

Ten cases of arteriovenous malformations located in critical areas of the cerebrum, i.e., motor and speech areas, the insular cortex, the corpus callosum, and the basal ganglia, were treated surgically by radical removal of the lesion. In four of the cases a neurologic examination before surgery was negative; five cases exhibited only mild hemiparesis or dysphasia; only one case exhibited hemiplegia and stupor. There were no postoperative deaths and a residual permanent deficit was noted in only one patient. Postoperative control angiograms failed to show any filling of the arteriovenous malformations in any of the cases. Recovery of the ability to work was achieved in all of the patients. The operating microscope and controlled hypotension made possible a precise resection at the edges of the lesion, and damage to surrounding important cerebral structures was thus avoided. Based on these results, microsurgery should be considered as an elective therapy for these kinds of arteriovenous malformations, even when preoperative symptoms are minimal.

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MICROSURGICAL TREATMENT OF TEN ARTERIOVENOUS MALFORMATIONS IN CRITICAL AREAS OF THE CEREBRUM

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The surgical treatment of arteriovenous malformations (AVMs) located in speech and motor areas, in the corpus callosum, and in the deep paraventricular structures continues to be a subject of controversy.^{4,11,12,14,16,20-24,27,29,31-35} This controversy is aroused by the fact that some of these AVMs cause only mild clinical impairment, while their surgical removal can result in possible permanent neurologic damage. In recent years, however, surgical experience in the removal of these particular types of AVMs has demonstrated the possibility of achieving excellent results in a percentage of cases so high that further development of this kind of surgery seems justified.^{1-3,9,12,13,19,21,26,33,36,37}

This article reports on 10 cases of AVMs that were located in motor and speech areas, in the insula, or in the midline structures, and discusses the indications for surgery, the surgical treatment, and the clinical results.

METHODS

We operated on these patients when a surgical approach appeared feasible. We were not deterred by the fact that in some cases the preoperative neurologic examination was normal or revealed only very minor deficits.

The surgical technique, suggested by Kunc,¹² was to proceed as close to the edges of the lesion as possible, in order to spare functionally important adjacent cerebral tissue and vessels.¹³ In three cases the "backward technique" of Per-tuiset²⁵ was also used, especially where the AVM had multiple draining veins.

Both the operating microscope and sodium nitroprusside-induced hypotension (deep or mean) are necessary for this type of surgery (for the specific technique of hypotension we refer the reader to a previous study⁵). The hypotensive blood pressure values were maintained above 50 mm of systolic arterial pressure in all of the cases, and thus were not as low as those used in aneurysm surgery. In addition, particular attention was paid to the total dose of sodium nitroprusside administered to the patient because of the toxic complications possible after the long periods of hypotension generally needed to complete the excision of the AVM.^{6,17,30} In the cases reported here, the total dose of sodium nitroprusside never exceeded 2 mg/kg of body weight,

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Table 1. Summary of the anatomic data in 10 cases of arteriovenous malformation (AVM).

Case	Sex	Age	Location of the AVM	Size	Arteries supplying the AVM
1	Male	44	Right motor area	Medium 3 × 3.5 × 2 cm	Right middle cerebral
2	Male	7	Left motor area	Medium 3 × 2 × 4.5 cm	Left middle cerebral Left lenticulostriate
3	Male	23	Left motor area	Large 8 × 6 × 5 cm	Left middle cerebral Left and right anterior cerebral
4	Male	35	Left motor area	Large 5 × 6 × 3 cm	Left middle cerebral Left lenticulostriate
5	Female	18	Right insula	Large 5 × 4 × 3 cm	Right middle cerebral Right lenticulostriate
6	Female	20	Left insula	Large 5 × 4 × 2.5 cm	Left middle cerebral Left posterior cerebral
7	Female	30	Speech area (angular gyrus)	Small 1.5 × 1 × 1 cm	Left middle cerebral
8	Female	14	Corpus callosum (pars media)	Small 2 × 1.5 × 1 cm	Left anterior cerebral
9	Male	37	Corpus callosum (genu)	Medium 3 × 2.5 × 1.5 cm	Left anterior cerebral
10	Female	39	Head of left caudate nucleus	Medium 3 × 4 × 4 cm	Left lenticulostriate

even in those few cases where hypotension had to be maintained for three or four hours.

Postoperative control angiograms were performed in all of the cases.

ANALYSIS OF THE CASES

From January 1976 to June 1978, 10 cases of AVMs located in functionally important cerebral areas were surgically treated by the senior author (R.DP.) in the Department of Neurosurgery of the Verona City Hospital. This series constitutes 20% of the total number of intracranial AVMs operated on in this department in the last seven years.

As shown in table 1, four of the AVMs were located in the motor area (three in the dominant hemisphere), two in the insula (one in the dominant hemisphere), one in the speech area, two in the corpus callosum, and one in the basal ganglia (left side). According to the classification of Mingrino,¹⁸ four of the AVMs were large (i.e., maximum diameter over 5 cm), four were medium (maximum diameter 2–5 cm), and two were small (less than 2 mm in diameter). Seven of the AVMs were fed by lenticulostriate arteries, and one AVM was fed by the posterior cerebral artery (PCA). Angiograms demonstrated that a

Table 2. Summary of the clinical data in 10 cases of arteriovenous malformation.

Case	Presenting history	Preoperative examination	Immediate postoperative course
1	Epilepsy	Left hemiparesis	Worsened transiently
2	SAH ^a Hematoma	Drowsiness dysphasia right hemiplegia	Improved
3	Epilepsy	Right hemiparesis	Worsened
4	Epilepsy SAH	Right hemiparesis	Unchanged
5	Epilepsy	Negative	Unchanged
6	Epilepsy	Negative	Worsened transiently
7	SAH Hematoma	Negative	Unchanged
8	SAH Hematoma	Right hemiparesis	Unchanged
9	SAH Hematoma	Drowsiness right hemiparesis	Improved
10	SAH	Negative	Worsened transiently

^aSAH = subarachnoid hemorrhage

single major arterial trunk fed the angioma in five cases, while multiple major trunks fed the angioma in the other five cases. The venous drainage in all of the cases flowed to the superficial sinuses, and in three cases it also flowed to the deep venous system. Five patients were males and five were females. Their ages ranged from 7 years to 44 years (average age: 26 years).

As shown in table 2, 6 patients had a history of subarachnoid hemorrhage (SAH), 5 cases had a history of epileptic seizures, and 3 cases had a history of intracerebral hematoma. There were no preoperative clinical disturbances in four cases and only minimal disturbances in another five cases (mild hemiparesis); only one case presented with a severe right hemisyndrome and marked impairment of consciousness.

There were no postoperative deaths, and only four cases suffered from transient neurologic disturbances (generally hemiparesis) (table 3). There was a permanent deficit in one case; at follow-up this patient was improving. Recurrence of symptoms was not observed in any of the five patients who had presented with epileptic seizures before the operation and who followed the prescribed antiepileptic therapy postoperatively. One patient who had not suffered any seizures preoperatively experienced one seizure 9 months postoperatively when phenobarbital therapy was interrupted. Her phenobarbital dose was resumed, and she reported no other seizures when she was last seen 10 months following surgery. Eight patients are now working full time or attending school, and two patients are working part time (the inability to work full time is a result of a preexisting neurologic deficit in one case and a result of new neurologic deficits in the second).

Postoperative control angiograms have demonstrated the exclusion of the AVM in all of the cases.

CASE REPORTS

Motor Area. Case 1. A 44-year-old male laborer experienced five episodes of sudden loss of strength and paresthesias in the left limbs, each lasting 5 min and occurring at intervals of many months over three years. An outpatient EEG revealed pathologic focal activity over the right hemisphere. A subsequent brain scan demonstrated hyperactivity of the radionuclide over the right parietal area.

