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Dural Arteriovenous Malformation of the Major Venous Sinuses: An Acquired Lesion

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Arteriovenous malformations of the dura are thought to be congenital. However, arteriographic investigations of four patients who, after a head injury, developed dural arteriovenous fistulae with features of congenital malformations suggest that these abnormal communications may also be acquired. Thrombosis or thrombophlebitis in the dural sinus or vein may be the primary event in their formation. The pathogenesis is probably "growth" of the dural arteries normally present in the walls of the sinuses during the organization of an intraluminal thrombus. This may result in a direct communication between artery and vein or sinus, establishing an abnormal shunt. Ultimate fibrosis of the sinus wall and intraluminal thrombus may be the factors responsible for the spontaneous disappearance of such malformations.

Most dural arteriovenous malformations (AVMs) that involve the major venous sinuses present either spontaneously or as incidental findings during arteriography performed for other reasons. They occur predominantly in women over age 40 years [1]. The angiomatous network, multiple feeding arteries, numerous arteriovenous (AV) shunts, and occasional association with cerebral angiomas [2], as well as a few cases reported in children [3], suggest that these AVMs are congenital. Thrombosis of the draining sinus or vein is thought to be responsible for the occasional spontaneous disappearance of these lesions [4, 5].

Our experience with four patients who, after a head injury, developed dural AV fistulae with features of congenital malformations prompted a review of the literature and this report. In two of our four cases, transverse and sigmoid sinus abnormalities were demonstrated angiographically before the AV fistulae developed. In case 3, angiography 2 years earlier for unrelated reasons was normal; no AV shunt was seen. Although in case 4, there was no angiographic evidence of a dural fistula before head injury, the posttrauma onset of symptoms and the angiographic findings strongly suggested that this lesion had been acquired.

Cases 1 and 3 were studied by serial angiography with selective internal, external, and common carotid injections, while stereo magnification serial angiography with selective and superselective catheterization of the feeding arteries was performed in cases 2 and 4. The first three patients were followed by periodic angiography for up to 4 years, and the AV shunts were seen to close spontaneously.

Case Reports

Case 1

A 56-year-old woman was examined for headaches and dizzy spells. Cerebral angiography to rule out transient ischemic attacks was normal (figs 1A and 1B). Control of her mild diabetes caused her headaches and dizzy spells to disappear.

She was brought to the emergency room after a head injury 2 months later. She had a bruise over the right occipitomastoid area, was confused and disoriented, but she had no

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neurologic deficit. When, after 1 week, her condition had not improved, cerebral angiography was performed, which showed a normal arterial phase. In the venous phase, however, the wall of the right sigmoid sinus was irregular and the internal jugular vein (fig. 1C) was not opacified, indicating thrombosis or thrombophlebitis.

She recovered completely, but 6 months later returned to the hospital complaining of tinnitus and a buzzing noise in her right ear. Auscultation revealed an audible bruit over the right mastoid region. Cerebral angiography showed multiple dural AV fistulae supplied by the occipital and middle meningeal branches of the right external carotid artery (figs. 1D–1E).

At surgery, the meningeal branches of the occipital artery and the mastoid emissary vein were ligated extracranially. Postoperatively, her complaints of tinnitus and buzzing noise behind her right ear disappeared, but, on auscultation, bruit was still audible. A repeat angiogram showed that AV shunts were still present but flow through them had decreased (fig. 1F). Follow-up arteriography a year later (not illustrated) showed that the right middle meningeal artery was no longer supplying the fistulous communications. Instead, the arterial supply from the transosseous and other meningeal branches of the occipital and the ascending pharyngeal arteries appeared to have increased. The major venous flow at this time was noted to be via the condyloid and other emissary veins into the paravertebral venous plexus. The internal jugular vein opacification was delayed, suggesting stenosis or occlusion in the region of the jugular bulb.

She returned to the hospital again 3 years after her first admission complaining of right occipital headaches. Right common, external, and internal carotid arteriography showed that the fistulous communications had disappeared spontaneously (fig. 1G). The right sigmoid sinus was abnormal; its lumen was narrowed and its wall irregular, particularly at the site of the previous fistulous communication (fig. 1H). The patient was treated conservatively, and her headaches improved with medication.

Case 2

A 50-year-old male construction worker was admitted to the hospital after a head injury that caused him to lose consciousness for 15 min. He was confused but neurologically intact on admission. Skull films showed diastasis of the right lambdoidal suture, but a computed tomography (CT) scan was normal. After 3 weeks, he had not improved and was increasingly disoriented. CT was repeated, and compression and blunting of the frontal horn of the left lateral ventricle were evident.

