Reflections upon the nature and management of intracranial and intraspinal vascular malformations and fistulae

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Evidence is presented that dural fistulae are preceded by sinus thrombosis and that their danger lies in arterialized venous pressure within the cranium or the orbit. Arterial side occlusion leads to recurrence, while venous side occlusion leads to permanent cure. Vein of Galen aneurysms embrace some features of cerebral arteriovenous malformations (AVM's), namely a reticulum, and some features of dural fistulae, namely evidence of previous sinus anomaly and direct drainage into a sinus. These aneurysms are also permanently cured by venous side thrombosis, although the dangers inherent in their reticulum demand that this be done in stages or preceded by arterial side embolization. A very limited experience with venous end occlusion of cerebral (and spinal) AVM's suggests that they, too, can be permanently cured by venous side occlusion without excision. Their reticulum demands maximum, multistage, preliminary arterial side embolization together with intraoperative hypotension during the venous occlusion stage in order to minimize intracerebral hemorrhage or swelling.

Schematic models of both fistulae and malformations are presented, together with reasons why particulate embolization is safer than glue embolization. The theory is advanced that dural fistulae, vein of Galen aneurysms, and AVM's are venous- rather than arterial-based lesions, which is consistent with the experience that permanent cure has been effected by venous side occlusion without excision in all three anomalies. It is speculated that there may be a developmental link between AVM and the venous malformation, the AVM being essentially a fistulized venous malformation.

KEY WORDS • arteriovenous malformation • dural fistula • thrombosis • vein of Galen aneurysm • venous malformation • venous occlusion

Traditionally, the surgeon has approached vascular lesions of the brain by initial arterial occlusion using extravascular methods, in contrast to initial venous occlusion or an endovascular approach. A chance encounter with a patient in whom a posterior communicating artery aneurysm had spontaneously thrombosed and calcified, subsequent to subarachnoid hemorrhage (SAH), led to consideration of nontraditional methods of treatment. For example, after laboratory experiments had determined the parameters of direct current thrombosis and metallic contact thrombosis, a clinical investigation of berry aneurysm thrombosis via either a stereotactic or an open craniotomy approach was undertaken using positive direct current, copper-clad steel needles, copper alloy wire, and balloons. Copper was the most thrombogenic of the metals and alloys that were investigated.

The experience gained from endovascular thrombosis using these materials together with more conventional materials of thrombus induction was then applied to carotid cavernous fistulae, the thrombus being induced on the venous side. The success of this venous approach led to an exploration of endovascular thrombosis of dural sinus fistulae, the vein of Galen aneurysms, and lastly, a very carefully selected series of cerebral and spinal arteriovenous malformations (AVM's). A vein of Galen aneurysm, which acquires its arterial input from intracranial arteries yet delivers its output to the dural sinus, incorporates some of the features of both a cortical AVM and a dural sinus fistula and thus acts as a bridge between two conceptually unrelated lesions.

This paper outlines some of the essential features of these discrete lesions and attempts to identify some
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Fig. 1. Case 1. **Left:** Angiogram demonstrating multiple sinus obstruction with one fistula. The cavernous fistula drains exclusively posteriorly, with a major egress of blood through the mastoid emissary vein. **Right:** Angiogram showing nonfistulous stenosis of the lateral sinus. The sigmoid sinus is completely obstructed.

specific factors that they share. These common factors may bear some relation to pathogenesis and clinical management.

**Dural Sinus Fistulae**

Since a description of spontaneous dural fistula by Houser, et al., in 1979, it has become increasingly evident that this type of fistula arises in an area of previous sinus occlusion. There may be multiple occluded areas, but not all become fistulae. A fistula will not produce serious symptoms unless veins entering the sinus become arterialized, whether in the orbit, the cranium, or both. The following case illustrates some of these features.

**Case 1**

This 70-year-old woman complained of a 3-month history of slight "red eye" and a bruit. Angiography revealed multiple sinus obstructions with one fistula that drained posteriorly (Fig. 1 left). The ophthalmic veins were not involved in the fistula but had been occluded previous to the development of the fistula. There was no distention of intracranial veins, and treatment of the fistula was deemed unnecessary. Angiography also demonstrated a second area of thrombosis in the lateral sinus and total occlusion of the sigmoid sinus (Fig. 1 right). There was an obstruction in the region of the jugular bulb, causing egress of blood from the mastoid emissary vein (Fig. 1 left). Further radiographic studies disclosed a small area of partial occlusion in the midsagittal sinus.