On admission to our department, the patient was only mildly hemiparetic on the left. A computed tomography (CT) scan with contrast medium showed a hyperdense area in the right superior parietal lobe. A right carotid angiogram showed an AVM ($3 \times 3.5 \times 2$ cm), which was nourished by two hypertrophied branches of the middle cerebral artery (MCA), with one large vein draining toward the superior sinus and another vein draining toward the sphenoidal sinus. Still mildly hemiparetic, the patient was taken to surgery two weeks after admission.

After a parietal craniotomy was performed, it was possible to see the large cortical vein draining into the longitudinal sinus and one of the two arterial feeders. By following this artery, we encountered the angioma subcortically. The patient was placed under deep hypotension. After the two feeding vessels and the two draining veins were clipped close to the AVM, the angioma was excised. Bleeding was minimal and deep hypotension was required for only 40 min.

The patient experienced an increase of left hemiparesis, particularly in the upper limb, for a few days after surgery. Two weeks after surgery a control angiogram documented the exclusion of the AVM. At discharge of the patient the following day, the mild preoperative paresis persisted. At a follow-up examination six months later, the paresis continued to persist, and the patient was only able to return to work part time.

Case 2. The patient, a seven-year-old male, was asymptomatic until a day before admission, when he suffered a SAH, followed by aphasia and right hemiparesis.

On admission to our department, he was stuporous and aphasic, with right hemiplegia and marked neck stiffness. An immediate left carotid angiogram showed a medium-sized ($3 \times 2 \times 4.5$ cm) AVM located in the left motor area and directed medially. The AVM was fed by two rolandic branches of the MCA and by a large lenticulostriate artery of the lateral group. The venous system drained into the superior longitudinal sinus and the transverse sinus. There was a mild shift of the midline vessels to the right, with separation of the anterior parietal branches of the MCA, suggesting a left parietal intracerebral hematoma. It was decided to postpone any operation until the patient's condition improved. A CT scan, one week after admission, showed a hyperdensity in the left parietal area, suggesting

Table 3. Summary of preliminary and follow-up data.

Case	Duration of intra-operative hypotension	Preoperative seizures and medication	Postoperative seizures and medication	Postoperative angiogram	Preoperative occupation	Postoperative occupation	Length of follow-up
1	40 min	3 in 5 yrs No medication	0 in 9 mos Diphenylhydantoin (Dilantin) 100 mg/day; phenobarbital 100 mg/day	No evidence of residual AVM at 2 weeks ^a	Laborer	Part time 6 mos after discharge	9 mos
2	2 hrs	0	0 Phenobarbital 100 mg/day	No evidence of AVM at 2 weeks	Student	Returned to school 3 mos postop	2 yrs
3	3½ hrs	6 in 1 yr Phenobarbital 200 mg/day	0 Dilantin 200 mg/day Phenobarbital 100 mg/day	No filling of AVM Diffuse cerebral edema at 2 days	Unemployed	Unemployed but working at home	8 mos
4	1½ hrs	4 in 27 yrs Mysoline 750 mg/day	0 Dilantin 200 mg/day Phenobarbital 100 mg/day	No filling of AVM at 2 days	Farmer	Farmer 3 mos postop	13 mos
5	2 hrs	Daily focal seizures; 2 grand mal in 6 yrs Mysoline 750 mg/day	0 in 9 mos Mysoline 500 mg/day Phenobarbital 100 mg/day	No evidence of AVM at 2 weeks	Optician	Resumed same occupation 2½ mos after discharge	9 mos
6	3 hrs	5 grand mal in 10 days Dilantin 150 mg/day Phenobarbital 150 mg/day	0 in 22 mos Dilantin 200 mg/day Phenobarbital 150 mg/day for 15 mos, then phenobarbital 100 mg/day	No evidence of AVM Mild shift to right at 3 weeks	Knitter	Knitter 4 mos after discharge	13 mos
7	0	0	1 grand mal at 9 mos	No evidence of AVM at 1 week	Trader	Trader 2 mos postop	10 mos
8	50 min	0	0 Valium 10 mg/day	No evidence of AVM at 2 weeks	Student	Returned to school 4 mos postop	17 mos
9	0	0	0 Phenobarbital 100 mg/day	No evidence of AVM at 19 days	Laborer	Back to work 6 mos; tennis 15 mos postop	15 mos
10	4 hrs	0	0 Phenobarbital 100 mg/day	No evidence of AVM at 12 days	Housewife	Resumed work 2 mos; fine knitting 21 mos postop	21 mos

^aAVM = arteriovenous malformation.

the persistence of the hematoma. The patient improved gradually following symptomatic treatment and was taken to surgery 25 days after admission. At this time he was awake but dysphasic and hemiparetic.

The AVM was approached through a left parietal craniotomy. A large draining vein was found, but the angioma was not immediately

visible. By following this vein, the angioma, surrounded by a small hematoma, was located subcortically. The AVM was progressively excised after all MCA feeders near the lesion were clipped. On proceeding deeper, we found and coagulated the feeder from the large lenticulostriate artery. After the two large draining veins were clipped, the AVM was removed completely.

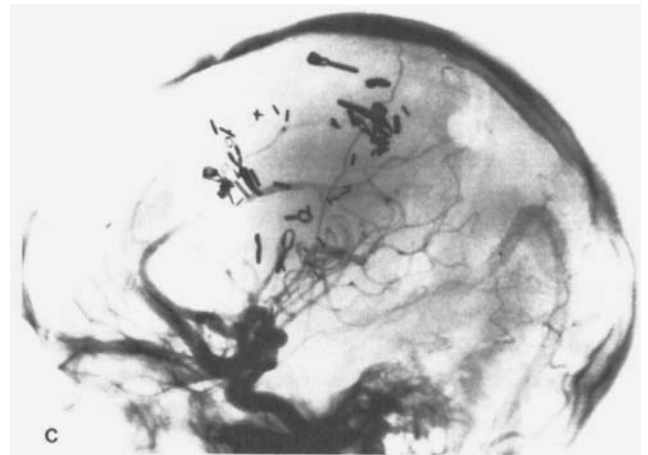
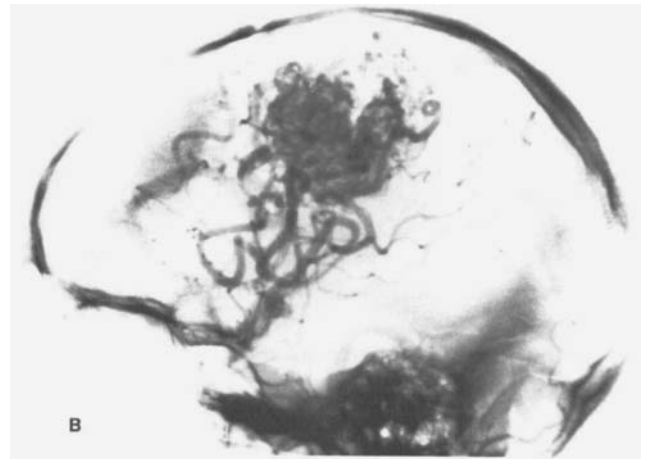


Figure 1. Left carotid angiograms in case 3. (A) Anteroposterior view. (B) Lateral view. Note the huge AVM involving the left motor area. (C) Postoperative lateral angiograms, after resection of the AVM, showing clips at the edges of the lesion.

The operation was performed with the patient under mean hypotension; there were no problems in controlling bleeding.

Following surgery the patient did very well. A control angiogram, performed 14 days after surgery, demonstrated the exclusion of the AVM. The patient was discharged the following day with persistent but improving mild right hemiparesis and motor dysphasia. At a follow-up examination two years later, the patient was successfully attending school and he had no residual neurologic deficits.