These findings prompted bilateral common carotid arteriography, which revealed a left frontoparietal subdural hematoma and an AV fistula in the scalp on the right side (figs. 2A and 2B). The subdural hematoma was evacuated, and it was decided to evaluate the scalp fistula at a later date. He was readmitted 6 months later. Angiography did not show the previous scalp lesion. Instead, multiple dural AV fistulae were seen, draining into the right lateral sinus (figs. 2C–2F). The patient refused treatment but returned a month later for embolization.

A preembolization angiogram showed that the fistulae had disappeared spontaneously; the lateral sinus was not opacified but the angiomatous network was still evident (fig. 2G). Irregularity of the right lateral sinus wall and the defect in its lumen noted previously were absent, perhaps indicating organization and fibrosis of the intraluminal thrombus (fig. 2H). Follow-up on this patient continued.

Case 3

A 38-year-old man was brought to the emergency room in a coma after a head injury. He had a bruise over the left temporooccipital area and was bleeding from the left ear. Skull films revealed a left temporal fracture traversing the mastoid air cells; a CT scan showed bifrontal intracerebral hematomas. He improved gradually with conservative treatment, but after 3 weeks he had a grand mal seizure. Cerebral angiography confirmed the bifrontal intracerebral avascular masses and, in addition, showed a dural AV fistula in the wall of the right lateral sinus (figs. 3B–3D) draining into a cortical vein.

Review of the right common carotid arteriogram obtained during a workup for headaches 2 years earlier was normal (fig. 3A). In that study no AV shunt was seen. Because the patient was in poor condition, he was treated conservatively and improved gradually. A repeat CT scan 8 weeks later revealed that the hematomas had been replaced by areas of decreased attenuation. Angiography 2 years after the patient was discharged showed the fistula had closed spontaneously (fig. 3E).

Case 4

A 66-year-old man with a left acoustic neurinoma had a complete hearing loss on the left and slight facial asymmetry, with minimal left facial droop. He also had slight weakness of his right arm that he attributed to a head injury 3 years earlier, at which time he was unconscious for 3 days. CT and cerebral angiography confirmed a mass lesion in the left cerebellopontine angle.

Left external carotid arteriography also revealed a dural AV fistula in the wall of the superior sagittal sinus in the falx (figs. 4A–4F). At surgery, all of the meningeal feeders were ligated and the dura excised. Surprisingly, after surgery, strength in his right arm increased. He was readmitted to the hospital 3 months later and the mass lesion in the left cerebellopontine angle, which proved to be an acoustic neurinoma, was successfully removed. An angiogram at this time showed the dural AV shunt had not recurred.

Discussion

Normal Arterial Supply of the Dural Walls of the Venous Sinuses

In considering the pathogenesis of these malformations, a brief review of the normal arterial supply of the dura walls of the great venous sinuses may be useful. The dura mater is highly vascular, especially in the region of the great venous sinuses. The middle meningeal artery normally ends in the anterior and posterior paramedian meningeal branches, which run along the insertion of the dural wall of the superior sagittal sinus at the convexity, as far as the cribiform plate anteriorly, and to the foramen magnum posteriorly. These paramedian vessels also receive collaterals from the anterior and posterior meningeal arteries, each of which, before termination, gives off branches to the dura of the roof and lateral wall of the superior sagittal sinus [2]. A similar rich dural arterial network is created in the walls of the lateral and sigmoid sinuses by the abundant collateral and anastomotic channels between the middle meningeal artery and the meningeal branches of the occipital, ascending pharyngeal, and internal carotid arteries. Physiologic AV shunts between these meningeal arterial networks and dural venous sinuses have been described [6–8].

Previous Reports

During the past decade, over 70 cases of dural AV malformations have been reported [9]. Although found in both

genders and at all ages, their incidence is highest in women over 40 years old [1]. Recent reports have described the angiographic anatomy of the feeding arteries [10–12]. A very high incidence of abnormalities of recipient venous channels has also been reported [13]. These abnormalities are described as irregularity and rigidity of the wall, stenosis and septation with filling defects in the lumen, and retrograde flow with partial or complete occlusion of the draining venous sinus or vein. Such changes are believed to be due to venous thrombosis, which is thought to be responsible for the occasional spontaneous disappearance of these lesions [4, 5].