A dural fistula exists in the wall of its sinus. Usually, as in Case 1, it drains into the sinus lumen, but occasionally it drains retrograde into an entering vein that lost contact with the sinus lumen during the thrombotic process. In this manner, it drains into and may distend a wide network of cortical veins. Treatment consists of detaching the fistulous vein; cauterization of its base may or may not be necessary.

**Case 2**

This 63-year-old man had been struck on the top left side of his head by heavy equipment 25 years previous to his current admission; he did not lose consciousness, but suffered a severe headache and was released from Army duty for 3 weeks. Recent symptoms consisted of an intermittent but progressive numbness of his right arm and tingling of the right side of his face over a period of 1 month. Angiography revealed a fistula confined to a very small area on the left lateral wall of the sagittal sinus. This location corresponded exactly to the well-remembered site of his earlier injury. The fistula was supplied extensively by bilateral skin and dural arteries and by the falx, with no internal carotid artery (ICA) supply. It was drained entirely by a single vein, discharging blood into the cortical network of veins over the left hemisphere (Fig. 2 left). From these veins, blood escaped to enter the sagittal sinus anteriorly via another single vein that had retained or regained access to the sinus (Fig. 2 center). Over an extensive length of the midsagittal sinus bilaterally, the cortical veins did not enter from either hemisphere, indicating that they had at one time been occluded and their sagittal sinus openings had not recanalized. On the other hand, the sinus itself had recanalized (Fig. 2 right). The presumption of an earlier thrombosis was based on the marked irregularity of the internal surface of the sinus. The fistula was eliminated by detaching the single vein of origin from the sinus and cauterizing its base. All other veins were left undisturbed. Postoperatively, all of the feeding arteries disappeared, although only ipsilateral arteries were interrupted by the surgery.

A point of interest is that although there was good reason to presume that this fistula was posttraumatic, its appearance and management were identical to those of a spontaneous fistula. It is, therefore, not unreasonable to speculate that posttraumatic sinus fistulae are also based on a period of sinus thrombosis. This case is of further interest in that the symptoms were exactly those attributed to "cerebral steal" of cortical AVM's, although in fact there was no cortical arterial filling in this case. There was, therefore, no arterial steal. It would appear probable that in a classical cerebral AVM, as in this case, symptoms of "steal" are best explained...
FIG. 2. Angiograms in Case 2. Left: Angiogram showing blood from multiple external carotid artery feeders into the sinus wall discharging into one distended cortical vein. Center: From this one vein, the blood fills a distended cortical network of veins and then enters the sagittal sinus through only one anterior channel (arrow). Right: The reconstituted sinus is filled from this one anterior channel (arrow). Double arrows indicate the posterior end of the cortical vein exclusion from the sinus.

FIG. 3. Angiograms in Case 3. Left: Angiogram demonstrating filling of a superior petrosal fistula via the right external carotid artery. Single arrow indicates the straight sinus and double arrows denote the lateral sinus. Note the dangerously distended bulbous veins. Center: Internal carotid angiogram pinpointing the very small precise site of the fistula (arrow). Right: The venous end of the fistula has been clipped (arrow). Note that all feeding arteries have totally disappeared.

by high venous pressure rather than by arterial insufficiency. The same mechanism is probably true in spinal AVM's.

The danger of hemorrhage consequent to bulging veins, the efficacy of venous end occlusion as the primary mode of treatment, and the total disappearance of all feeding arteries after this is accomplished are illustrated in Case 3.