Case 3. A 23-year-old male had been complaining of dizziness and disturbances of gait since his childhood. One year before admission he had experienced a jacksonian seizure involving the right half of the body and the right leg and lasting a few minutes. Four similar epileptic seizures were noted in the following months. In May 1978, following another episode, a residual right hemiparesis was noted.

On admission to our department, the patient

was only mildly hemiparetic and had no other neurologic signs. A carotid angiogram (fig. 1A and B) revealed a huge AVM ($8 \times 6 \times 5$ cm) involving the left motor area and the left prefrontal area, which was supplied by numerous branches of the left MCA and by both anterior cerebral arteries (ACAs) (the left ACA being huge). The venous drainage flowed toward the superior sagittal sinus and the middle cerebral vein through an enlarged vein of Trolard. Fifteen days after admission the patient underwent surgery when still mildly hemiparetic.

After a wide left frontotemporoparietal craniotomy was completed, an extensive network of vessels was noted overlying the cortex. This network was composed of feeders from the MCA, a large vein draining into the sphenoidal sinus, and other large veins running to the longitudinal sinus. By dissection along the inter-hemispheric fissure, both ACAs were exposed and their feeding branches were clipped very close to the malformation. After the patient was

placed under deep hypotension, we decided to approach the angioma with a "backward" technique because identification of the MCA feeders was very difficult. First, almost all of the veins draining into the sagittal sinus were clipped; there was no swelling of the angioma. By following the veins, we clipped the MCA feeders close to the AVM and gradually excised the malformation. The residual large vein draining to the sagittal sinus was then closed, and the angioma was totally removed. It should be noted that the angioma reached the ventricular wall, where control of bleeding was particularly difficult. The procedure took more than 9 hours and deep hypotension was maintained for 3½ hours.

The patient had a stormy postoperative course—stupor, aphasia, and severe right hemiparesis appeared immediately after surgery. A carotid angiogram performed two days after surgery (fig. 1C) and a CT scan performed four days after surgery suggested the presence of diffuse cerebral edema, but there were no signs of intracerebral hematoma; the AVM had completely disappeared. The patient was treated symptomatically and a progressive improvement in his neurologic condition was noted from the second week after surgery. At discharge five weeks after surgery, the dysphasia had disappeared and only a right hemiparesis persisted. At follow-up three months after surgery, the patient was partially working and able to walk unassisted.

Case 4. This patient, a 35-year-old male, suffered from a generalized epileptic seizure when he was 7 years old. Eight months before admission to our department, he experienced a subarachnoid hemorrhage followed by dysphasia and right hemiparesis. He was referred to another hospital, where a diagnosis of a parietal AVM was made from angiograms. No surgical treatment was undertaken. Following another SAH, the patient was admitted to our department, mildly hemiparetic on the right and suffering from neck stiffness. A complete angiographic study (fig. 2A, B, and C) showed a large AVM (5 × 6 × 3 cm) involving the left motor area and left prefrontal area (especially the frontoparietal operculum) and reaching the caudate nucleus medially. The AVM was fed by frontoparietal opercular branches of the MCA, by a large lenticulostriate artery (lateral group) and by a few small branches of the left ACA. The very extensive venous system drained toward the

sagittal sinus, the middle cerebral vein, and the internal cerebral vein. Three weeks after admission the patient underwent surgery, when still mildly hemiparetic.

The approach was through a left frontotemporoparietal craniotomy. A large malformation was found overlying the motor cortex, with huge draining veins departing from it in all directions. The AVM was approached while the patient was under deep hypotension. The superficial MCA feeders, with some draining veins, were closed separately with clips. The dissection proceeded precisely along the edges of the AVM. As the surgery advanced into the underlying white matter, two large feeders from the MCA and a hypertrophied lenticulostriate artery were closed with clips. After the exclusion of the remaining large draining veins (especially a huge vein of Trolard), hypotension was discontinued. The recovery of normal blood pressure, however, caused diffused bleeding from various small vessels, especially in the paraventricular area. Complete control of hemorrhage was achieved by clips and bipolar coagulation.

The postoperative course was uneventful, but a mild right hemiparesis persisted. This was still evident at discharge. A postoperative control angiogram (fig. 2D and E), performed two days before discharge, showed the complete exclusion of the AVM. At a follow-up examination one year later, the patient had returned to work, and was suffering only residual hyperreflexia of the right limbs.

The Insula. *Case 5.* An 18-year-old female optician experienced a generalized epileptic seizure at the age of 12 years. In the following years, she complained of many episodes of tonic seizures, with no loss of consciousness, involving the left face and upper limb. Another generalized epileptic seizure occurred at the age of 15 years. Because of the frequency of the tonic episodes, the patient was admitted to another hospital for a complete workup. A brain scan showed hyperactivity of the radionuclide over the right temporoparietal area. A bilateral carotid angiogram suggested the presence of a large AVM in the parietal area.

When transferred to our department one month later, the patient was neurologically negative. A CT scan with contrast medium performed at admission showed a marked hyperdensity in the right temporal area. Five days later, a complete angiographic study revealed a