While these malformations are generally considered congenital [14], a few authors maintain that they are acquired [1, 2, 15]. Aminoff [15] suggested that since dural arteries and veins (unlike brain parenchymal vessels) are closely associated anatomically, trauma, especially a fracture crossing these vessels, can cause their simultaneous rupture, resulting in a fistulous communication. However, this concept holds true only for major branches of the middle meningeal arteries and their accompanying veins. It does not apply to dural AV malformations located in the walls of major venous sinuses, as venous blood from the dural walls of these sinuses appears to drain directly into the sinuses. Furthermore, middle meningeal and other posttrauma AV fistulae are usually seen in an immediate postictus period and have a classic angiographic appearance [16], with a single AV shunt between the ruptured artery and vein.

We found at least three case reports of dural AV malformations located in the walls of the venous sinus in which the onset of symptoms was preceded by head injury [4, 17, 18]. In each of these, as well as in our cases, the lesion consisted of multiple AV shunts, and the interval between trauma and the time at which symptoms appeared varied from a few weeks to 6 months. Because most reported cases did not have a history of trauma, we feel that a mechanism other than direct trauma itself is responsible for the development of these fistulae.

Pathogenesis

Thrombosis or thrombophlebitis of the draining vein or sinus appears to be a common feature of these lesions and may be related to their origin.

In a discussion of these fistulae, Djindjian and Merland [2] described in detail the venous abnormalities indicating sinus thrombosis and suggested that such venous abnormalities resulted from previous medical problems. They noted that in almost all of their patients, complaints of bruit and other symptoms had been preceded by a variety of medical illnesses, surgical and obstetric procedures, and head trauma. They postulated that thrombosis or thrombophlebitis resulting from such prior events precedes the development of fistulous communications. According to this hypothesis, the abnormal communications are merely a pathologic "opening-up" of physiologic AV shunts in the dural walls of the venous sinuses in response to phlebitis.

This hypothesis was partly confirmed by Houser et al. [1], who angiographically demonstrated that venous abnormalities preceded the formation of fistulous communications in two of their patients. They postulated that "the indigenous dysplastic dural vessels present within the sinus wall develop further, establishing a direct artery-to-sinus communication during the organization of an intravenous thrombus." No experimental work supporting either of the above hypotheses is available. However, patient histories, angiographic findings of prior venous abnormalities, the prolonged interval before fistulae develop, and the dysplastic vessels in surgical specimens and in angiograms (fig. 2G) tend to support the hypothesis of Houser et al. [1].

The cases reported in children may be explained by compression and injury of the dural sinuses from calvarial molding during birth [19], resulting in sinus thrombosis and fistulous communications during the organization and recanalization of the intraluminal thrombus. No matter what the mechanism, once the shunts are established, the rapid flow of blood through them causes the preexistent rich meningeal arterial network in the sinus wall and surrounding dura mater to dilate and hypertrophy. Abundant collateral and anastomotic channels between various meningeal arteries result in multiple feeders.

Our Experience

In our first two cases, the shunts were located in the walls of the lateral and sigmoid sinuses and drained into the lumen of the respective sinus. In contrast, in cases 3 and 4, even though the abnormal shunts were located in the walls of the lateral and the superior sagittal sinus, they drained into the cortical veins in a retrograde manner. The fistulae were located at a point where the recipient cortical veins normally entered the sinus. The retrograde flow in these veins suggested that thrombosis and occlusion had occurred at their junction with the respective sinus. Close examination of the early films of the arterial phase in each case revealed that, like congenital malformations, the abnormal shunts were multiple and confined to an abnormal segment of the dural wall of the respective sinus.

The spontaneous disappearance of these abnormal dural fistulae have been reported previously [4, 5] and it is thought to be due to thrombosis of the recipient sinus or vein. However, preexisting venous abnormalities in two of our cases, as well as in those reported by Houser et al. [1], indicate that thrombosis itself is not responsible for the spontaneous closure of these shunts. Ultimate fibrosis of the intraluminal thrombus and the inflamed sinus wall probably resulted in occlusion of the fistulous communications. Partial or complete obliteration of the dural sinus [4] may also result. If the sinus remains patent, narrowing and stenosis of its lumen and irregularity (cases 1 and 2) of its wall, particularly at the site of the shunts, may be seen. The long interval before the shunts closed spontaneously may be related to the slow organizing and fibrosing process [20]. That the angiomatous network persisted after the shunts had closed (case 2) is possibly related to alterations in the normal lamination of elastic muscle fibers in the walls of dural arteries as a result of hemodynamic changes.