Case 3

This 69-year-old female nursing supervisor suffered a severe intracerebellar hemorrhage and SAH. She was stuporous for 2 weeks, waking up slowly with right cerebellar ataxia, dysarthria, and left arm and leg long-tract weakness. Angiography revealed a fistulous right superior petrosal sinus, which was blocked anteriorly and posteriorly and drained exclusively through a tortuous and bulbous vein toward the straight sinus. The fistula was filled extensively by the right ICA and external carotid artery and by the left ICA (Fig. 3 left). Right external carotid artery embolization had little effect upon the appearance of the fistula. The clue to its management lay in a single frame that showed the right ICA branches focused upon a single draining vein (Fig. 3 center) as the external carotid artery branches did in Case 2. Following right temporal craniotomy, elevation of the temporal lobe, and section of the tentorium, the vein was located and clipped. Its base could not be coagulated because of its proximity to the fourth cranial nerve. Two-tenths of a milliliter of glue was injected in a retrograde manner proximal to the clip. In retrospect, this might not have been necessary. After this simple procedure, all filling arteries from both sides disappeared without a trace (Fig. 3 right) as in Case 2.

In contrast to this total disappearance of the entire dural fistula and all of its feeding arteries following occlusion of the venous end, it is well known that arte-
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Case 4

This 49-year-old man presented with headache, epilepsy, papilledema, and right eye proptosis. Angiography revealed that the proptosis was due to an arterialized ophthalmic vein, as high intracranial venous blood pressure from an extensive lateral and sigmoid sinus fistula sought exit through the cavernous sinus and the ophthalmic vein (Fig. 4 upper left). Initially (in 1978), this high-volume sinus fistula was dearterialized according to an ingenious concept popular at the time: the dura above and below the sinus and the portion of the tentorium attached to it were divided over the entire length of the fistula, leaving the fistulous sinus apparently isolated from all arterial supply. The patient’s symptoms disappeared; however, within 2 years, they had begun to return (Fig. 4 upper center). Within 6 years, his symptoms were more severe than previously (Fig. 4 upper right). The fistula was shut down via a small craniotomy over the sinus. The fistulous segment was isolated by inserting two No. 4 Fogarty balloons, one directed toward the torcular herophili and the other toward the midsigmoid sinus. The intervening length of sinus was packed with Gelfoam and Surgicel. As in Cases 2 and 3, all arterial feeding vessels disappeared (Fig. 4 lower), and there has been no recurrence.

This case is of special interest in that it drew attention to the phenomenon of neoangiogenesis and to the concept of a “satellite” fistula. There was no ICA blood supply before the first operation. Two years later, several new feeding vessels arose from the ICA territory and followed a course not previously identified. Moreover, they arose not directly from the wall of an existing artery, but from a distended cortical vascular plexus. Four years later, these vessels were much more prominent. Following venous occlusion of the fistula, the vessels together with the cortical plexus of their origin simply disappeared. The mechanism of development of both the feeding vessel and the cortical vascular bed that was tapped is completely unknown. One must presume that the fistula, perhaps specifically the fistulous endothelium, has the capacity to create a vasogenic hormone. The structure of these vessels, whether more arterial or venous in pattern, is likewise unknown.
Venous of Galen Aneurysms

The well-known problem of vein of Galen aneurysms, by far the most common of the congenital dural sinus fistulae, is characterized by the frequent absence of the straight sinus in its usual position and by the substitution of a falcine sinus higher up in the falx, entering the sagittal sinus or an enlarged torcular herophili in quite an unusual manner. Occasionally, the sinus is completely obstructed and the vein of Galen drains by an alternative route. Just as it is now recognized that an acquired dural sinus fistula is preceded by an abnormality in the sinus, namely an occlusion, it is clear that a vein of Galen aneurysm, which is in fact a fistula, is associated with (and may be preceded by) a sinus anomaly. This aneurysm can be obliterated, without recurrence, via deletion of its venous sac. There are three types of vein of Galen aneurysms: 1) one in which the feeding vessels empty directly into the vein; 2) one with an external rete; and 3) one with both features. Type 1 aneurysm patients are more likely to present early with heart failure. Type 2 aneurysm patients may present later in childhood with hemorrhage. Case 5 belongs to the Type 2 aneurysm group; it was chosen for illustration in that it represents the most extreme example of straight sinus anomaly: the straight sinus was missing.