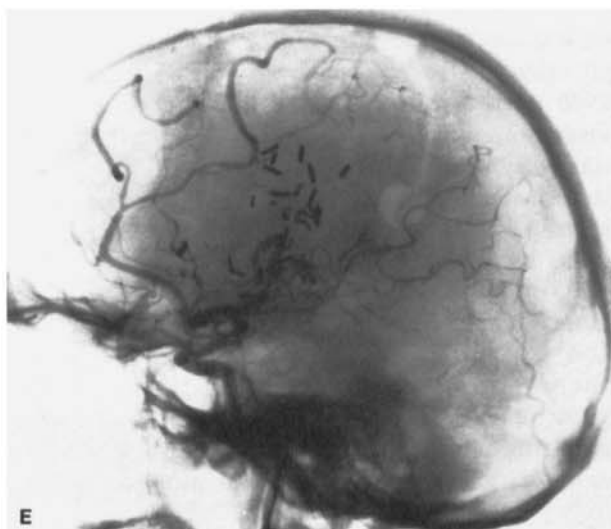
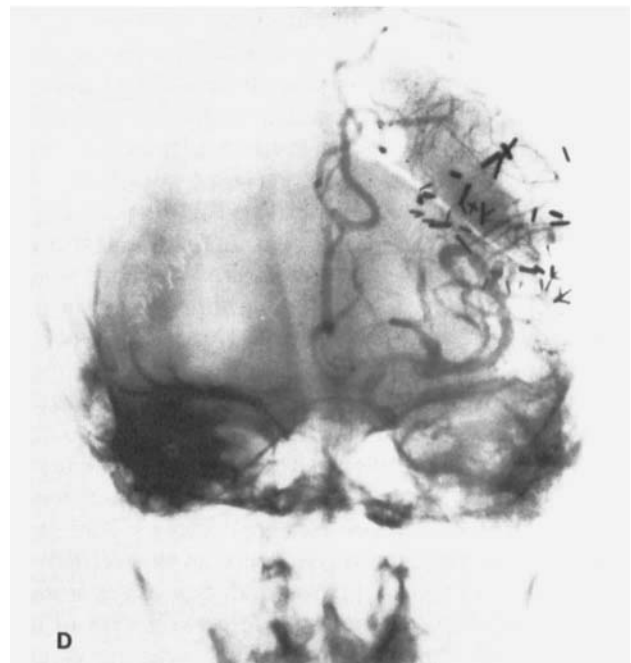
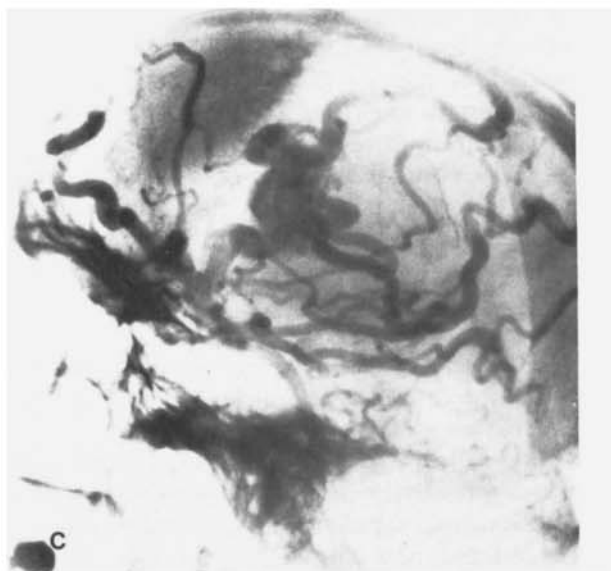
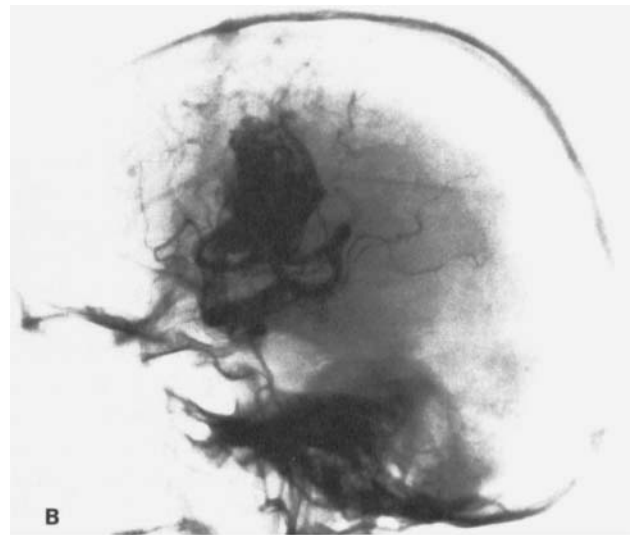
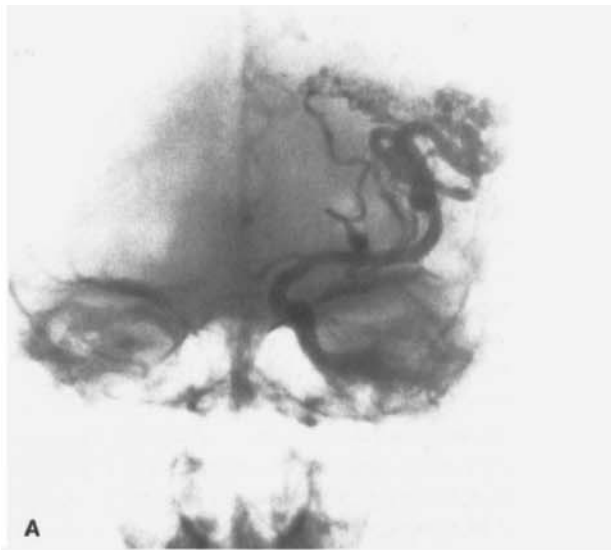


Figure 2. Left carotid angiograms in case 4. (A) Anteroposterior view. The AVM reaches the apex of the caudate nucleus, where it is fed by a large lenticulostriate artery. (B) and (C) Lateral views. The large AVM involves the frontoparietal operculum and has extensive venous drainage. (D) and (E) Postoperative angiograms, anteroposterior and lateral views after resection of the AVM.

large AVM ($5 \times 4 \times 3$ cm) involving the insular cortex and the corona radiata underlying the pre- and postcentral gyrus, with a medial part reaching the caudate nucleus. The vessels feeding the malformation were the insular branches of the MCA and a few lenticulostriate arteries of the lateral group. The venous system drained toward the superior longitudinal, the transverse, and the sphenoidal sinuses. One week after admission, the patient underwent surgical removal of the AVM.

After a large right frontotemporoparietal craniotomy had been performed, a huge arterialized vein of Trolard was found over the cortex. By dissecting deeply into the sylvian fissure, it was possible to expose the angioma, which was located in the insular cortex and was fed by two branches of the MCA. After the patient was placed under deep hypotension, collapse of the huge vein of Trolard was noted. The two feeding vessels were then closed with small clips and the angioma was dissected gradually from the surrounding tissue. As the surgery proceeded deeper, the lenticulostriate arteries supplying the angioma were coagulated and a deep draining vein was closed with a clip. After the temporal horn of the lateral ventricle was opened, it was also possible to close the venous drainage underlying the vein of Trolard and to remove the angioma completely. Deep hypotension was maintained for two hours. Following recovery of blood pressure to normal, the deep draining vein began bleeding and required careful hemostasis.

Following surgery, the patient experienced mild drowsiness for several days. The subsequent postoperative course was uneventful. A control angiogram performed two days before discharge showed the complete exclusion of the AVM. At discharge, the patient was neurologically negative and symptom-free. At a follow-up nine months later, she had returned to her work as an optician and had no neurologic complaints.

Case 6. This 20-year-old female was asymptomatic until 15 days before admission, when she experienced five generalized tonic seizures over a period of a few days. A brain scan made before admission showed hyperactivity of the radionuclide in the left superficial temporal area.

On admission to our department, the patient's neurologic examination was negative. A complete angiographic study (fig. 3A, B, and C)

revealed a large AVM involving the left insula, supplied by two insular branches of the MCA and two small temporal branches of the PCA. The venous system drained into the superior sagittal sinus and the superior petrosal sinus. Two weeks later the patient underwent surgery.

The operation proceeded through a left temporal craniotomy. Large venous channels were seen over the temporal cortex. By dissecting toward the underlying insular cortex, it was possible to visualize the malformation, which received two small feeding vessels from the MCA. With the patient under deep hypotension, these feeders were clipped close to the AVM, and the dissection advanced medially, after exclusion of the large venous drainage system that flowed to the petrosal sinus. The small feeding vessels from the PCA were located last and closed with clips. After the veins draining to the sagittal sinus were closed, the malformation was removed in toto and the lateral ventricle was opened at the trigone. Deep hypotension allowed the surgeon to keep the blood loss within safe limits (the patient required 2 liters of transfused blood). The control of bleeding was particularly difficult in the paraventricular area, where hemostasis had to be continued for more than 2 hours. Surgery in this case lasted for more than 10 hours, and hypotension was maintained for 3 hours.

The patient developed severe dysphasia and right homonymous hemianopia on the day after the operation. A CT scan five days after surgery showed a mild ventricular shift toward the right with a hypodensity in the left temporal region. The patient was treated symptomatically for cerebral edema and showed a progressive improvement of speech in the next weeks. A control angiogram (fig. 3D and E) three weeks after surgery demonstrated the complete exclusion of the AVM with a minimal shift to the right. At discharge 22 days after surgery the patient was markedly improved, with only a mild dysphasia. At follow-up 13 months later, the patient was neurologically negative and had returned to her job as a knitter.

Speech Area. *Case 7.* A 30-year-old female suffered a SAH followed by dysphasia two weeks before admission to our department. A left carotid angiogram, performed in another hospital, demonstrated a small AVM ($1.5 \times 1 \times 1$ cm) located in the speech area (left angular gyrus).

