

Microsurgical Treatment of Juxtapeduncular Angiomas

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Five patients harboring juxtapeduncular angiomas were all treated by a direct microsurgical approach. Anatomic considerations, angiographic data on the feeding arteries, and related anomalies are discussed. The operating microscope and deep hypotension were very useful for the complete removal of these lesions, which were approached in most cases by a "backward" technique. Diffuse bleeding from the paraventricular area was difficult to handle. There were no postoperative deaths. At follow-up examination, 1 patient had hemiparesis and hemianopia, 2 patients had mild visual field defects, and 2 patients had normal neurological examinations. All but one of the patients have resumed their previous occupations. It is concluded that microsurgery constitutes the best treatment for these malformations.

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Treatment of angiomas involving the medial part of the temporal lobe and extending into the ambient cistern (so-called juxtapeduncular angiomas) is controversial. Although alternative methods of treatment such as stereotactic clipping [23], embolization [14], balloon occlusion [28], and focused high-energy radiotherapy [31] have been proposed, a direct surgical approach to these lesions has been advocated by very few authors [4, 9, 20] because of the difficulties associated with this critical location.

In the present report we describe our experience with juxtapeduncular angiomas in 5 patients, all radically resected by a microsurgical technique; there was no mortality and there was acceptable morbidity.

Material

From May, 1979, to October, 1980, 5 patients with juxtapeduncular angiomas were operated on by one of us (R.D.P.). They constitute 4% of the 126 patients with arteriove-

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nous malformations (AVM) treated in our department. Four patients were male and 1 female; their ages ranged from 19 to 43 years. History in all patients included a subarachnoid hemorrhage, in 2 patients associated with intracerebral hematoma and in 1 patient with epilepsy. As shown in the Table, 4 malformations were on the right and 1 on the left. According to the classification of Mingrino [15], 2 arteriovenous malformations were large, extending into the temporal horn, and 3 were of medium size (maximum diameter, 2 to 5 cm). In all patients, feeding vessels were branches of the posterior cerebral artery, with its inferior temporal and posterolateral choroidal branches, and in 2 patients the anterior choroidal artery also contributed. The posterior cerebral artery originated directly from the carotid artery (fetal type) in 2 patients. Venous drainage was directed medially in 4 patients with frequent dilatation of the vein of Galen and laterally in 4 patients. Computed tomographic (CT) scan with and without enhancement was routinely used for correct localization of the angioma and for documentation of the hemorrhage and mass effect.

On preoperative examination, 3 patients had no deficits, 1 patient was slightly confused, and only 1 patient exhibited a right hemiparesis. There were no postoperative deaths. Postoperative angiography demonstrated complete exclusion of the AVM in all patients. At discharge, severe disability was noted only in the patient who had had right hemiparesis, dysphasia, and hemianopia. Hemianopia was noted in 2 patients, one associated with minimal hemiparesis. Two patients had normal neurological examinations. Two to fifteen months after operation, 4 patients are working full-time; 1 has not yet resumed his previous occupation because of hemiparesis and dysphasia. In the patient with a previous history of epilepsy, and at present under anticonvulsant therapy, convulsions have not recurred.

Anatomic and Angiographic Considerations

These AVMs are all located in the medial and inferior portion of the temporal lobe, involving the parahippocampal gyrus, the hippocampus, and not infrequently the temporal horn of the lateral ventricle, as shown in Figure 1. They sit above the tentorium and extend into the cisterna ambiens through the tentorial notch, in close relationship with the upper brainstem and especially with the cerebral peduncles.

Summary of Cases

Patient No.	Patient Sex, Age (yr)	Signs & Symptoms	Side/Size; Other Findings	Feeders	Draining Veins	Duration of Operation (hr)	Hypotension (hr)/Blood (ml)	Postoperative Course
1	M, 28	SAH; mild right hemiparesis	Left/large; left temporal & intraventricular hematoma	Fetal PCA, AChA	Vein of Galen, petrosal sinus	7	3/2,500	Worsening of paresis, lasting hemianopia, transient dysphasia
2	M, 43	SAH, epilepsy, confusion	Right/large	Fetal PCA	Vein of Galen, superior sagittal & lateral sinuses	9	2/1,500	Lasting hemianopia, transient hemiparesis
3	F, 27	2 SAHs	Right/medium	Inferior temporal, PLC arteries	Vein of Galen, lateral & petrosal sinuses	6	2/1,200	Uneventful
4	M, 31	SAH	Right/medium	Inferior temporal, PLC, AChA	Vein of Galen, basal vein	10	3/2,000	Lasting hemianopia, transient hemiparesis
5	M, 19	SAH, mild confusion	Right/medium; right temporal & intraventricular hematoma	Inferior temporal, PLC arteries	Lateral & sphenoidal sinuses	7	2/1,200	Uneventful

SAH = subarachnoid hemorrhage; PCA = posterior cerebral artery; AChA = anterior choroidal artery; PLC = posterolateral choroidal artery.

The main feeding vessel to these angiomas (Fig. 2) is the posterior cerebral artery (PCA) through: (1) the inferior temporal arteries, arising as a single trunk or as separate branches (hippocampal, anterior, middle, and posterior temporal arteries), feeding the inferomedial part of the temporal lobe; and (2) 1 to 9 lateral posterior choroidal arteries passing through the choroidal fissure and feeding the region surrounding the temporal horn of the lateral ventricle and the choroid plexus of the temporal horn.

The other vessel supplying these angiomas is the anterior choroidal artery, with terminal branches that anastomose with the lateral posterior choroidal arteries in the choroid plexus of the temporal horn (Fig. 2).

Anomalies of the posterior cerebral artery are relatively common, particularly originating directly from the carotid artery (fetal type) with absent or poorly developed pre-communicating segments. The venous drainage of these angiomas is mainly toward the deep venous system and to a minor extent toward the lateral, sigmoid, and cavernous sinuses and cortical veins.

Surgical Technique

A temporal approach was adopted in all patients, as suggested by Drake [3, 5], for aneurysms of the basilar and posterior cerebral arteries. The temporal flap was extended anteriorly to reach the lesser sphenoid wing and to gain better exposure of the anterior portion of the temporal lobe. The veins draining toward the lateral sinus were spared

when possible, especially on the left. In our patients, it was never necessary to incise the tentorium. The brain was relaxed by osmotic agents and by opening the ambient cistern. The operating microscope was particularly useful in the narrow operative field with limited space for surgical maneuvers. In some patients, as suggested by Drake and Amacher [5], it was convenient to remove part of the parahippocampal gyrus overlying the angioma using suction—a technique very similar to the removal of the gyrus rectus for approaching anterior communicating artery aneurysms. In all patients the posterior cerebral artery was very deep, hidden in the choroidal fissure. Also, the presence of a fetal type posterior cerebral artery caused further difficulty in identifying the single feeding vessels [18]. The venous drainage, often extensive and overlying the angioma itself, was approached by inducing deep hypotension, allowing closure of draining veins before the feeding arteries were occluded. This "backward" technique was without complications. Deep hypotension and the operating microscope were extremely useful in the progressive closure of the feeding vessels by clipping and by normal or bipolar coagulation. Repeated hemorrhages during dissection of the often numerous lateral choroidal branches, as well as diffuse bleeding from the paraventricular area especially at the end of operation, were difficult and often caused a delay of hours for the complete removal of the malformation. During deep hypotension induced by sodium nitroprusside [2], blood pressure values were maintained at

