
- scalp venous vascular malformation which may communicate with dural sinuses intracranially; in this case there is no intracranial venous developmental anomaly.
- true venous varix which reflect a dysplastic lesion with a probable orbital rim obstacle; in this situation no intracranial DVA can be demonstrated.

4 Conclusion

When reviewing the literature, one should then look at both the lesions and the remaining tissues to avoid a narrow analysis. This emphasizes again the quality of information given by selective studies compared to the poor contribution of venous or global injections. The concept of malformation should be categorized into functional malformations (AVMs, Sturge Weber etc.) and anatomical malformations, and the term "venous angioma" abandoned, for developmental venous anomaly (DVA).

References

[1] Ahmadi, J., C. A. Miller, H. D. Segall, S. H. Park, C. S. Zee, R. L. Becker: CT patterns in histopathologically complex cavernous hemangiomas. AJNR 6 (1985) 389–393

[2] Avman, N., C. Dinger: Venous malformation of the aqueduct of sylvius treated by intraventriculostomy: 15 years follow-up. Acta Neurochir. 52 (1980) 219–224

[3] Beers, G. J., A. P. Carter, J. I. Ordia, M. Shapiro: Sinus pericranii with dural venous lakes. AJNR 5 (1984) 629–631

[4] Brauch, C. E., A. M. Kunath, J. H. Buscemi: Sublingual venous angioma. Marker of intracranial lesion? Arch. Neurol. 38 (1981) 259–260

[5] Burke, L., R. A. Berenberg, K. S. Kim: Choreoballismus: a nonhemorrhagic complication of venous angiomas. Surg. Neurol. 21 (1984) 245–248

[6] Cabanes, J., R. Balsco, M. Garcia, L. Tamarit: Cerebral venous angiomas. Surg. Neurol. 11 (1979) 385–389

[7] Constans, J. P., D. Dilenge, C. L. Vedrenne: Angiomes veineux cérébraux. Neurochirurgie 14 (1968) 641–650

[8] Courville, C. B.: Morphology of small vascular malformations of the brain, with particular reference to the mechnism of their drainage. J. Neuropathol. Exp. Neurol. 22 (1963) 274–284

[9] Crevits, L., J. de Reuck, E. van de Velde, H. van der Eecken: Venous malformation drained by the precentral cerebellar vein. An angiographic and postmortem study. Clin. Neurol. Neurosurg. 80 (1978) 149–155

[10] Cushing, H., P. Bailey: Tumors arising from the blood vessels of the brain. Springfield, Illinois, Charles C. Thomas 1928

[11] Diamond, C., A. Torvik, P. Amudsen: Angiographic diagnosis of telangiectasias with cavernous angioma of the posterior fossa. Acta Radiol. [Diagn] (Stockh.) 17 (1976) 281–288

[12] Doran, Y., M. D. Peyser: Sinus pericranii – angiographic demonstration. Neurochirurgia 14 (1971) 108–110

[13] Duvernoy, H. M.: The superficial veins of the human brain. Springer Verlag, Heidelberg 1975

[14] Fierstien, S. B., H. W. Pribram, G. Hieshima: Angiography and computed tomography in the evaluation of cerebral venous malformations. Neuroradiology 17 (1979) 137–148

[15] Hacker, D. A., R. E. Latchaw, S. N. Chou, L. H. A. Gold: Bilateral cerebellar venous angioma. J. Comput. Assist. Tomogr. 5 (1981) 424–426

[16] Hamby, W. B.: The pathology of supratentorial angiomas. J. Neurosurg. 15 (1958) 65–75

[17] Hammer, B., H. Pohlhammer: Das venöse Angioma des Gehirns. Diagnostik im Computertomogramm und in der Angiographie. Radiologe 20 (1980) 113–117

[18] Handa, J., K. Suda, M. Sato: Cerebral venous angioma associated with varix. Surg. Neurol. 21 (1984) 436–440

[19] Hirata, Y., Y. Matsukado, S. Nagahiro, J. Kuratsu: Intracerebral venous angioma with arterial blood supply: a mixed angioma. Surg. Neurol. 25 (1986) 227–232

[20] Huang, Y. P., B. S. Wolf: The veins of the posterior fossa – anterior or petrosal draining group. AJR 104 (1968) 35–56