Case 5

This previously healthy 12-year-old boy suffered a devastating intracerebral hemorrhage (ICH), emerging from coma with marked right hemiplegia, hemisensory loss, and considerable speech difficulty. Some arterial embolization had already been carried out prior to the patient being transferred to our neurosurgical service (Fig. 5 left). The plan of management was to embolize the filling artery to the limits of safety and then occlude the venous sac with thrombogenic wire. Not all feeding arteries were occluded since they also served vital brain. The sac was then obliterated by insertion of 40 feet of phosphor bronze wire through a small craniotomy via a parafalcine approach. The sac, as is the rule with all congenital vein of Galen aneurysms, was almost as tough as a dural sinus. Again, as in Cases 2, 3, and 4, all of the arteries that supplied the aneurysm and that had not been thrombosed completely disappeared (Fig. 5 right).

Cerebral Arteriovenous Malformations

Case 6 bears some resemblance to a vein of Galen aneurysm in that the lesion was congenital and the patient presented with cardiac failure. The lesion was, however, a cortical AVM.

Case 6

This 4-year-old boy had suffered intermittent heart failure since birth and had a left hemianopsia. Computed tomography (CT) demonstrated that his right occipital lobe, the site of the AVM, and the overlying osseous skull had failed to develop (Fig. 6a). He was intellectually bright, but lacked physical energy. Transfemoral catheterization failed to place a catheter far enough distally into the posterior cerebral artery (PCA) to avoid embolic occlusion of proximal perforating branches that might supply the thalamus and internal capsule (Fig. 6b). The main PCA was surgically clipped where it entered the venous sac. A subdural hygroma developed and was shunted without a valve into the peritoneal cavity on Day 17 after clipping. The malformation continued to have a substantial arterial input from the perforators of the PCA and from the anterior cerebral artery (ACA) and the middle cerebral artery (MCA), none of which was easily amenable to occlusion without a major operative invasion of the hemisphere. It was, therefore, decided to thrombose the sac using thrombogenic wire and to occlude its two visible exiting veins, one of which passed medially toward the straight sinus and the other laterally toward the lateral sigmoid sinus junction. This procedure was performed 3 months later, with the patient at his normal blood pressure of 70/50 mm Hg. This sac was exposed via a limited occipital craniotomy and thrombosed with 40 feet of phosphor bronze wire. The medial exiting vein was clipped, but the lateral vein was not identified (Fig. 6c). Due to the rather wide subdural space, a cotton pack over Gelfoam was placed against the sac to prevent oozing. Fifteen days later, this pack was removed. At that time, the branches of the MCA, which at the previous craniotomy had been seen to pulsate as they coursed over the lateral surface of the cortex and to descend along the medial surface before entering the AVM, were now observed to be completely thrombosed (Fig. 6d). Postoperative angiography performed...
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![Image]

Fig. 6. Case 6. a: Computerized tomography scan revealing a deep occipital arteriovenous malformation (AVM) with failure of development of the occipital lobe and overlying skull. b: Preoperative angiogram, lateral view, showing filling of the AVM via the vertebral artery. The main posterior cerebral artery feeds directly into the AVM, and there are many perforating vessels. Drainage is to both the straight and lateral sinuses. c: Postoperative angiogram showing the results of the occlusion procedure, including two clips on the posterior cerebral artery, one on the draining vein that entered the straight sinus, thrombogenic wire behind the latter clip, and a subdural peritoneal shunt tube. The sac, entering arteries, and draining veins have all disappeared. Note the difference in the perforating vessels compared with those seen in b. d: Preocclusion angiogram showing the middle cerebral vessels coursing over the lateral surface of the hemisphere and down its medial surface to enter the AVM. e: Angiogram obtained after aneurysmal sac occlusion, revealing the disappearance of middle cerebral vascular supply.

2 days previously (Day 13 after venous occlusion) showed absence of all feeding arteries (Fig. 6e) as had been seen in Cases 2 through 5. There is no precise information as to when thrombosis occurred, in other words, on the duration of hemorrhage risk that was accepted from the moment of venous occlusion until these feeding arteries spontaneously thrombosed. Presumably, thrombosis occurred within several hours of obliteration of the sac. The child, alert and vivacious, has had no further cardiac difficulty in the 4 years that have elapsed since the AVM obliteration; his hemianopsia persists.

Two further case histories describe specific points in relation to in situ thrombosis of cortical AVM's. The first of these (Case 7) again illustrates the disappearance of arterial feeders and “satellite AVM’s” once the venous end has been sealed.