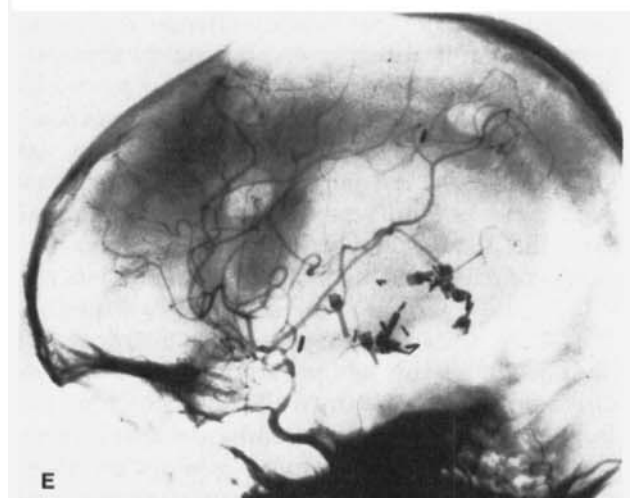
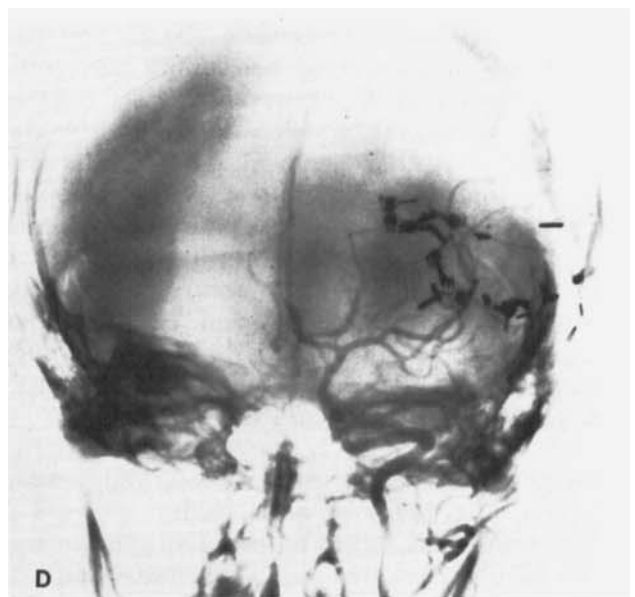
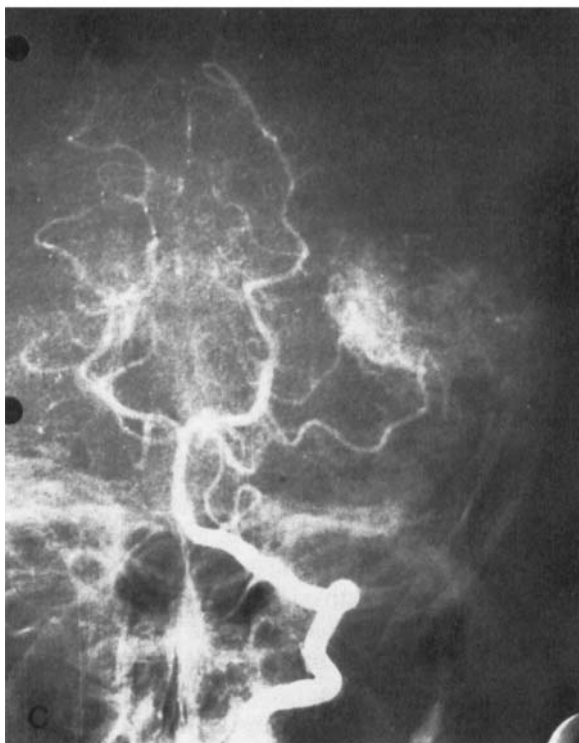
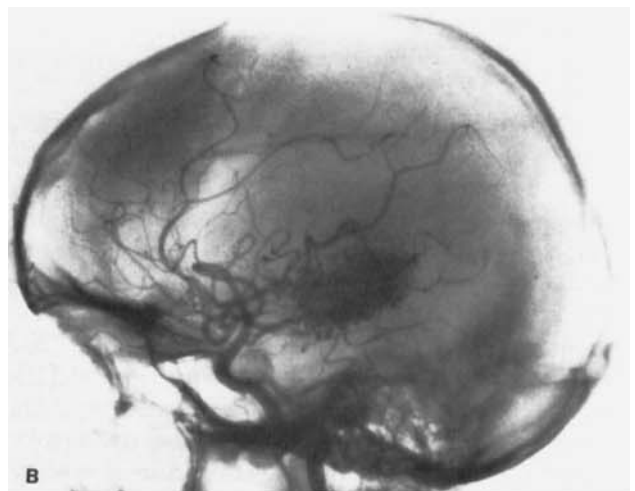
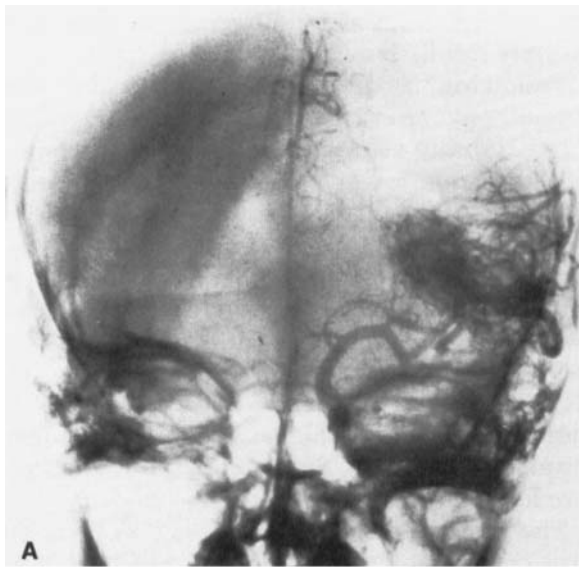


Figure 3. Left carotid angiograms and left vertebral angiograms in case 6. (A) Anteroposterior view; (B) lateral view; (C) anteroposterior vertebral view. The large AVM involving the left insula is fed by branches of the middle cerebral and posterior cerebral arteries. (D) Anteroposterior and (E) lateral views of postoperative angiograms after resection of the lesion. Note the microclips near the paraventricular area, which were needed for complete hemostasis.

This AVM was fed by a branch of the MCA with a large vein draining toward the longitudinal sinus.

On admission to our department, the patient was neurologically negative, with no neck stiffness. Three days later, a CT scan revealed a hyperdensity in the left posterior temporo-parietal area, suggesting an intracerebral hematoma. Two days later the patient was taken to surgery.

After a left temporoparietal craniotomy had been performed, the large draining vein and the artery feeding the angioma were seen over the angular gyrus. On dissection below the artery, many small branches departing from it and directed to the angioma were exposed and clipped separately. The angioma was progressively removed from the surrounding tissue, and the underlying intracerebral hematoma was completely suctioned. An aneurysm-like sac (visible on the angiogram), which was drained by the large vein, was then removed; the vein was ultimately clipped. Hypotension was not employed.

Following surgery, the patient recovered completely and had an uneventful postoperative course. A CT scan and a control angiogram showed the absence of the lesion. Ten days after surgery the patient was discharged, neurologically negative. At a follow-up ten months later, she was again working and symptom-free.

Corpus Callosum. *Case 8.* A 14-year-old female student, previously in good health, suffered a typical SAH, which was followed by aphasia and right hemiparesis. She was immediately admitted to our department.

On examination, she was lethargic and exhibited a severe right hemisindrome. A bilateral carotid angiogram (fig. 4A, B, and C) showed a small AVM ($2 \times 1.5 \times 1$ cm) located in the left gyrus cinguli and partially in the pars media of the corpus callosum. The malformation was supplied by the left pericallosal and callosomarginal arteries and was drained by a single vein into the superior sagittal sinus. There was a mild contralateral displacement of the left ACA under the falx and inferior displacement of the MCA, findings suggesting an intracerebral hematoma in the left parietal area. It was decided not to operate until the neurologic picture improved. A progressive reduction in the size of the intracerebral hematoma was noted by repeated CT scans. The patient was taken to sur-

gery four months after suffering the SAH and was only mildly hemiparetic on the right.