The four dural AV malformations reported here appear to be acquired and this may be the mechanism in many others. Thrombosis or thrombophlebitis of the recipient venous

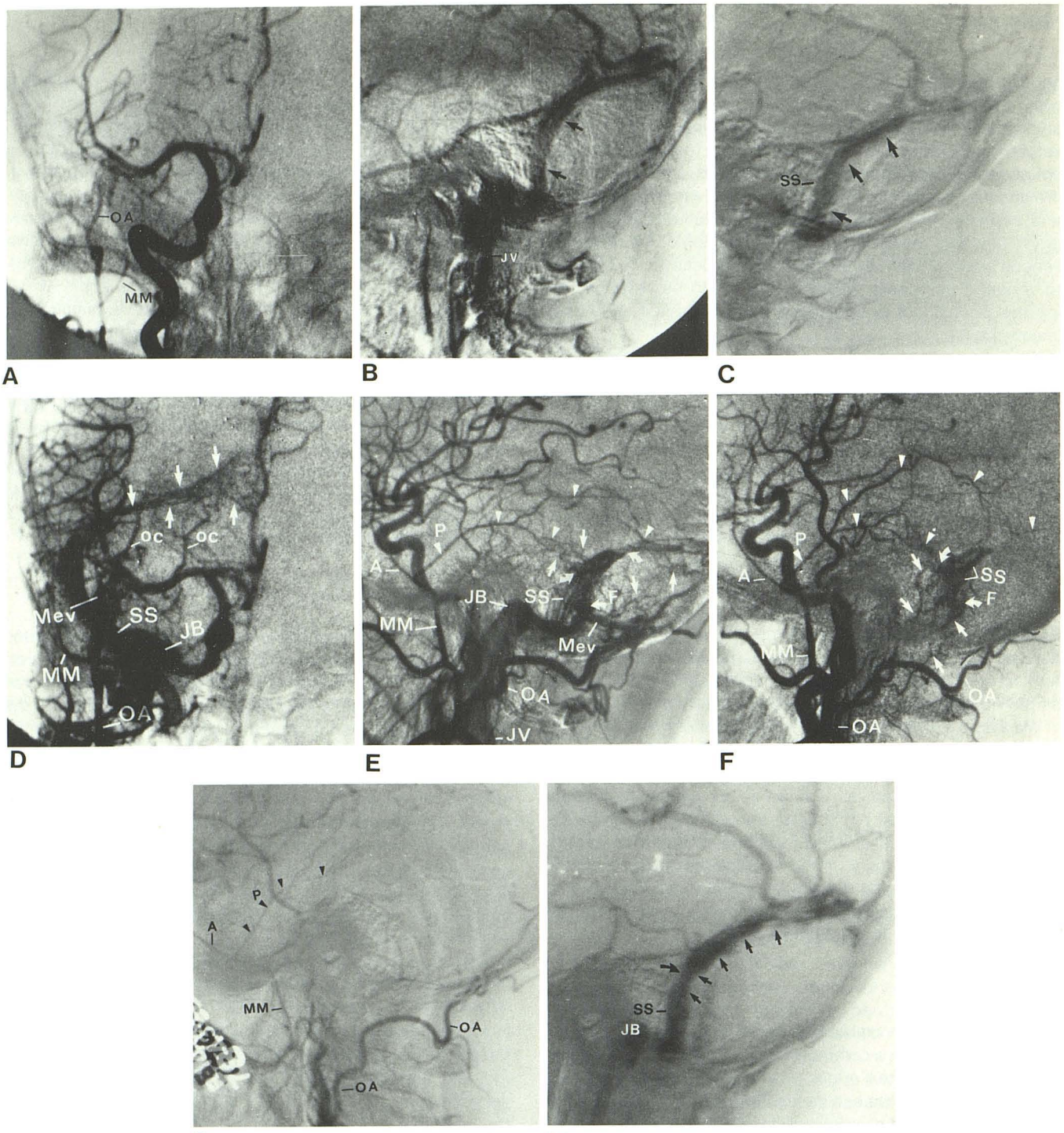

Fig. 1.—Case 1. Normal arterial **(A)** and venous **(B)** phases of right common carotid arteriogram 2 months before trauma. Occipital (OA) and middle meningeal (MM) arteries of normal size and course. Sigmoid sinus (*arrows*) and jugular vein (JV) are normally opacified. **C**, 1 week after injury. Irregular wall and lumen (*arrows*) of sigmoid sinus. Jugular vein not opacified. Arterial phase was normal. **D** and **E**, 6 months later. Hypertrophied middle meningeal (*arrowheads*) and occipital artery branches (OC) terminate directly into meningeal vascular network (*straight arrows*) in wall of lateral and sigmoid sinuses. This supplies multiple fistulous communications (F, *curved arrows*) in wall of sigmoid sinus (SS). Sigmoid sinus, mastoid emissary vein (Mev), and jugular vein (JV) are opacified early in arterial phase. JB = jugular bulb; A = anterior and P = posterior branches of middle meningeal artery. **F**, 1 week after surgery and 7 months after trauma. Meningeal branches of occipital artery ligated extracranially. However, fistulae (F, *curved arrows*) persist and are supplied by posterior branch of middle meningeal artery (*arrowheads*) and by a few transosseous meningeal branches of occipital artery (*straight arrows*). **G**, External carotid arteriogram 2½ years after trauma. Occipital and middle meningeal arteries of normal size. No AV shunt. **H**, Irregular sinus wall (*straight arrows*) and apparently stenotic lumen of sigmoid sinus (*curved arrow*) at site of previously demonstrated shunt.