[21] Huang, Y. P., B. S. Wolf: The basal cerebral vein and its tributaries. In: Newton, T. H., G. Polts (eds.): Radiology of the skull and brain Vol. II, book 3, Capt. 74, p. 2111–2154. 1974

[22] Huang, Y. P., B. S. Wolf: Precentral cerebellar vein in angiography. Acta Radiol. [Diagr.] (Stockh.) 5 (1966) 250–262

[23] Huang, Y. P., B. S. Wolf: Veins of the white matter of the cerebral hemispheres (the medullary veins). AJR 92 (1964) 739–755

[24] Kaernbach, A., G. B. Bradex, R. Ferstz, K. Halbhübner: Angiographisch stumme zerebrovaskuläre Malformationen. Radiologe 21 (1981) 243–248

[25] Kaplan, H. A.: The transcerebral venous system. An anatomical study. Arch. Neurol. 1 (1959) 148–152

[26] Kasamo, S., E. Kobayashi, H. Awa, R. Kanemaru, K. Kusumoto, M. Nuro, T. Asakura: A case of cerebral venous angioma as an epileptogenic lesion detected by CT scan and surgically treated. Neurol. Med. Chir. 20 (1980) 865–873

[27] Koussa, A., J. Chiras, B. Poirier, J. P. Carpena, J. Bories: Aspect tomodensitométrique et angiographique des angiomes veineux du cerveau. Neurochirurgie 31 (1985) 161–168

[28] Kramer, R. A., S. D. Wing: Computed tomography of angiographically occult cerebral vascular malformations. Radiology 123 (1977) 649–652

[29] Maehara, T., A. Tasaka: Cerebral venous angioma: computed tomography and angiographic diagnosis. Neuroradiology 16 (1978) 296–298

[30] McCormick, W. K.: The pathology of vascular ("arteriovenous") malformations. J. Neurosurg. 24 (1966) 807–816

[31] McCormick, W. F., J. M. Hardman, T. R. Boulter: Vascular malformations ("angiomas") of the brain with special reference to those occuring in the posterior fossa. J. Neurosurg. 28 (1968) 241–251

[32] Meyer, J. D., P. Baghai, R. E. Latchaw: Cerebral varix and probable venous angioma: an unusual isolated anomaly. AJNR 4 (1983) 85–87

[33] Michels, L. G., J. R. Bentson, J. Winter: Computed tomography of cerebral venous angiomas. J. Comput. Assist. Tomogr. 1 (1977) 149–154

[34] Moritake, K., H. Handa, K. Mori, M. Ishikawa, M. Morimoto, Y. Takebe: Venous angiomas of the brain. Surg. Neurol. 14 (1980) 95–105

[35] Nakayama, T., Y. Matsukado: Sinus pericranii with aneurysmal malformation of the internal cerebral vein. Surg. Neurol. 3 (1975) 133–137

[36] Numaguchi, Y., K. Kitamura, M. Fukui, J. Ikeda, K. Hasuao, T. Kishikawa, T. Okudera, K. Uemura, K. Matsuura: Intracranial venous angiomas. Surg. Neurol. 18 (1982) 193–202