Following a left parietal craniotomy and immediately after the dura was opened, the single draining vein was found at its confluence with the superior sagittal sinus in the motor area. By following the vein, it was possible to reach the gyrus cinguli overlying the pars media of the corpus callosum where the small AVM could be exposed. After the vein was closed (with no consequent swelling of the angioma), the lesion was dissected free until the two feeding vessels from the left ACA were exposed. These were closed with clips and the malformation was then completely removed. Signs of an old hematoma were found around the AVM.

The postoperative course was uneventful, and a control angiogram (fig. 4D) one day before discharge showed the complete exclusion of the AVM. At discharge the patient was still mildly hemiparetic, but able to walk without assistance. At the follow-up 17 months later, the patient was again attending school with good results and had no residual neurologic deficits.

Case 9. This patient, a 37-year-old male laborer previously in good health, experienced sudden aphasia and right hemiparesis followed by a typical SAH four days before admission to our department. He was transferred to neurosurgery from the neurological service.

At admission, he was mildly stuporous, with marked neck stiffness, mild dysphasia, and right hemiparesis. A complete angiographic study (fig. 5A and B) revealed a medium-sized AVM ($3 \times 2.5 \times 1.5$ cm) located partly in the genu of the corpus callosum and partly in the left anterior frontal lobe. This AVM was supplied by the left frontopolar artery, with one large vein draining into the superior longitudinal sinus and another one into the vein of Labbé. There was a marked shift of the midline vessels toward the right, suggesting a left frontal intracerebral hematoma. The CT scan confirmed the presence of the shift and of the large left frontal hematoma. Three days after admission the patient was taken to surgery drowsy and hemiparetic.

After a left frontotemporal craniotomy, a small incision was made over the cortex of the anterior frontal lobe for partial drainage of the large hematoma in the underlying tissue. As the surgery proceeded toward the midline, it was possible to see the angioma, which had a large vein lateral to it draining toward the group of

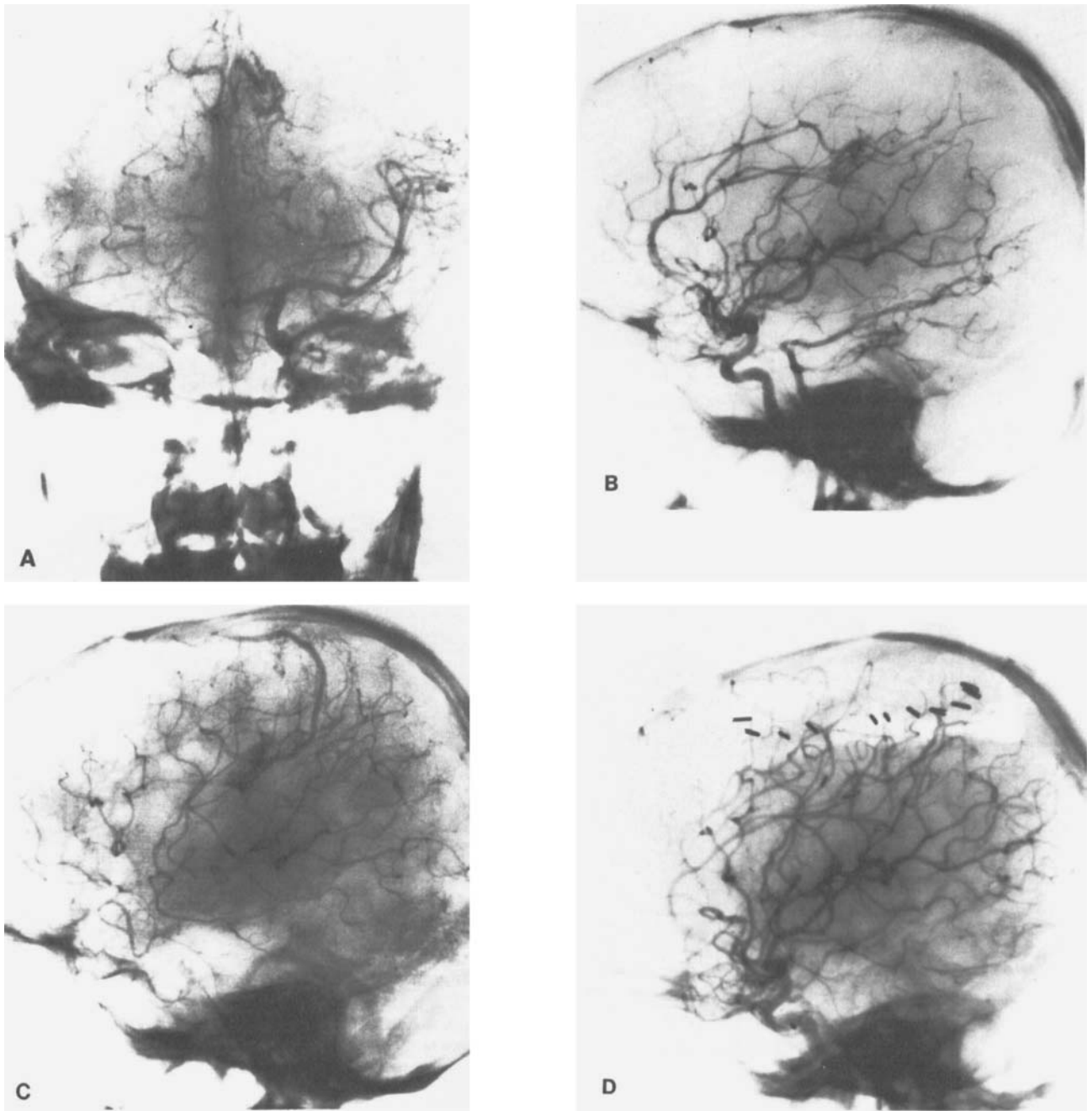


Figure 4. Left carotid angiograms in case 8. (A) Anteroposterior view; (B) and (C) lateral views. The small AVM is located in the gyrus cinguli and pars media of the corpus callosum. Note the single draining vein that was coagulated first using a "backward" technique. (D) Postoperative lateral angiogram after resection of the AVM. The pericallosal and callosomarginal arteries were preserved.

sylvian veins. By dissection into the interhemispheric fissure and after limited opening of the falx, the left frontopolar artery, with small branches departing from it toward the angioma, was exposed. These vessels were all clipped near the lesion. The AVM did not collapse, however,

because of the presence of other feeding vessels from the frontopolar artery, which were visible after both of the ACAs in the interhemispheric fissure had been exposed. It was thus decided to close the frontopolar artery directly with a clip. After collapse of the AVM, the lesion was re-