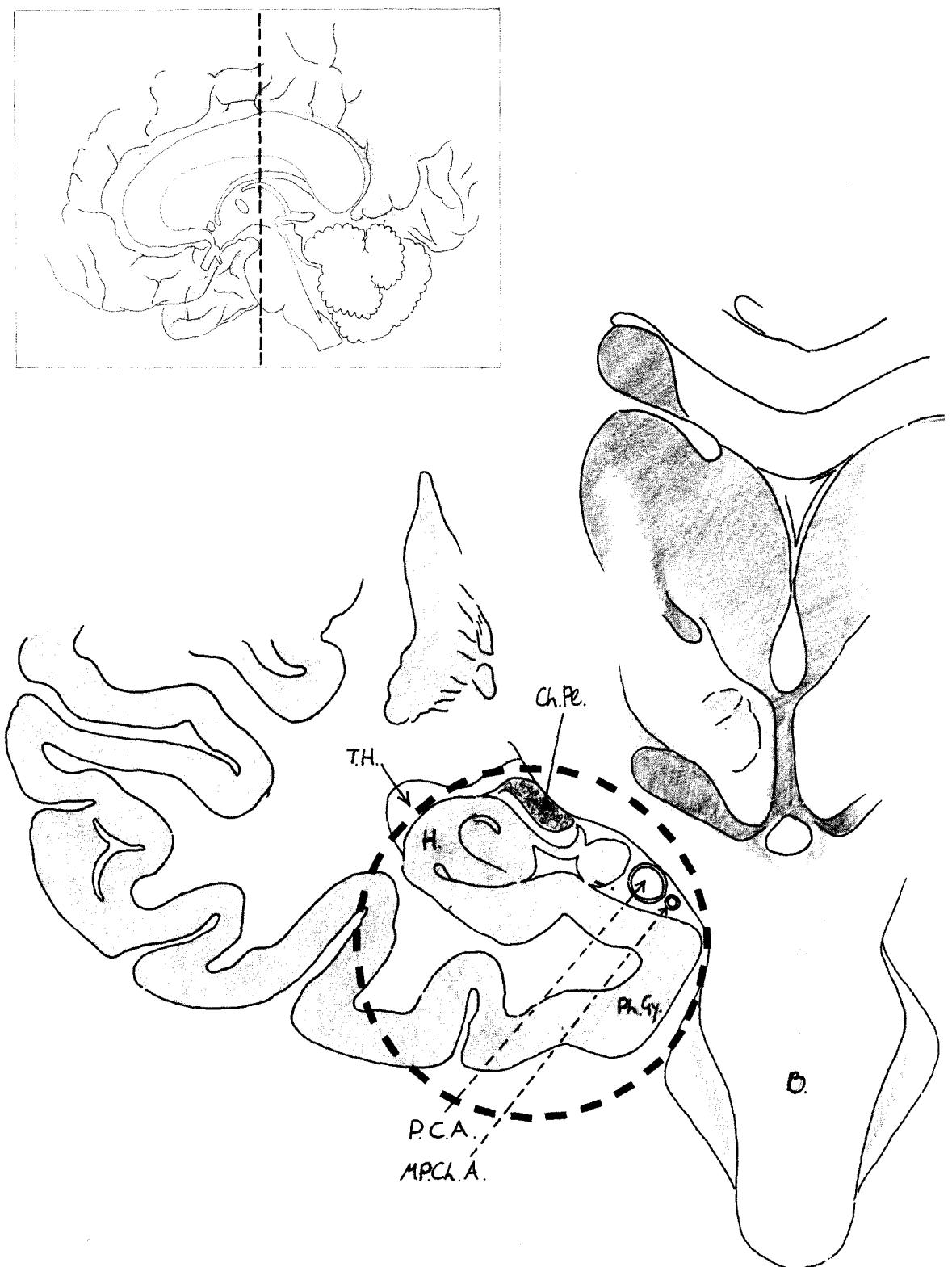


Fig. 1. Location of juxtaependymal angiomas in a vertical section.
ChPl = choroid plexus of the temporal horn; PCA = posterior cerebr al artery; MPChA = postero-medial choroidal artery; PhGy = parahippocampal gyrus; H = hippocampus; TH = temporal horn of the lateral ventricle; B = brainstem. The area of AVM is indicated by the dashed circle.

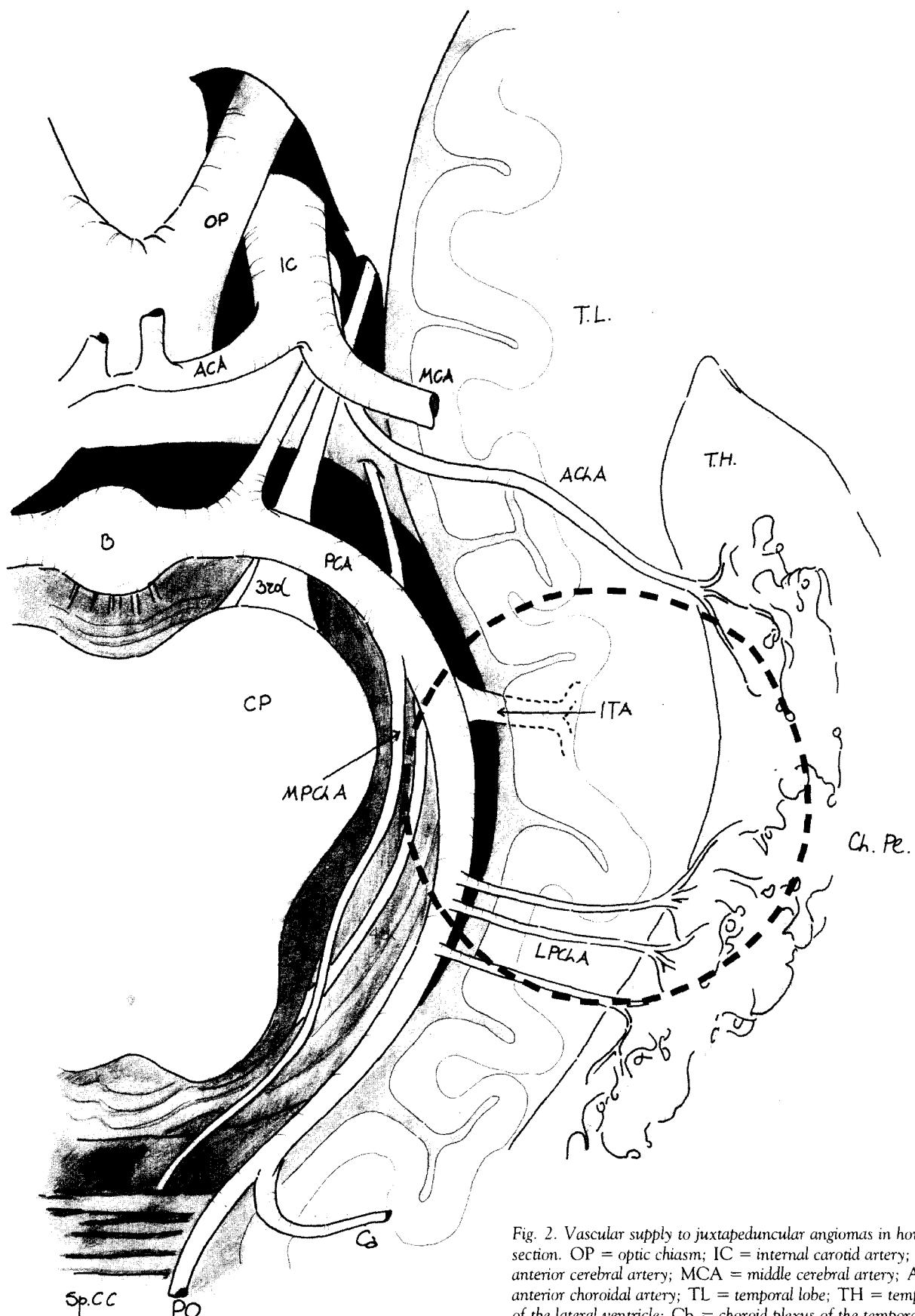


Fig. 2. Vascular supply to juxta-peduncular angiomas in horizontal section. OP = optic chiasm; IC = internal carotid artery; ACA = anterior cerebral artery; MCA = middle cerebral artery; AChA = anterior choroidal artery; TL = temporal lobe; TH = temporal horn of the lateral ventricle; Ch = choroid plexus of the temporal horn; B = basilar fundus; 3rd = oculomotor nerve; PCA = posterior cerebral artery; CP = cerebral peduncle; ITA = inferior temporal artery(ies); MPChA = posteromedial choroidal artery; LPChA = posterolateral choroidal arteries; 4th = trochlear nerve; Ca = calcarine artery; PO = parietooccipital artery; SpCC = splenium of corpus callosum. The area of AVM is indicated by the dashed circle.