Case 7

Sixteen years previously, this 41-year-old woman suffered a serious ICH. The blood clot was removed and some major feeding arteries were occluded. She was hemianoptic. A recent severe headache suggested further hemorrhage. Arteriography showed a deep parietal occipital AVM, in a position rather similar to that in Case 6 with some associated arterial clips. The AVM was supplied by an interrupted, but reconstituted, PCA and by the MCA and ACA. Of special interest was the presence of two satellite AVM’s which filled from the MCA and rather slowly discharged into the main AVM (Fig. 7 left and center). With the patient under hypotensive anesthesia, the main vein was clipped and the sac filled by thrombogenic wire. Postoperatively, all arterial feeding vessels and the satellite AVM’s had disappeared (Fig. 7 right).

Case 8 illustrates that the maximum arterial side interruption followed by venous side occlusion under hypotensive anesthesia may be effective in shutting down some rather formidable AVM’s.

Case 8

This 33-year-old man presented with seizures. Arteriography revealed a deep right parietal AVM which was considered to be too large for resection (Fig. 8a). The AVM was fed by a very large right PCA that appeared to drain directly into it and by major contributions from the right MCA and ACA. The right ACA was supplied across the midline from the left via an anterior communicating artery harboring a large aneurysm. Three years later, the patient suffered an intraventricular hemorrhage, from which he made a good recovery. Occlusion was carried out in stages. The right PCA was occluded in three steps using transfemoral platinum coils. Next, the anterior communicating aneurysm was clipped and the input from the ACA was subtotally interrupted. An attempt was made to embolize the MCA contribution but, despite the asymptomatic occlusion of several cortical arteries, filling persisted (Fig. 8b). In three subsequent craniotomies with the patient under hypotensive anesthesia, the remaining
FIG. 7. Case 7. Left: Early angiogram demonstrating middle cerebral artery filling of an arteriovenous malformation (AVM) fed mainly by the posterior cerebral artery. Note "satellite AVM" formations (arrows).

Center: Late angiogram showing filling by feeding vessels (arrows) arising from the satellite AVM's. Right: Postoperative angiogram revealing that the entire middle cerebral feeding vessels, including the satellite AVM's and the efferent vessels, have disappeared. The same was true of the extensive posterior and lesser anterior cerebral filling vessels. The small clips were placed during partial arterial occlusion performed 16 years previously. The thrombogenic wire coils within the sac and the aneurysm clip on the draining vein were placed during the recent occlusion procedure.

major ACA vessels were interrupted and the venous sac and veins were occluded. The fistulous veins were approached in two of these craniotomies via a very narrow tunnel (Fig. 8c). These procedures were carried out over a period of 3 months. In the final angiographic series, there was no trace of any arterial feeding vessel, rete, or sac (Fig. 8d) despite the fact that, as in all other cases in this series, no excision was made. The patient has returned to his professional teaching job and full physical activity without evidence of any impairment.

Spinal Arteriovenous Malformations

It is feasible that full arterial embolization plus exit venous occlusion could be carried out for selected intraspinal AVM's.

Case 9

Over a 2-year period, this 14-year-old boy had suffered progressive stiffness of his legs due to a known intramedullary AVM at the T10–11 level. Despite consultations at many medical centers, no treatment was recommended. When the patient subsequently fell off his bicycle, he became completely paralyzed. It has been stated, but not confirmed, that a CT scan obtained immediately after the accident showed the presence of blood in the cord. The boy recovered to his preaccident level in about 3 weeks, but then continued his preaccident downward course. He could not run, nor keep up with his classmates when walking, and climbing was especially difficult. His legs were spastic. There was no sensory or sphincter loss. The malformation was supplied from both sides at the T-11 level and from the right side only at T-10. A large anterior vein drained both up and down (Fig. 9 left). A coronary artery distention balloon was inserted into the left artery at T-11 and inflated at the point of origin of the AVM arterial supply. The patient suffered no clinical or electrophysiological deficit. The artery was then occluded at this point by platinum wire coils. A few days later, the right artery at T-11 was catheterized, but this vessel no longer filled the AVM. Next, the right artery at T-10 was catheterized, temporarily occluded by a balloon, and then occluded by coils without incident (Fig. 9 right).