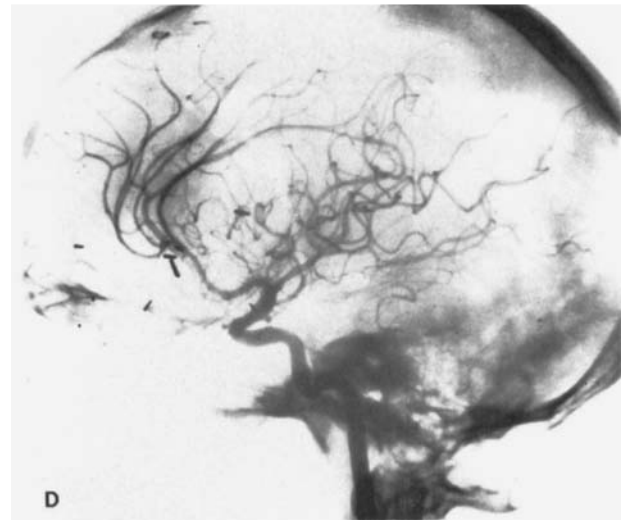
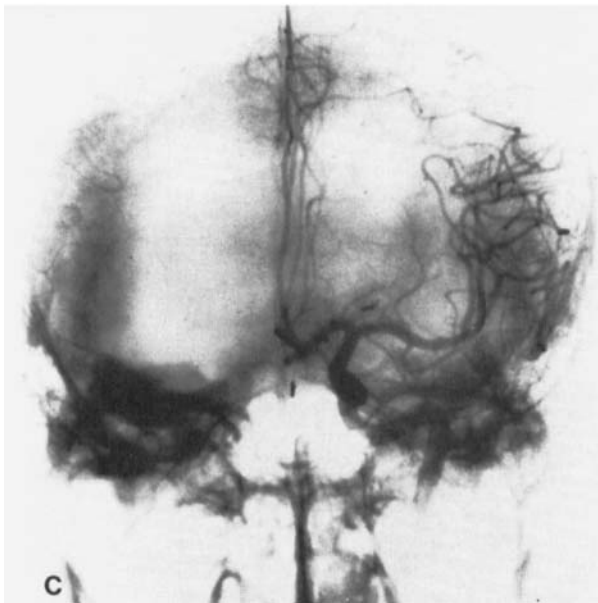
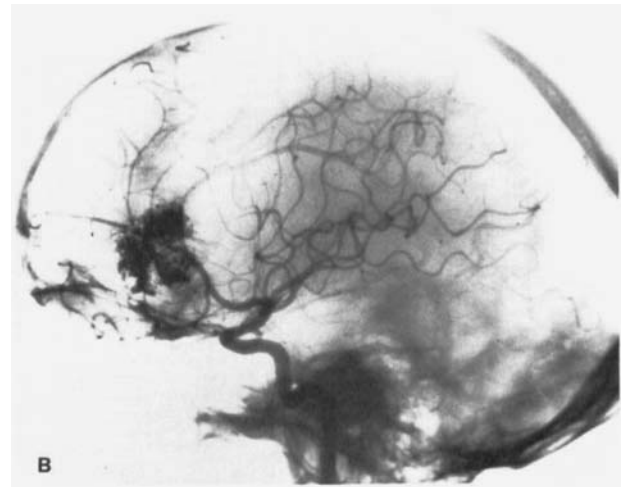
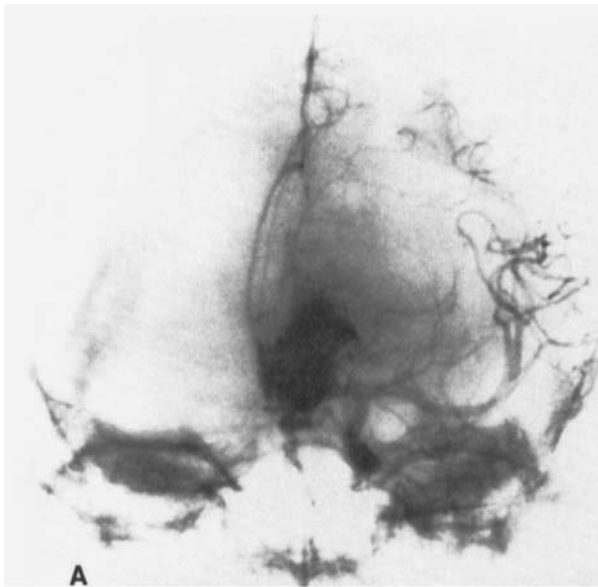


Figure 5. Left carotid angiograms in case 9. (A) Anteroposterior view; (B) lateral view. The AVM involves the genu of the corpus callosum and the left anterior frontal lobe. (C) Postoperative anteroposterior view and (D) lateral view.

moved completely, and the two large draining veins were closed with clips.

The patient recovered from surgery but experienced an increase in lethargy one day postoperatively. A CT scan showed only minimal signs of hematoma with a mild increase in ventricular shift; this was most likely the result of cerebral edema. The patient was followed carefully, and the neurologic picture progressively improved from the second week after surgery. Sixteen days after surgery no ventricular shift could be discerned on the CT scan. A control an-

giogram (fig. 5C and D) three days later showed the complete exclusion of the AVM, with no residual shift. At discharge four weeks after surgery the patient was fully awake and neurologically negative. At follow-up six months later, he had returned to work and had no neurologic complaints.

Basal Ganglia. Case 10. A 39-year-old female housewife was admitted to our department in March 1976 for a SAH with aphasia and right hemiplegia. At that time a carotid angiogram

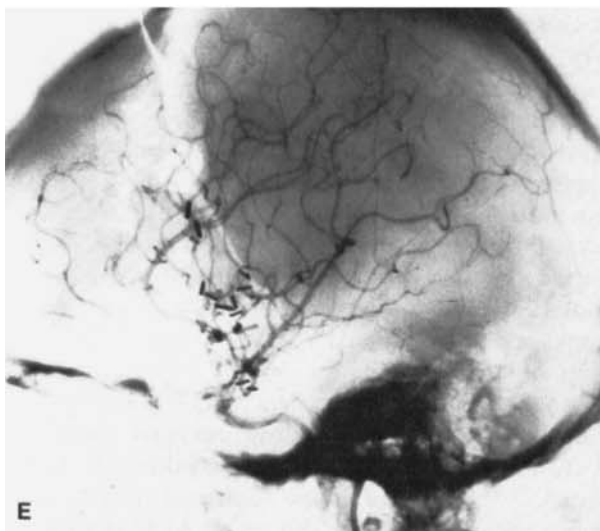
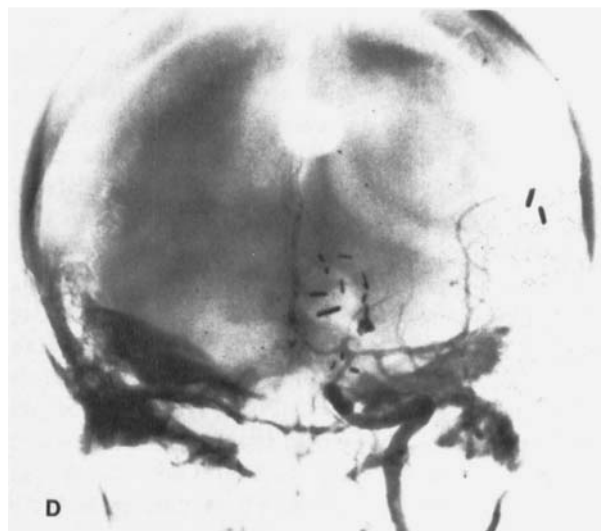
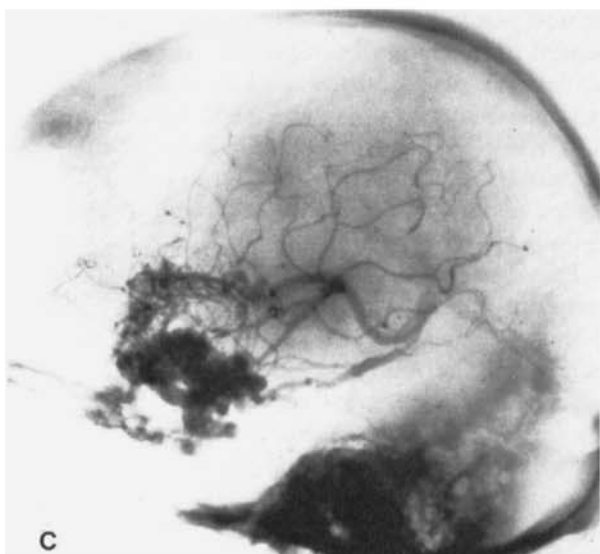
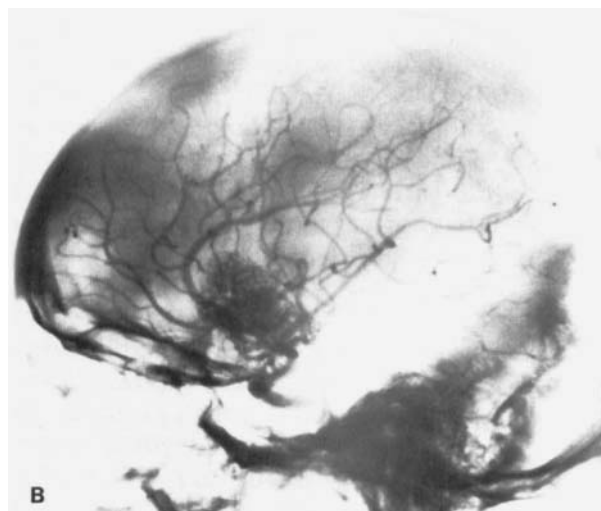
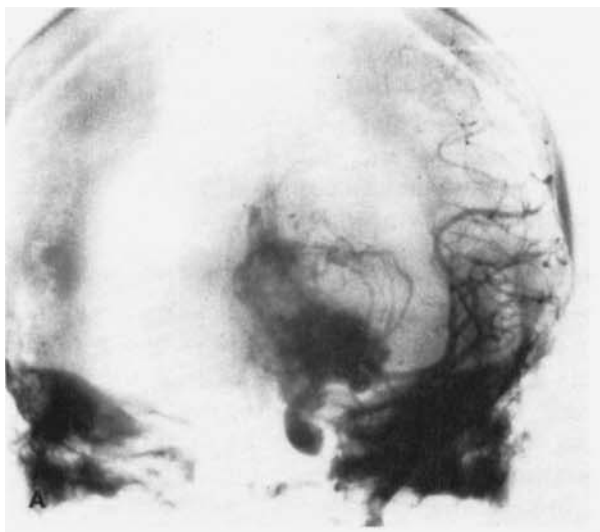