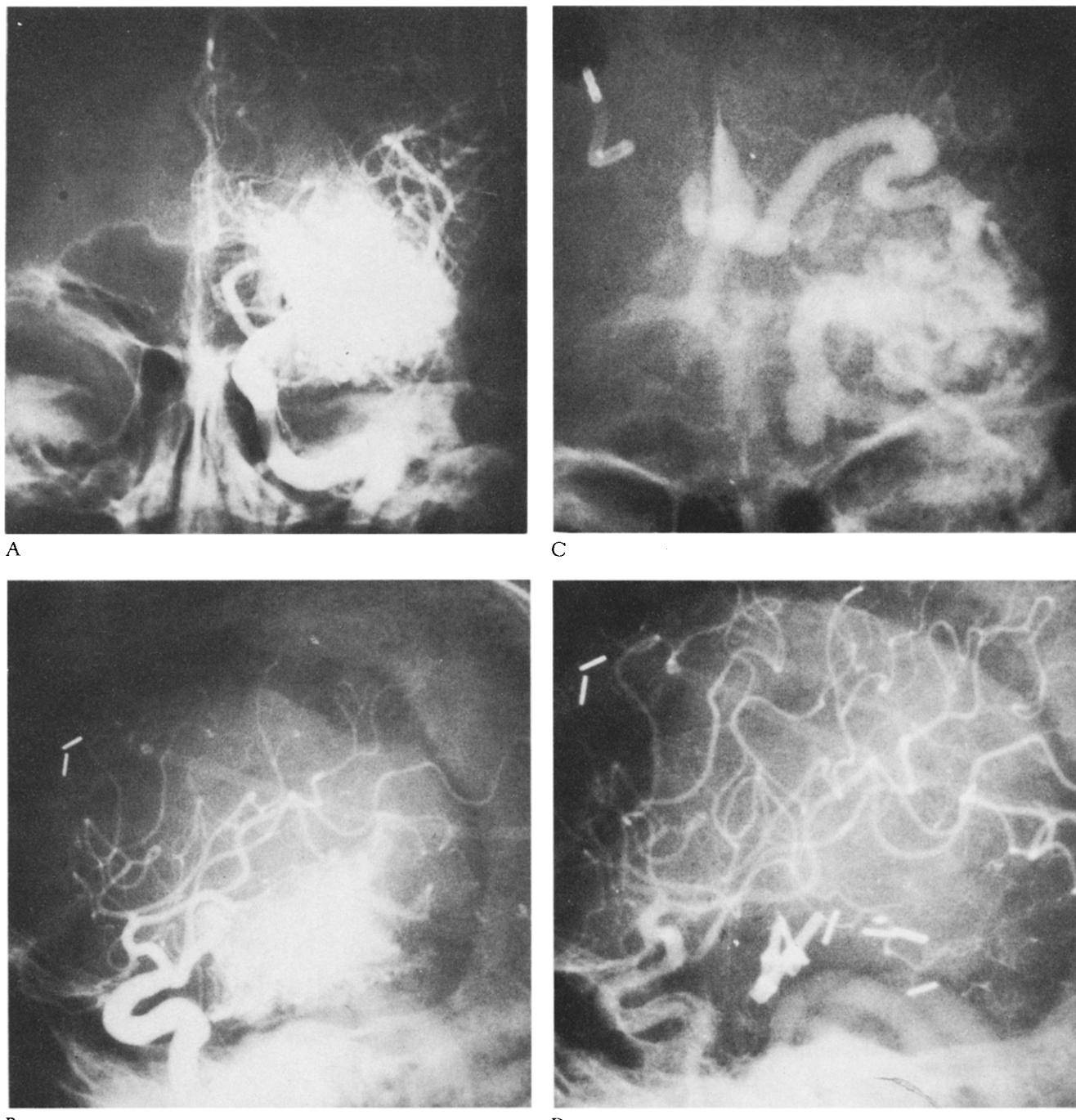


Fig. 3. Patient 1. Left carotid angiograms. (A) Anteroposterior and (B) lateral views: huge AVM of the left mesial temporal lobe fed by a fetal posterior cerebral artery and by the anterior choroidal artery. (C) Venous drainage toward the vein of Galen and petrosal sinus. (D) Postoperative view: angioma removed with marked spasm of posterior cerebral artery.

about 50 mm Hg systolic in all patients; the total dose of the drug never exceeded 2 milligrams per kilogram of body weight, even when deep hypotension was maintained for hours. It should be stressed that all the surgical procedures lasted six to ten hours, and each patient required more than 1 liter of blood.

Case Reports

Patient 1

A 28-year-old man had been operated on in another hospital at the age of 8 years for the evacuation of a left tem-

poral hematoma. Ten days after a verified subarachnoid hemorrhage, he was admitted to our department on March 21, 1979, with marked stiffness of the neck and a mild right hemiparesis. A left carotid angiogram (Fig. 3) showed a

huge AVM involving the left mesial temporal lobe, fed by a fetal type posterior cerebral artery and by the anterior choroidal artery; the venous drainage was toward the vein of Galen and the petrosal sinus. A CT scan showed a left temporal hematoma with intraventricular hemorrhage. After the insertion of a ventriculoatrial shunt for control of progressive ventricular dilatation, the patient was operated upon six weeks after admission, at which time he was still hemiparetic.

After a left subtemporal approach, the angiomatic vessels appeared on the inferomedial surface of the temporal lobe. Before the angioma was approached, the posterior cerebral artery was exposed in its anterior part, showing an altered anatomic relationship to the basilar fundus (the pre-communicating tract was absent) and to the cerebral peduncle. The feeders to the malformation were numerous: by approaching the angioma at its edges, these vessels were progressively closed. The main trunk of the posterior cerebral artery could be seen until its entrance into the angioma, and then not distally. The venous drainage, both lateral and medial, was occluded. Medially, the angioma was adherent to the cerebral peduncle, but could be dissected free. The complete removal of the lesion took almost three hours, and was performed with the patient under continuous deep hypotension and with adequate control of bleeding. A diffuse hemorrhage from the ventricular walls, after removal of the angioma, constituted a very difficult problem; however, hemostasis was finally achieved.

After a difficult postoperative course, the patient was discharged three weeks after the operation with dysphasia, severe right hemiparesis, and right homonymous hemianopia. Subsequent angiography (Fig. 3D) showed a marked spasm of the posterior cerebral artery. On examination fifteen months later after three revisions of the shunt system, the patient showed improvement from the right hemiparesis, persistence of the hemianopia, and disappearance of the dysphasia; nevertheless, he has not yet resumed his former occupation.