[37] Ohta, T., S. Waga, S. Nishimura, T. Mitani: Sinus pericranii. J. Neurosurg. 42 (1975) 704–712
[38] Padget, D. H.: Development of cranial venous system in man, from view-point of comparative anatomy. Contrib. Embryol. 36 (1957) 79–140
[39] Pak, H., S. C. Patel, G. M. Malik, J. I. Ausman: Successful evacuation of a pontine hematoma secondary to rupture of a venous angioma. Surg. Neurol. 15 (1980) 164–167
[40] Pardatscher, K., D. L. Fiore, F. Galligioni, G. Iraci: Diagnosis of cerebral venous angioma by rapidly enhanced CT scan. Surg. Neurol. 14 (1980) 111–113
[41] Partain, C. L., C. Guinto, J. H. Scatliff, J. Limbacher, P. Janicki, C. C. Heindel: Cerebral venous angiomas: correlation of radionuclide brain scan, transmission computed tomography and angiography. J. Nucl. Med. 20 (1979) 1166–1169
[42] Pelz, D. M., F. Vinuela, A. J. Fox: Unusual radiologic and clinical presentations of posterior fossa venous angiomas. AJNR 4 (1983) 81–84
[43] Preissig, R. S., S. H. Preissig, J. A. Goree: Angiographic demonstration of a cerebral venous angioma. Case report. J. Neurosurg. 44 (1976) 628–631.
[44] Roberson, G. H., C. S. Kase, E. R. Wolpw: Telangiectases and cavernous angiomas of the brain stem: "crytic" vascular malformations. Neuroradiology 8 (1974) 83–89
[45] Rothfus, W. E., A. L. Albright, K. F. Casey, R. E. Latchaw, H. M. N. Roppolo: Cerebellar venous angioma: "benign entity"? AJNR 5 (1984) 61–66
[46] Sadeh, M., I. Shacked, A. H. Rappaport, R. Tadmor: Surgical extirpation of a venous angioma of the medulla oblongata simulating multiple sclerosis. Surg. Neurol. 17 (1982) 334–337
[47] Saito, Y., N. Kobayashi: Cerebral venous angiomas. Clinical evaluation and possible etiology. Radiology 139 (1981) 87–94
[48] Sartor, K., E. Fliedner, K. Weber: Venöse Angiome des Gehirns. RÖFO 128 (1978) 171–176
[49] Sarwar, M., W. F. McCormik: Intracerebral venous angioma. Case report and review. Arch. Neurol. 35 (1978) 323–325
[50] Scott, J. A., G. T. Augustyn, R. L. Gilmor, J. R. Mealey, E. W. Olson: Magnetic resonance imaging of a venous angioma. AJNR 6 (1985) 284–286
[51] Scotti, L. N., R. L. Goldman, G. R. Rao, E. R. Heinz: Cerebral venous angioma. Neuroradiology 9 (1975) 125–128
[52] Senegor, M., G. J. Dorhmann, R. L. Wollmann: Venous angiomas of the posterior fossa should be considered as anomalous venous drainage. Surg. Neurol. 19 (1983) 26–32
[53] Sherry, R. G., M. L. Walker, M. V. Olds: Sinus pericranii and venous angiomas in the Blue-Rubber Bled Nevus Syndrome. AJNR 5 (1984) 832–834
[54] Solomon, E. H., Ch. T. Bonstelle, M. T. Modic, B. Kaufman: Angiographic and computed tomographic correlation in cerebral venous angiomas. Comput. Tomogr. 4 (1980) 217–220
[55] Suganuma, Y., K. Oie, K. Tanigawa, Y. Matsushima, Y. Inaba: A case of cerebral venous angioma. No Shingei Geka. Surg. Neurol. 6 (1978) 77–83
[56] Symond, C.: Cough headhache. Brain 79 (1956) 557–568
[57] Thron, A., D. Petersen, K. Voigt: Neuroradiologie, Klinik und Pathologie der zerebralen venösen Angiome. Radiologe 22 (1982) 389–399
[58] Valavanis, A., J. Wellauer, M. G. Yasargil: The radiological diagnosis of cerebral venous angioma: cerebral angiography and computed tomography. Neuroradiology 24 (1983) 193–199.
[59] Vaquero, J., R. G. de Sola, R. Martinez: Lateral sinus pericranii. Case report. J. Neurosurg. 58 (1983) 139–140
[60] Wackenheim, A. E., J. P. Braun: Angiography of the Mesencephalon. Springer Verlag, New York 1970
[61] Wendling, L. R., J. S. Moore, Jr., S. A. Kieffer, H. I. Goldberg, R. E. Latchaw: Intracerebral venous angioma. Radiology 119 (1976) 141–147
[62] Wolf, B. S., Y. P. Huang, C. M. Newman: The lateral anastomotic mesencephalic vein. AJR 89 (1963) 411–422
[63] Wolf, B. S., Y. P. Huang: The superficial sylvian venous drainage system. AJR 89 (1963) 398–410
[64] Wolf, B. S., Y. P. Huang: The subependymal veins of the lateral ventricles. AJR 91 (1964) 406–426
[65] Wolf, P. A., N. P. Rosman, P. F. J. New: Multiple small crytic venous angiomas of the brain mimicking cerebral metastases. A clinical, pathological and angiographic study. Neurology 17 (1967) 491–501
[66] Wortzman, G., A. F. Sima, T. P. Morley: Cerebral dysplastic vascular malformation: a developmental arrest. Radiology 148 (1983) 443–446

Prof. Dr. Pierre Lasjaunias
Unité de Neuroradiologie
Hôpital Bicêtre
78, rue du Général Leclerc
F – 94270 Le Kremlin Bicêtre, France