Figure 6. Left carotid angiograms in case 10. (A) Anteroposterior view; (B) and (C) lateral views. The AVM involves the left basal ganglia. Note the feeders from the medial group of lenticulostriate arteries and the drainage through Rosenthal's vein and the internal cerebral vein. (D) Anteroposterior and (E) lateral views of the postoperative angiograms.

(fig. 6A, B, and C) revealed a medium-sized AVM ($3 \times 4 \times 4$ cm) involving the left basal ganglia (especially the head of the caudate nucleus). It was supplied by the medial group of the lenticulostriate arteries. The venous drainage was directed to the sphenoidal sinus, to Rosenthal's vein, to the internal cerebral vein, and to the superior longitudinal sinus. Because of the particular location of the AVM, the patient did not undergo operation; she was discharged one month later, still hemiparetic. Following a second SAH with aphasia and right hemiplegia in April 1977, however, the patient was readmitted.

On examination she was stuporous and aphasic and exhibited a severe right hemiparesis, especially involving the upper limb. Five weeks later, after a slow but progressive improvement, she was taken to surgery. At that time she was neurologically negative.

The malformation was approached through a left frontopterional craniotomy. Three large veins were visible on the cortex, all draining into the longitudinal sinus. During exposure of the left optic nerve, it was noted that the internal carotid artery was covered by draining veins coming from the sylvian fissure and from the gyrus rectus. By enlarging the exposure through the sylvian fissure, other large veins were noted covering the initial tracts of the left MCA and ACA and draining toward the sphenoidal sinus. The anteromedial part of the frontal lobe was then resected, and two of these large veins were closed. On proceeding toward the corpus callosum and the gyrus rectus, it was possible to see the left ACA and to open the frontal horn of the lateral ventricle, whose anterior wall was covered by anomalous vessels. With the patient under deep hypotension, the malformation was finally approached. It was dark-colored and surrounded by an old hematoma. The many small vessels supplying the angioma (terminal branches of the lenticulostriate arteries) were individually clipped or coagulated. As the surgery advanced deeper in this direction, it was possible to close the three large veins draining toward the midline venous system with small clips. After a thorough inspection of the feeding and draining vessels close to the angioma, the veins covering the carotid artery and the first part of the ACA and MCA were closed. Deep hypotension was maintained for four hours, and surgery lasted nine hours.

Postoperatively, only transient dysphasia and mild right hemiparesis were noted for a few

days after surgery. A control angiogram (fig. 6D and E) 12 days after surgery showed the complete exclusion of the angioma. Two weeks after surgery the patient was discharged with no apparent neurologic deficits. At follow-up 14 months later, she had resumed her duties in the home and was symptom-free.

DISCUSSION

The surgical treatment of AVMs located in functionally important cerebral areas was limited, until a few years ago, to patients exhibiting severe or progressively worsening neurologic signs, or to patients with huge intracerebral hematomas.¹⁰ The fundamental consideration for broadening the surgical approach to these malformations is based on the fact that an AVM is a congenital lesion composed of nonfunctioning nervous tissue.^{8,13,15,22} Therefore, in the AVMs located in functionally important cerebral areas, the neurologic functions are located in the cerebral tissue surrounding the AVM and not in the AVM itself.^{13,28} Therefore, the removal of these particular AVMs is possible with a reasonable degree of safety, provided that the surrounding nervous tissue is spared.

Previous experience and this report document the possibility of removing AVMs located in these cortical areas, with excellent results, even when the AVMs are quite large. Thus, the indications for surgical removal of these AVMs can be extended to cases with a negative preoperative clinical picture. In our series, four patients were neurologically negative before the operation and another four patients were only mildly hemiparetic.

We consider Kunc's technique, i.e., to excise as close to the edges of the AVM as possible, a very useful one. However, this technique does present problems related to the severity of hemorrhage: in fact, the arterial feeders at the edges of an AVM often send off their branches to very fragile venous lakes. A significant hemorrhage in this area could force the surgeon to use excessive coagulation to stem the flow, with consequent damage of the surrounding functional nervous tissue. We recommend the use of the operating microscope and hypotension to avoid the possibility of such damage.

Another method that has been used to reduce damage to surrounding nervous tissue to a minimal degree is the "backward technique."^{12,25,33} In the cases reported above, this technique was used for the small AVM in case 8,

in which the closing of the single draining vein did not cause swelling of the AVM (most likely because the patient was under hypotension). In two other cases the same technique was used, by closing a large draining vein first, but in these cases there existed a multiple venous drainage system. The importance of hypotension must be stressed. It is very useful in the removal of critical AVMs, as already noted by Kunc¹² and Pertuiset.²⁵

The difficulty of controlling hemorrhage toward the end of the surgical procedure seems to be linked to the paraventricular location of many of these AVMs, and is caused by the presence of small vessels, which are very difficult to coagulate or clip. The problem of this abnormal bleeding is worth careful evaluation, as already pointed out by Pertuiset.²⁵

Our results have shown that postoperative disturbances can be expected, but that they are generally transient and most likely linked to the altered vascular dynamics.⁷

It should be stressed that all of the patients experiencing epilepsy before surgery had not had any further seizures at follow-up examinations ranging between six months and two years. Another important result is the absence of any signs suggesting mental deterioration in these patients, even at relatively late follow-up.

In conclusion, our series confirms the possibility of surgically removing AVMs located in functionally important areas of the brain with good clinical results.

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