Patient 2

A 43-year-old man had suffered a mild subarachnoid hemorrhage fifteen years before, and for more than seventeen years had had a history of uncinate convulsions that responded little to medical therapy. After a grand mal seizure, he was admitted to another hospital, where a right carotid angiogram (Fig. 4) showed a large AVM of the right mesial temporal lobe, fed by a fetal posterior cerebral artery, with huge veins draining medially to the vein of Galen and laterally to the lateral and superior sagittal sinuses. On admission to our department on July 2, 1979, the patient was found to be slightly confused. A contrast-enhanced CT scan (Fig. 5) showed the location of the angioma within the temporal lobe and in the temporal horn, with medial extension to the ambient cistern and with dilated venous drainage. Three weeks after admission, the patient was op-

erated upon while he was still in a slightly confused condition.

By a right frontotemporal approach, the sylvian fissure and the undersurface of the temporal lobe, covered by draining veins, were exposed. After removal of the anterior tip of the temporal lobe and occlusion of the venous drainage toward the sphenoidal sinus, the angioma could be approached deeply. After the large fetal posterior cerebral artery was found more medial and deeper than normal, the feeding vessels to the angioma were progressively closed. Finally, the large posterior cerebral artery, apparently terminating in the angioma, was temporarily closed by a clip, thus reducing the size of the angioma. Further exploration using the microscope allowed identification of the distal trunk of the posterior cerebral artery, from which a "hair-comb" of vessels emerged to feed the angioma; by closing these vessels, it was possible to remove the clip on the posterior cerebral artery and to excise the angioma completely, sparing the posterior cerebral artery. The angioma extended into the temporal horn and reached the cerebral peduncle.

Three weeks later the patient experienced much improvement from a left hemiparesis and homonymous hemianopia, which developed during the postoperative course. A subsequent angiogram (Fig. 4D) showed preservation of the posterior cerebral artery and complete exclusion of the angioma. On examination eleven months later, the patient still had a visual deficit, whereas the hemiparesis had almost completely disappeared. He has now resumed his previous occupation and according to relatives has shown a significant psychological improvement since the operation. It is interesting to note that the patient has suffered no further uncinate convulsions, even though the anticonvulsant therapy is the same as before the operation (phenobarbital and carbamazepine).

Patient 3

A 27-year-old female teacher was admitted to another hospital after two mild episodes of verified subarachnoid hemorrhage. Four-vessel angiography (Fig. 6) showed a medium-sized right juxtapeduncular angioma involving the parahippocampal gyrus, with arterial supply from the posterior cerebral artery through inferior temporal and posterolateral choroidal branches. The venous drainage was toward the vein of Galen, and toward the petrosal and lateral sinuses. On admission to our department on March 3, 1980, the patient had no neurological deficits. A CT scan confirmed the location of the angioma. One week after admission, the patient was operated upon.

The parahippocampal gyrus was exposed through a right temporal craniotomy: the angioma involved this gyrus and extended into the ambient cistern adherent to the cerebral peduncle. The trunk of the posterior cerebral artery was exposed; the inferior temporal and posterolateral choroidal branches, departing from the trunk itself, were all occluded near the malformation. To dissect the angioma from the

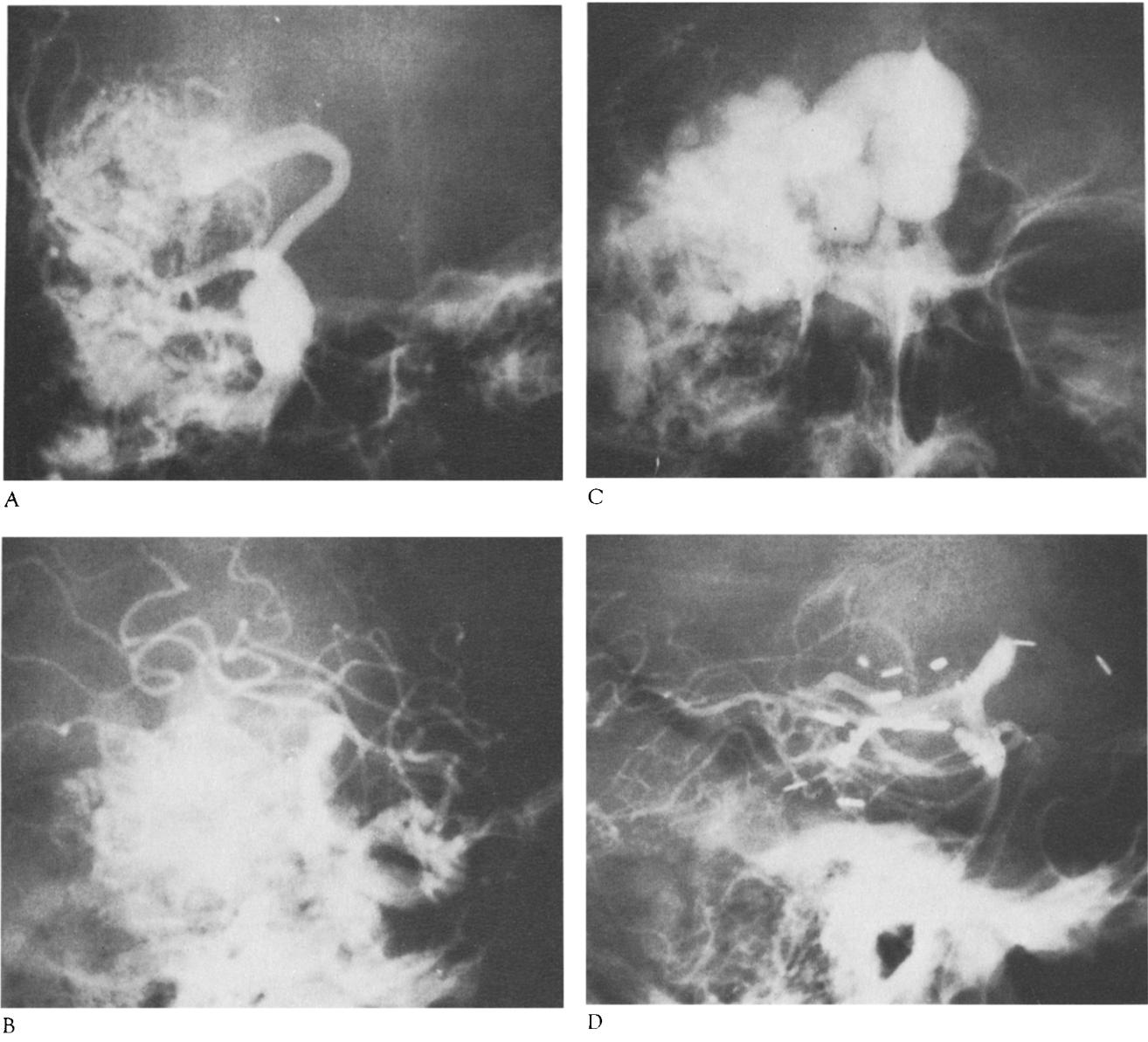


Fig. 4. Patient 2. Right carotid angiograms. (A) Anteroposterior and (B) lateral views: large AVM of the right mesial temporal lobe (note marked hypertrophy of fetal posterior cerebral artery). (C) Extensive venous drainage toward the vein of Galen and the lateral sinus. (D) Postoperative view, showing complete exclusion of the AVM (note the angulated clip parallel to the posterior cerebral artery).

peduncle, it was necessary to close a large medially draining vein, thus exposing the underlying last feeders; after their closure, the angioma slackened and could be removed after closure of the remaining medially-draining veins. Hemostasis was particularly difficult to maintain in the area of the temporal horn of the lateral ventricle.

After an uneventful postoperative course, the patient was discharged one week later with no deficit. At a subsequent examination eight months later, the patient had resumed teaching and was free of disturbances.