At surgery, the cord was seen to be distended by a red and bulboous malformation. There was no serpiginous collection of dorsal arterialized veins and only moderate evidence of enhanced dorsal arterial activity. A large intradural vessel coursing into it from below was found to be occluded by separated serum and thrombus. Apparently, both intercostal vessels at T-11 supplied this single vessel extradurally. It was much narrower than the two distended veins that emerged anteriorly and flowed up and down longitudinally. A biopsy was not performed. The exact pathway of the vessel filling the occluded artery at T-10, which was known to be separate, was not identified. The midline dorsal vascular bulge and the two emerging anterior veins were cauterized, resulting in no electrophysiological changes. It might be held that the malformation had elements of both a dural fistula and an AVM. A magnetic resonance image of the sagittal and transverse sections certainly show that the AVM lay within, not on the surface of, the cord. This was verified by direct observation. In situ thrombosis of intramedullary spinal AVM's depends upon access to dedicated arterial fillers and their temporary occlusion with the patient in the awake state to determine safety.

Discussion

On close analysis, dural sinus fistulae, vein of Galen aneurysms, and cerebral and spinal AVM's thought to be quite separate entities, have one thing in common. If their venous sac can be safely sealed, their entire arterial input will disappear spontaneously and permanently. This is in contrast to the revascularization that notoriously follows arterial interruption (Case 4). This common factor might suggest that these malformations are all indeed primarily venous-based, rather than arterial-based, lesions.

The pathology of the dural sinus fistula appears to lie within its wall. It is generally believed that fistula
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There are many variations on these themes of occlusion and recanalization that determine the symptomatology and the significance of these fistulae (Fig. 10).

Vein of Galen aneurysms frequently incorporate the features of both an intracranial rete and a failure in development of the normal straight sinus. This suggests the possibility that, at some stage, a normally developing sinus and its entering vein of Galen become occluded. If this were true, then the same mechanism that produces a dural sinus fistula subsequent to thrombosis could produce an aneurysm on a thrombosed vein of Galen. In other words, the rete is secondary, not pri-

formation is preceded by an episode of thrombosis. The possibility exists that, during thrombosis, there was also thrombosis of the venules within the wall and of the ends of the cortical veins entering the sinus. When the sinus lumen recanalizes, the fistula that has developed within the wall drains into the sinus and produces a standard sinus fistula. If the fistula volume is large or the sinus lumen is not completely open, then back pressure will cause retrograde flow through the cortical or ophthalmic veins. Occasionally, the fistula will drain retrograde into one of the entering veins that has not regained continuity with the lumen (Case 2).

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FIG. 9. Case 9. Left: Magnetic resonance image demonstrating a spinal arteriovenous malformation at the T10-11 level, supplied from both sides at the T-11 level and from the right side only at T-10. A large anterior vein drained both up and down (arrow). Right: Left preoperative angiogram at T-11 showing the site of temporary balloon occlusion (arrow).

The idea of some form of disorganization of the hemispheric venous system in relation to AVM's is now receiving increased recognition. Not uncommonly, this disorganization takes the form of a middle cerebral vein complex that fails to connect to the cavernous sinus. In Case 8, the venous drainage of this deeply situated right hemisphere AVM, incorporating three large venous "aneurysmal" dilatations, coursed circuitously to the left hemicranium where it created two further dilatations before escaping into the right lateral sinus. From there, the blood drained back across the torcular herophili into the right lateral sinus.

In the orientation of the venous sac within the wedge of an AVM, at right angles to, rather than on, the surface...
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FIG. 11. Angiogram, lateral view, showing a deep venous malformation which could form the template of the venous core of an arteriovenous malformation had it become arterIALIZED.

and in the associated overlying abnormality of surface veins, the typical AVM resembles another congenital vascular anomaly, the venous malformation. Could a venous malformation with its star cluster be an AVM precursor or template that failed to become fistulous? There is an early embryological system of penetrating vessels that antecedes the adult system of well-recognized surface veins. If the surface system is prevented from developing, the penetrating system must persist in order to drain the hemisphere. This becomes the venous malformation (Fig. 11). If this failure involves a thrombotic process, then the stage is set for the development of an AVM. As in the case of a dural sinus lesion, not all incidents of thrombosis need necessarily lead to fistulization, thus resulting in two very different outcomes and sets of symptoms.