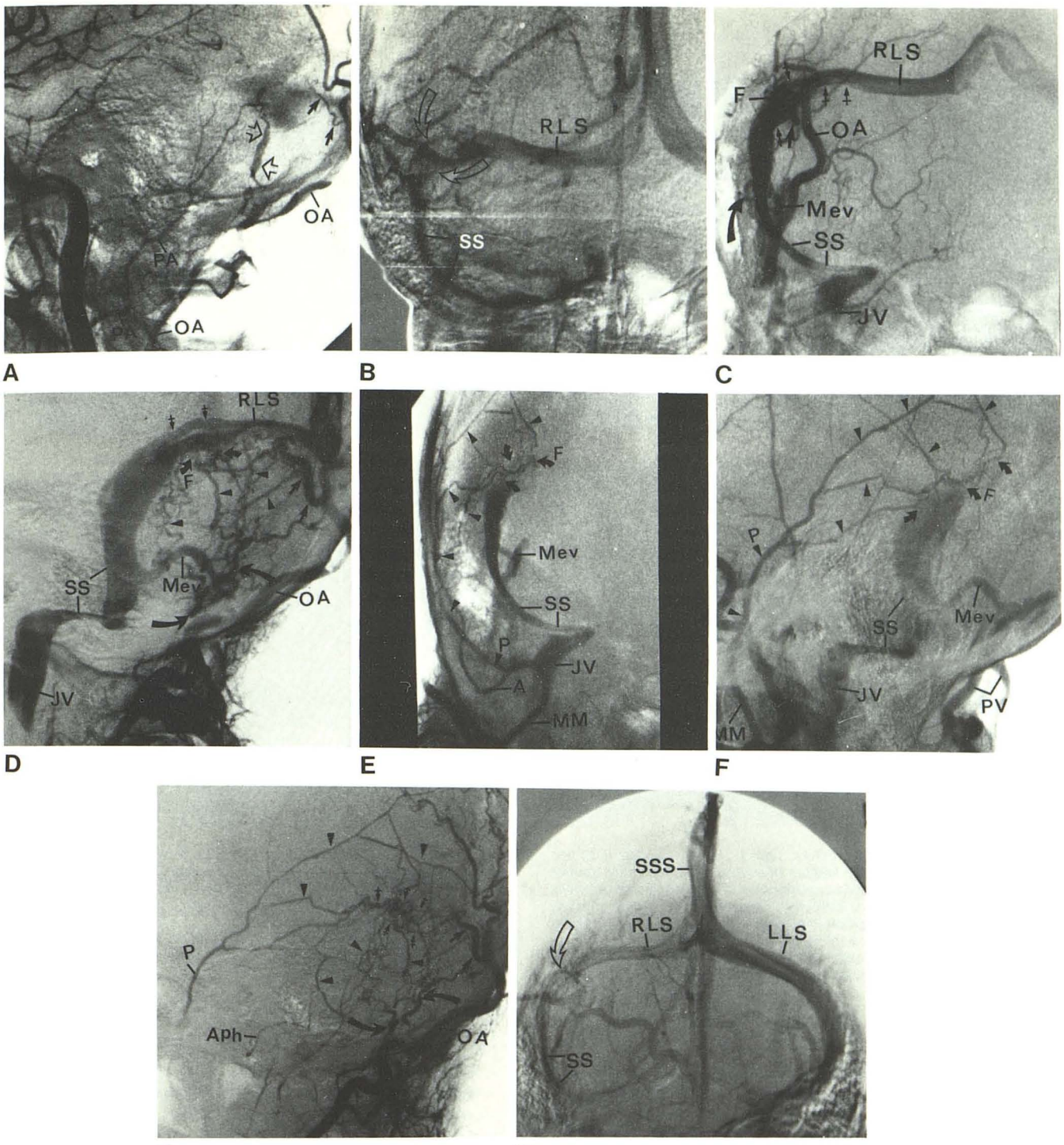

Fig. 2.—Case 2. **A**, 3 weeks after trauma. Posterior auricular artery (PA) supplies fistulous communication in scalp. Early appearing vein (*open arrows*) drains into paravertebral venous plexus. Normal occipital artery and meningeal branches (*solid arrows*). **B**, Anteroposterior view of venous phase. Right lateral sinus (RLS) is normal up to its bend. Filling defect in lumen (*arrows*) where sinus is crossed by diastatic lambdoidal suture. Sigmoid sinus markedly narrowed and stenotic. **C** and **D**, 6 months after injury. Selective arteriogram of right occipital artery. Hypertrophied occipital artery. Mastoid meningeal branches (*long curved arrows*) and transosseous meningeal branches (*straight arrows*) also enlarged and supply rich dural vascular network (*arrowheads*). Fistulae (F, *short curved arrows*) between meningeal vascular network and right lateral sinus. Lesion mimics malformation and consists of multiple AV shunts, indicated by numerous meningeal arteries terminating directly into lumen of right lateral sinus. Shunts at previously seen intraluminal defect (**B**). Sigmoid sinus (SS), mastoid emissary vein (Mev), and jugular vein (JV) opacified early in arterial phase. Wall is irregular (*crossed arrows*) with septation and narrowing of lumen of right lateral sinus. Sigmoid sinus also narrowed in transverse diameter. **E** and **F**, Right internal maxillary artery injection. Posterior (P) temporal convexity branch