Patient 4

A 31-year-old male teacher was admitted to another hospital after a verified subarachnoid hemorrhage. A complete angiographic study (Fig. 7) showed a medium-sized right

juxtapeduncular angioma involving the hippocampal gyrus and fed by the posterior cerebral artery through inferior temporal and posterolateral choroidal branches and by the anterior choroidal artery. The venous drainage was toward the vein of Galen and the basal vein. A CT scan (Fig. 8) confirmed the location of the angioma, extending into the ambient cistern. On admission to our department on May



A



B

Fig. 5. Patient 2. CT scan with contrast enhancement: (A) right medial temporal angioma extending to the ambient cistern; (B) dilated venous drainage.

5, 1980, the patient had a normal neurological examination. One week later, he was operated upon.

After a right temporal craniotomy, the hippocampal gyrus, covered with angiomatic vessels, was exposed. The posterior cerebral artery was identified in the choroidal fissure, and numerous branches departing from its trunk and feeding the angioma were progressively closed inside the hippocampal gyrus. It was necessary to close a large draining vein to expose other feeders to the angioma. The identification of these vessels was particularly difficult because of a severe hemorrhage, even though the patient was under hypotension. After control of the hemorrhage and closure of the feeding vessels and of the medially draining veins, the angioma could be removed by dissection of its points of adherence to the brainstem and to the cerebellum. The surgical procedure in this case was particularly difficult because of the severity of the hemorrhage.

A mild left hemiparesis that developed in the early postoperative course had disappeared by discharge two weeks later; a left homonymous hemianopia persisted unchanged. Subsequent angiography (Fig. 7D) showed complete exclusion of the angioma with preservation of the posterior cerebral and anterior choroidal arteries. At an examination six months later, the patient had resumed his previous activity.

Patient 5

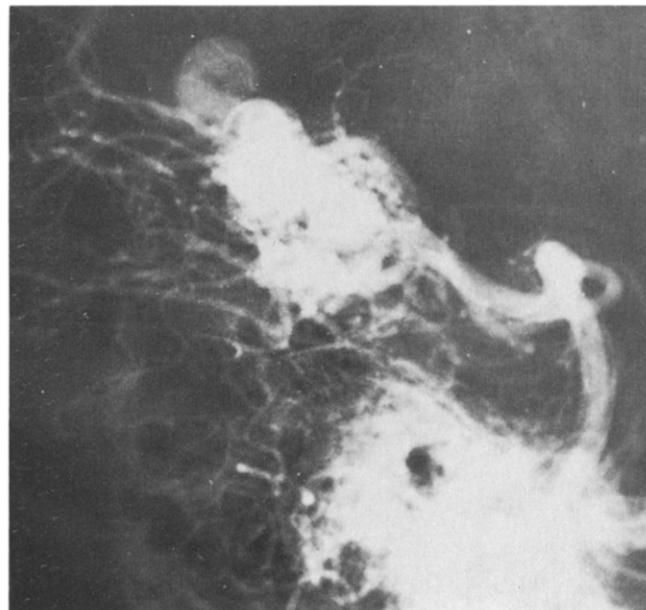
A 19-year-old male student, with a long history of severe recurrent headaches, was admitted to our department on September 22, 1980, two days after a verified subarachnoid hemorrhage. On admission, he had marked stiffness of the neck and slight confusion. A CT scan showed a right temporal hematoma with intraventricular hemorrhage. A right vertebral angiogram (Fig. 9) showed a medium-sized AVM involving the parahippocampal gyrus, fed by the posterior cerebral artery through the inferior temporal, posterolateral choroidal, and parietooccipital arteries. The venous drainage was toward the lateral and sphenoidal sinuses. After reabsorption of the intraventricular hemorrhage, the patient was operated upon one month later, with no neurological deficits.

By a right temporal approach, the red veins draining to the sphenoidal sinus were closed before the angioma was approached. The parahippocampal gyrus, covered by angiomatic tissue, and the posterior cerebral artery, hidden in the choroidal fissure, were exposed. Because of the presence of the hematoma, the undersurface of the temporal lobe was incised and the temporal horn, filled with clots, was exposed; the angioma extended to the medial walls of the temporal horn. After closure of the numerous feeding vessels from the posterior cerebral artery, the venous drainage toward the lateral sinus became dark. The whole angioma was then removed together with the parahippocampal gyrus after dissection from the cerebral peduncle.

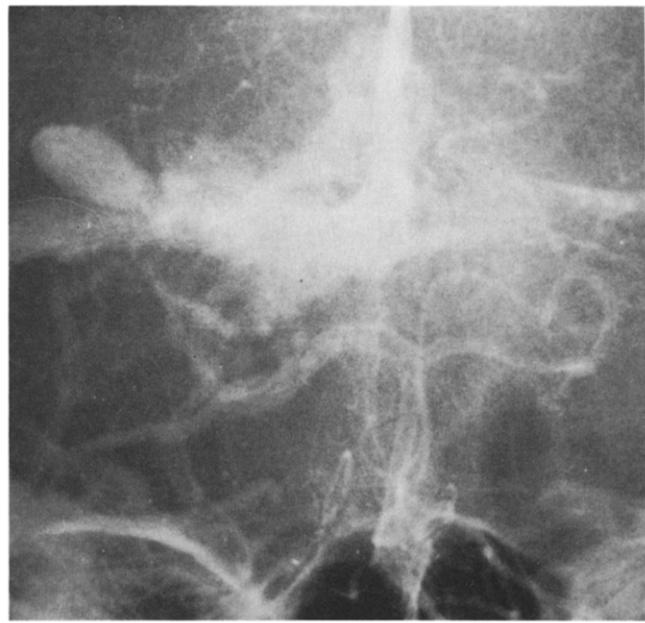
After an uneventful postoperative course, the patient



A



B



C



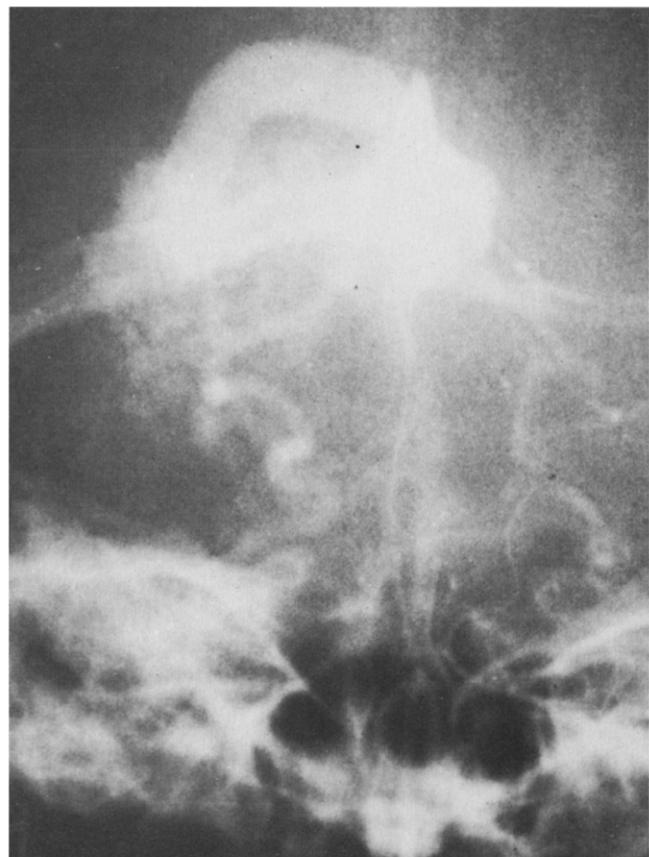
D

Fig. 6. Patient 3. Vertebral angiograms. (A) Anteroposterior and (B) lateral views of right juxtapeduncular angioma. (C) Venous drainage toward the vein of Galen and the petrosal and lateral sinuses. (D) Postoperative view showing exclusion of the AVM.