Theory derives from experience and management and in turn may affect management. It seems quite clear to express the opinion, indeed the conviction, that dural sinus fistulae should be treated by occluding the fistulous sinus. Arterial side embolization is mainly temporizing or palliative; it rarely facilitates definitive occlusion. Whether definitive venous side treatment should be carried out via the percutaneous transvenous method or an open surgical endovascular method depends upon the sinus involved. It might be stated that venous side occlusion is also appropriate for vein of Galen aneurysms, provided that the flow is first reduced by arterial side embolization or the venous occlusion is carried out in stages, because sudden occlusion has been known to cause a period of marked intravascular hypertension and brain swelling.

By contrast, the concept of in situ thrombosis of cortical AVM's remains very tentative. There are four patterns of feeding vessels to an AVM (Fig. 12) which might best be seen when an AVM resides in one major arterial territory (for example, the MCA) but derives blood from neighboring territories. 1) "Dedicated" vessels course without interruption or significant branching from an easily visualized artery and enter the AVM directly, without supplying cerebral tissue. They may be tortuous and bulbous or may form a rete and may thus radiographically obscure the sac. 2) An expanded cortical network of vessels from which specific vessels arise which enter the AVM. This cortical network is constituted by the major arterial territory (for example, the MCA) but may be significantly supplemented by branches from the adjoining territories (for example, the ACA and PCA). These adjoining territory arteries more rarely contribute "dedicated" vessels. 3) Occasionally, a cortical area, a satellite AVM at a distance, is tapped by a long "dedicated" vessel (Case 7). 4) A normal perforating artery may enlarge and enter the AVM from the depth of the white matter, becoming in fact a "dedicated" vessel. It is believed that the tortuous and bulbous veins supplying a dural sinus fistula are more dangerous than straight, less distended veins. Perhaps the "dedicated" vessels that create a bulbous distortion in relation to the AVM reticulum are more dangerous than straighter vessels, although the analogy is
not precise since the vessels in one case are efferent and in the other are efferent in relation to the sac.

The concept of distinctive types of feeding vessels relates to the safety of preoperative embolization. Glue, or slurry, embolization of the cortical-derived network (Pattern 2 feeding vessel) is inappropriate as it will cause ischemia of the cortex. Glue embolization of a "dedicated" vessel (Pattern 1 feeding vessel) may cause hemorrhage by obstructing this dedicated vessel distally or by partial or total occlusion of the sac, while its proximal segment persists, distends, and bursts. Wire coil embolization of "dedicated" vessels proximally has, in a limited experience, been quite safe, as has been embolization via Silastic sponge particles of similar dimensions in a much more extensive experience. This is because the obstruction is proximal to the weakest portion of the reticulum. Large particle subtotal embolization of the cortical network that reduces but does not eliminate flow has also been found to be a safe treatment protocol. A single initial symptom of tingling, numbness, weakness, or speech or vision impairment can be calculated to resolve, provided no further emboli are used. The extensive blood supply through the cortex to these lesions will quickly compensate for occlusion of one branch to the network, as was seen with the embolic occlusion of several cortical vessels in Case 8 without clinical incident. Straight coils 2 mm in length seem most appropriate for the many stages of arterial side interruption, percutaneous or via craniotomy, which are efferent in relation to the sac.

In situ occlusion of intracerebral AVM's subsequent to maximum safe craniotomy arterial embolization has been successfully accomplished in 13 patients; in these, any disruption of cerebral tissue inherent in AVM resection was avoided. It would appear, as in Case 7, that many stages of arterial side interruption, percutaneous or via craniotomy, may be required before final occlusion of the venous end (with the patient under hypotensive anesthesia) is attempted. It may be possible that hypothermic supplementation of intraoperative hypotension would be desirable. It is not known how long hypotension should be carried into the postoperative period since it is also not known exactly when the arterial side thrombosis is complete. In situ occlusion of intraspinal AVM's has been successfully carried out in two patients, but the experience is too limited for further comment.

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