of middle meningeal artery (MM) is hypertrophied and supplies dural vascular network (*arrowheads*). Vascular network also supplies fistulous communications (*curved arrows*). Multiple meningeal arteries terminate directly into lumen of right lateral sinus. PV = paravertebral vein. **G**, Superselective right occipital arteriogram 1 month later (7 months after trauma). Fistulous communications have closed spontaneously. Occipital artery, its mastoid meningeal branches (*curved arrows*), and transosseous meningeal branches (*straight arrows*), although still hypertrophied, are relatively small in caliber. Meningeal vascular network still present, and there is retrograde opacification of posterior (P) temporal convexity branch of the middle meningeal artery and meningeal branch of ascending pharyngeal (Aph) artery, indicating free communication between various meningeal arteries. There is also a network of dural arteries (*crossed arrows*), perhaps representing newly formed vessels in wall of abnormal segment of lateral sinus. No opacification of the venous system. **H**, Right internal carotid injection with compression of left jugular vein. Small right lateral sinus (RLS) but previous irregularity of wall, septation, and defect in lumen (*arrow*) (**B–D**) are absent. Sigmoid sinus is still narrowed and stenotic. Despite compression of left jugular vein, major venous flow is into left lateral sinus (LLS), indicating compromise of lumen of right lateral and sigmoid sinuses.