Fig. 7. Patient 4. Vertebral angiograms. (A) Anteroposterior and (B) lateral views of right juxtapeduncular angioma. (C) Venous drainage toward the vein of Galen and basal vein. (D) Postoperative view, with angioma completely excluded (note the numerous clips and the preservation of a patent posterior cerebral artery).



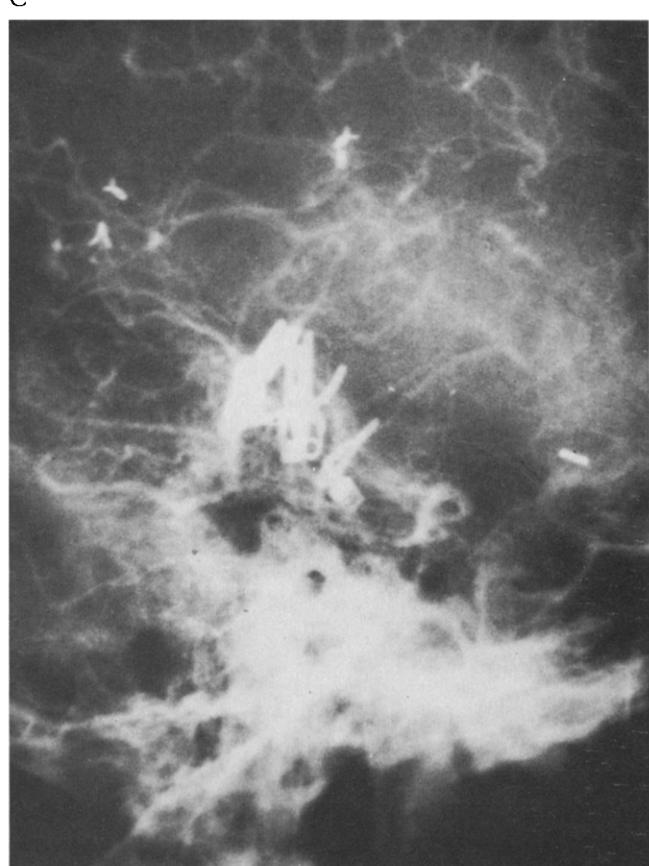
A



C



B



D

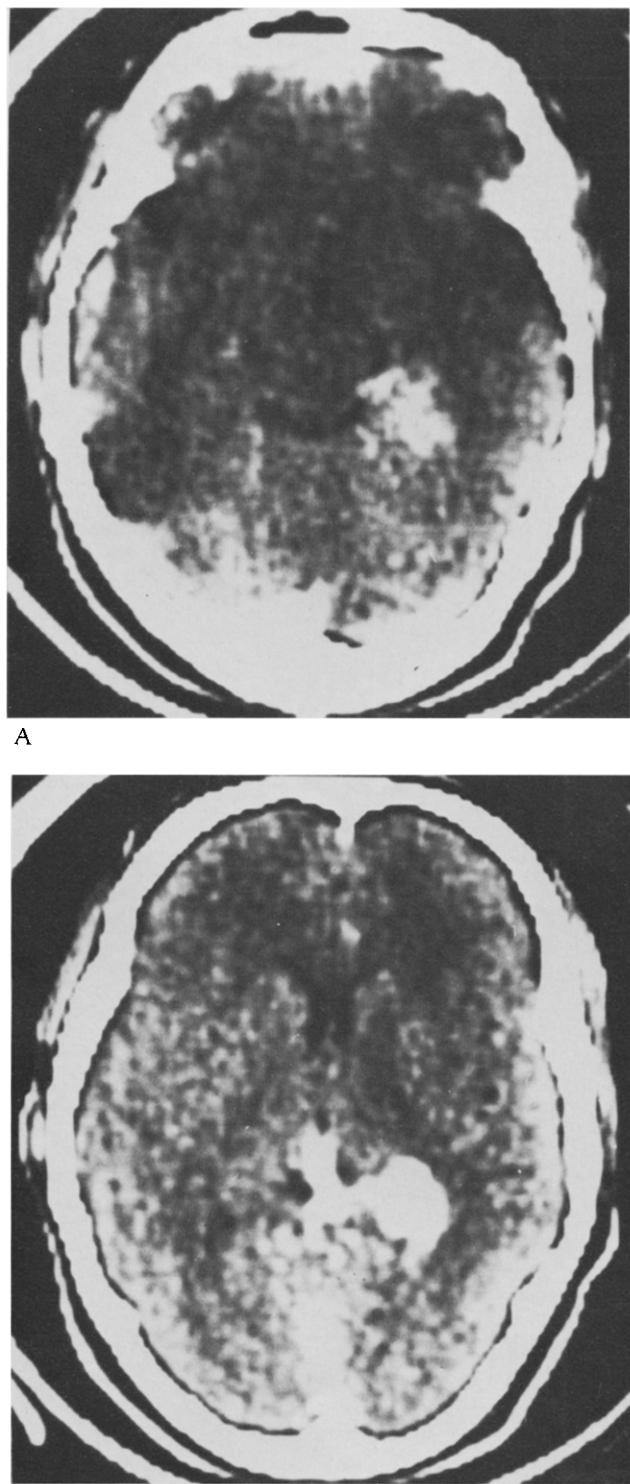


Fig. 8. Patient 4. CT scan with contrast enhancement. (A) Right juxtapeduncular angioma extending to the ambient cistern; (B) dilated venous drainage.

was discharged two weeks after the operation with a normal neurological examination. A subsequent angiogram on November 6, 1980 (Fig. 9C), showed the complete exclusion of the AVM. On examination two months later the patient showed no residual disturbances and had been attending school regularly.

Discussion

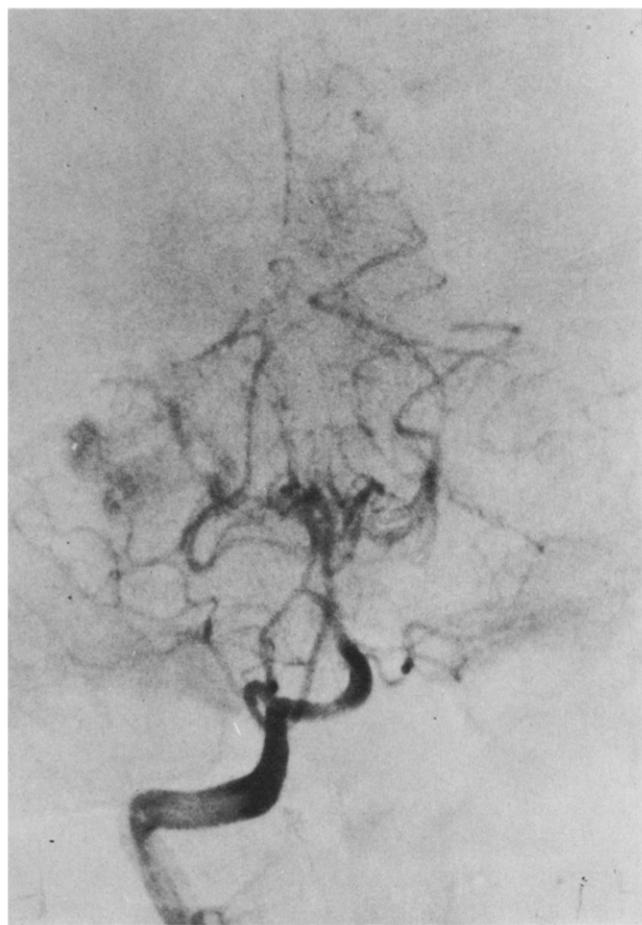
Juxtapeduncular angiomas are rare; our experience supports the findings of other authors who claim that they constitute only about 3 to 4% of cerebral AVMs [4, 19].