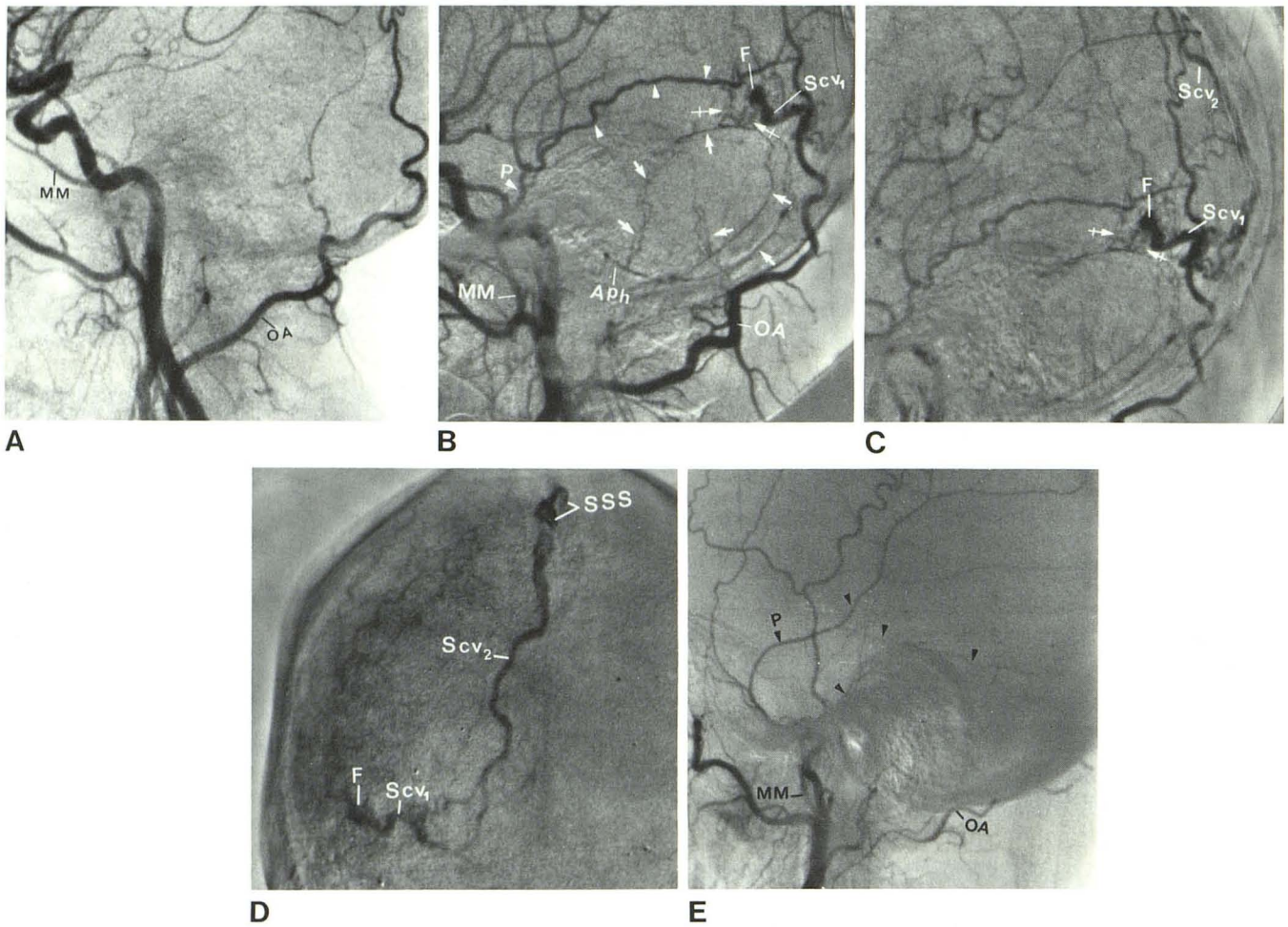

Fig. 3.—Case 3. **A**, 2 years before trauma. Normal occipital (OA) and middle meningeal (MM) arteries and no AV shunt. **B**, 3 weeks after trauma. Hypertrophied occipital and middle meningeal arteries. Meningeal branches of ascending pharyngeal artery (Aph), meningeal branches of occipital artery (*uncrossed arrows*) and of middle meningeal artery (MM) supply vascular network (*crossed arrows*) in dural wall of lateral sinus. Multiple small meningeal arteries from this network terminate into superficial cortical vein (Scv_1), which is opacified retrogradely through these shunts. Fistula (F) is where cortical vein normally enters sinus. Posterior branch of middle meningeal artery (P, *arrowheads*). **C** and **D**, Late arterial and capillary phases. Superficial cortical vein (Scv_1) drains into another cortical vein (Scv_2) through collateral veins. Cortical vein Scv_2 drains upward into superior sagittal sinus (SSS). Vascular network (*crossed arrows*). **E**, 2 years after trauma. Occipital artery and posterior temporal convexity branch (*arrowheads*) of middle meningeal artery (MM) are normal and there is no AV shunt. Selective occipital artery injection was also normal.

channel seems to precede the development of abnormal AV communications. One may speculate that similar thrombosis of developing venous channels during the embryologic period may be similarly responsible for brain parenchymal AV malformations. Selective, and, if possible, superselective, catheterization of the feeding arteries with magnification filming is the most appropriate way to study these lesions. Close scrutiny and a detailed review of the draining venous channels hold the key to understanding their topography and pathophysiology.