Their treatment is controversial because of the difficulties presented by their surgical removal. These lesions have rarely been operated on in the past [20], and with poor results. More recently, direct surgical treatment has been advocated by a few authors [4, 9] on the basis of more encouraging experiences. However, most reports still advise alternative methods of treatment, such as embolization [13, 14, 16, 26, 27, 30], stereotactic clipping [10, 23, 24], cryotherapy [37, 38], focused high-energy radiotherapy [8, 11, 31, 32], and balloon occlusion [28], even though none of these methods is free from risk. Even nonoperative treatment has been proposed in view of the relatively low incidence of rebleeding [33, 35, 36].

However, complete surgical obliteration of the lesion is undoubtedly the best type of treatment when coupled with the prospect of low morbidity. In this connection, hemianopia should be considered a minor concern when evaluating the possibility of a fatal hemorrhage when left untreated. Even with surgical closure of the main trunk of the posterior cerebral artery, the development of a neurological deficit is not always the rule: other authors [5, 7] have reported permanent occlusion of this artery without consequence to the patient. It should be pointed out that the vascularization of the calcarine cortex is far from exhibiting a uniform pattern; collateral flow may well nourish this area in the absence of supply from the posterior cerebral artery [29]. Unfortunately, this was not the case in our first patient, in whom the neurological impairment after spasm of the posterior cerebral artery was severe, possibly because of the high output of the fetal posterior cerebral artery.

As regards anatomic considerations about the operability of these angiomas, the possibility of dissection from the surface of the brainstem should be stressed. In fact, even if these angiomas extend into the cisterna ambiens, in our experience they are not closely adherent to the brainstem. These angiomas may sometimes be difficult to differentiate from mesencephalic angiomas in which surgical treatment is not clearly indicated, as pointed out by Drake [4].

In our experience, the operability of juxtapeduncular angiomas is not related to the direction of the venous outflow: whereas a medially-directed venous drainage is regarded by some authors as indicating inoperability [9, 20], it did not prevent excision of the angiomas in our patients.



A



B

Fig. 9. Patient 5. Vertebral angiograms: (A) anteroposterior and (B) lateral views of right juxtapeduncular angioma. (C) Postoperative view showing exclusion of AVM.



C

The precise identification of the tentorium, achieved by CT scan [17], is important for preoperative localization of the AVM above or below the tentorium. In all of our patients, the lesion was entirely supratentorial or paratentorial, which facilitated a direct subtemporal approach.

As shown by our 2 patients, anomalies of the posterior cerebral artery are common when dealing with large AVMs fed by this artery. The fetal type posterior cerebral artery, originating directly from the carotid artery, has been widely described elsewhere [18, 25, 39]. This anomaly causes a major alteration of the normal anatomic relations between this artery, the brainstem, and the inferomedial part of the temporal horn. Consequently, identification and dissection of the branches departing from the main trunk of the posterior cerebral artery present considerable difficulties, as shown in our first patient.

The normal rich vascularization of the area surrounding the temporal horn supplied by the anterior and posterolateral choroidal arteries is even more luxuriant in the presence of a juxtapeduncular angioma. As pointed out by others [6], the lateral posterior choroidal arteries can be numerous—as many as 9 in a normal hemisphere. The difficulty in handling hemorrhages from these tiny, sparse vessels cannot be overemphasized. Moreover, diffuse varicosities on the ventricular walls, a cause of annoying bleeding, are found not only in these angiomas, but also in any angioma extending to the ventricular system anywhere. Our experience with other patients [1] suggests that this problem arises from the extensive anastomoses between the

terminal branches of the various choroidal arteries—in the lateral, third, and fourth ventricles—as illustrated by Rhoton and colleagues [6, 22, 25, 39]. These anastomoses are certainly engorged in the presence of angiomatic tissue in proximity; it is also possible that, in these conditions, the capillary walls prove extremely fragile to even minimal mechanical manipulation, thus explaining the diffuse paraventricular bleeding.

As to the value of deep hypotension for surgical removal of AVMs, our previous experience [1] was confirmed by these 5 cases. Apart from better control of bleeding, especially valuable in these highly vascularized lesions, deep hypotension was useful in our "backward" approach, i.e., initial occlusion of the draining veins from the malformation. In fact, during hypotension, closure of the slackened veins does not cause swelling of the angioma as it does under normotension. The "backward" technique, suggested by others before [12, 21, 34], facilitates the approach to the individual feeding vessels, easily identified after partial exclusion of the venous drainage.

The operating microscope proved necessary in the surgical treatment of these lesions, which are located deep in the brain, thus allowing only a limited field for surgical maneuvers.

In conclusion, with the use of modern operative and anesthesiological facilities, surgical treatment of juxtapeduncular angiomas has become not only possible but even advisable in view of the low morbidity when compared to the risks of alternative or conservative treatment.