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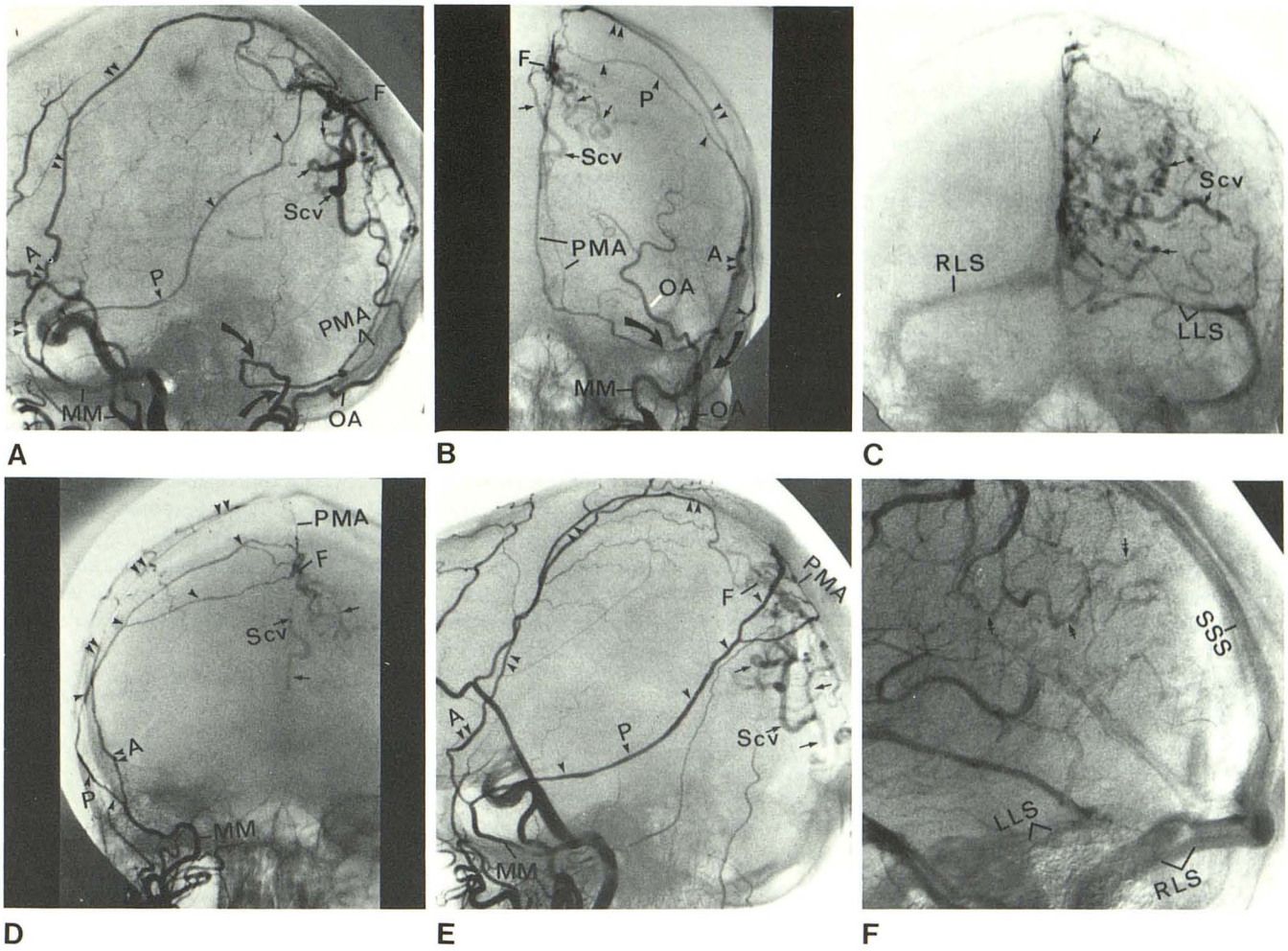

A B C

D E F

Fig. 4.—Case 4. **A** and **B**, Left external carotid arteriograms. Hypertrophied mastoid meningeal (*curved arrows*) branches of occipital artery (OA), anterior (A, *double arrowheads*), and posterior (P, *single arrowheads*) convexity branches of middle meningeal (MM) artery constitute paramedian meningeal artery (PMA), which supplies fistulous communications (F) in wall of superior sagittal sinus. Venous drainage into numerous superficial cortical (Scv, *straight arrows*) veins of left cerebral hemisphere. **C**, Same injection. Superficial cortical veins (*arrows*) of left cerebral hemisphere drain retrogradely into left lateral sinus. **D** and **E**, Right external carotid arteriograms. Anterior (*double arrowheads*) and posterior (*single arrowheads*) convexity branches of right middle meningeal artery also supply fistulous communications (F) through paramedian meningeal artery (PMA). Again, venous drainage is into superficial cortical veins of left cerebral hemisphere, which are opacified retrogradely. **F**, Venous phase of left internal carotid injection. Lack of superficial cortical veins apparent in left parietooccipital region and blood appears to drain from this area through collateral venous channels (*arrows*) into superior sagittal sinus (SSS), a clearly acquired feature.

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