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References

1. Da Pian R, Pasqualin A, Scienza R, Vivenza C: Microsurgical treatment of ten arteriovenous malformations in critical areas of the cerebrum. *J Microsurg* 1:305-320, 1980
2. Da Pian R, Pasqualin A, Scienza R, Vivenza C, Malesani GC: Deep controlled hypotension with sodium nitroprusside in the surgical treatment of intracranial arterial aneurysms. *J Neurosurg Sci* 23:109-120, 1979
3. Drake CG: Further experience with surgical treatment of aneurysms of the basilar artery. *J Neurosurg* 29:372-392, 1968
4. Drake CG: Cerebral arteriovenous malformations: considerations for and experience with surgical treatment in 166 cases. *Clin Neurosurg* 26:145-208, 1979
5. Drake CG, Amacher AL: Aneurysms of the posterior cerebral artery. *J Neurosurg* 30:468-474, 1969
6. Fujii K, Lenkey C, Rhoton AL Jr: Microsurgical anatomy of the choroidal arteries: lateral and third ventricles. *J Neurosurg* 52:165-188, 1980
7. Hunt WE, Hess RM: Aneurysm of the posterior cerebral artery with unexpected postoperative neurological deficit. Case report. *J Neurosurg* 26:633-635, 1967
8. Johnson RT: Radiotherapy of cerebral angiomas. With a note on some problems in diagnosis, in Pia HW, Gleave JRW, Grote E, Zierski J (eds): *Cerebral Angiomas. Advances in Diagnosis and Therapy*. Berlin: Springer-Verlag, 1975, pp 256-266
9. Juhász J: Surgical treatment of arteriovenous angiomas localised in the corpus callosum, basal ganglia and near the brain stem. *Acta Neurochir (Wien)* 40:83-101, 1978
10. Kandel EI, Peresedov VV: Stereotaxic clipping of arterial aneurysms and arteriovenous malformations. *J Neurosurg* 46:12-24, 1977
11. Kjellberg RN, Poletti CE, Robertson GH, Adams DA: Bragg peak proton beam treatment of arteriovenous malformations of the brain, in Carrea R (ed): *Neurological Surgery (With Emphasis on Non-Invasive Methods of Diagnosis and Treatment)*, Proceedings of the 6th International Congress of Neurological Surgery, São Paulo, June, 1977. Amsterdam: Excerpta Medica, 1978, pp 181-187
12. Kunc Z: Operability of arteriovenous malformations in functionally important cortical regions, in Fusek I, Kunc Z (eds): *Present Limits of Neurosurgery*. Prague: Avicenum, 1972, pp 293-298
13. Luessenhop AJ: Artificial embolization of inoperable arteriovenous malformations, in Pia HW, Gleave JRW, Grote E, Zierski J (eds): *Cerebral Angiomas. Advances in Diagnosis and Therapy*. Berlin: Springer-Verlag, 1975, pp 198-205
14. Luessenhop AJ, Presper JM: Surgical embolization of cerebral arteriovenous malformations through internal carotid and vertebral arteries. Long-term results. *J Neurosurg* 42:443-451, 1975
15. Mingrino S: Supratentorial arterio-venous malformations of the brain, in Krayenbühl H (ed): *Advances and Technical Standards in Neurosurgery*. Vienna: Springer-Verlag, 1978, Vol 5, pp 93-123
16. Mullan S, Kawanaga H, Patronas NJ: Microvascular embolization of cerebral arteriovenous malformations. *J Neurosurg* 51:621-627, 1979
17. Naidich TP, Leeds NE, Kricheff II, Pudlowski RM, Naidich JB, Zimmerman RD: The tentorium in axial section, I and II. *Radiology* 123:631-648, 1977
18. Newton TH, Hoyt WF, Margolis MT: The posterior cerebral artery. Sections I, II, and III, in Newton TH, Ports DG (eds): *Radiology of the Skull and Brain: Angiography*. St. Louis: Mosby, 1974, Vol 2, Book 2, pp 1540-1627
19. Perret G, Nishioka H: Report on the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage. Arteriovenous malformations. An analysis of 545 cases of cranio-cerebral arteriovenous malformations and fistulae reported to the cooperative study. *J Neurosurg* 25:467-490, 1966
20. Pertuiset B, Sachs M, Guyot JF: Les anévrismes artérioveineux des parois juxta-pédonculaires de la fente de Bichat. *Presse Med* 71:2341-2342, 1963
21. Pertuiset B, Sicchez JP: The backward technique in the total excision of cerebral arteriovenous malformations: experience with 86 cases, in Wüllenweber R et al (eds): *Advances in Neurosurgery*. Berlin: Springer-Verlag, 1978, Vol 6, pp 148-153
22. Rhoton AL Jr, Fujii K, Fradd B: Microsurgical anatomy of the anterior choroidal artery. *Surg Neurol* 12:171-187, 1979
23. Riechert T: Stereotactic treatment of central angiomas, in Pia HW, Gleave JRW, Grote E, Zierski J (eds): *Cerebral Angiomas. Advances in Diagnosis and Therapy*. Berlin: Springer-Verlag, 1975, pp 129-135
24. Riechert T, Mundinger F: Combined stereotaxic operation for treatment of deep-seated angiomas and aneurysms. *J Neurosurg* 21:358-363, 1964
25. Saeki N, Rhoton AL Jr: Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. *J Neurosurg* 46:563-578, 1977
26. Sano K, Jimbo M, Saito I, Basugi N: Artificial embolization of inoperable angioma with polymerizing substance, in Pia HW, Gleave JRW, Grote E, Zierski J (eds): *Cerebral Angiomas. Advances in Diagnosis and Therapy*. Berlin: Springer-Verlag, 1975, pp 222-229
27. Seeger W: The artificial embolization of inoperable angiomas, in Pia HW, Gleave JRW, Grote E, Zierski J (eds): *Cerebral Angiomas. Advances in Diagnosis and Therapy*. Berlin: Springer-Verlag, 1975, pp 213-221
28. Serbinenko FA: Balloon catheterization and occlusion of major cerebral vessels. *J Neurosurg* 41:125-145, 1974
29. Smith CG, Richardson WF: The course and distribution of the arteries supplying the visual (striate) cortex. *Am J Ophthalmol* 61:1391-1396, 1966

30. Stein BM, Wolpert SM: Surgical and embolic treatment of cerebral arteriovenous malformations. *Surg Neurol* 7:359-369, 1977
31. Steiner L: Radiosurgery for arteriovenous malformations, presented at the Italian Neurosurgical Society, Rome, April, 1978
32. Steiner L, Leksell L, Forster DMC, Greitz T, Backlund EO: Stereotactic radiosurgery in intracranial arterio-venous malformations. *Acta Neurochir (Wien)* Suppl 21:195-209, 1974
33. Sven HJ, Mc Rae JA: Arteriovenous anomalies of the brain. Fate of patients not having definitive surgery. *J Neurosurg* 23:23-28, 1965
34. Tönnis W, Walter W: Die Indikation zur Totalexstirpation der intrakraniellen arteriovenosen Angiome. *Dtsch Z Nervenheilk* 186: 279-298, 1964
35. Troupp H, Marttila I, Halonen V: Arteriovenous malformations of the brain. Prognosis without operation. *Acta Neurochir (Wien)* 22:125-128, 1970
36. Trumpy JH, Eldevik P: Intracranial arteriovenous malformations: conservative or surgical treatment? *Surg Neurol* 8:171-175, 1977
37. Walder HAD: Application of cryotherapy in arterio-venous aneurysms. An experimental and clinical study. *J Neurol Neurosurg Psychiatry* 34:105, 1971
38. Walder HAD: Freezing arteriovenous anomalies in the brain, in Pia HW, Gleave JRW, Grote E, Zierski J (eds): *Cerebral Angiomas. Advances in Diagnosis and Therapy*. Berlin: Springer-Verlag, 1975, pp 183-193
39. Zeal AA, Rhoton AL Jr: Microsurgical anatomy of the posterior cerebral artery. *J Neurosurg* 48:534-559, 1978

Book Review

Clinical Neuro-ophthalmology, ed. 2

By Bryan Ashworth and Ian Isherwood, Oxford, St. Louis, Blackwell Scientific Publications, 1981
298 pp., \$86.00

Reviewed by J. Lawton Smith, Miami, Florida

This text is by Dr. Ashworth, neurologist at the University of Edinburgh, and Dr. Isherwood, Professor of Radiology at the University of Manchester, England; fundus illustrations were provided by Dr. Emanuel Rosen of the Manchester Royal Eye Hospital.

It is probably appropriate to begin by considering the positive features of the book. It is printed on high-quality paper, has an attractive appearance, size, and format, and the printing and illustrations are nicely reproduced. In my opinion, by far the best chapter in the book is chapter 8, "Radiology." The reproductions of the roentgenograms are good, the chapter is well written, and the section on computed tomography is reasonably up-to-date. Chapter 6, "Retinal Photography," is also good, and there is a nice series of color plates of various optic nerve problems. Chapter 3, "History," is well written, including such pertinent statements as, "The presence of relatives avoids the need to go over the story again, and it is always worth asking at the end if they have anything to add."

It must be added, however, that there are significant negative factors about this text that should be mentioned. The price is probably justified when one considers that there are 13 pages of colored illustrations in the book. There are, however, many points that are either inadequate or in error. For example, on page 13 a pinhole "1 mm. in diameter" is recommended. In my experience, this is much

too small. Again, concerning the pinhole test, the statement is made, "If the acuity is worse there may be a macular lesion." This does not mention the fact that any patient with opacity in the media, as well as a central scotoma, will also see poorer with a pinhole aperture than without it. On page 16 the statement, "If the diplopia is vertical, proptosis should be sought," certainly appears cryptic to me. Serological tests for syphilis usually mention the Wassermann reaction, and I did not see any mention of the FTA-ABS (fluorescent treponemal antibody absorbed) test in the text. Many would disagree with the statement on page 21 that, "The perimeter is of limited value in neurological work. . . ." Finger wiggling is advocated on confrontation on page 23, and many would think that could be improved. On page 23 the statement is made, "When a structural lesion is visible by ophthalmoscopy or there is opacity of the cornea, lens or media the value of recording the subjective field is limited." One would certainly have to modify this statement, I believe. There are many other examples of questionable observations which limitations of space do not allow me to comment on here. The description of the afferent pupil on page 12 is certainly open to question. The statement, "Constriction, followed at once by dilatation to the previous size, is known as the afferent pupillary sign . . .," is made without really relating the detection of an afferent pupil to the swinging flashlight test.

In summary, there are enough areas of question that many ophthalmologists would find in reading this book that I should express reservations about encouraging its purchase. It might, however, serve as a worthwhile introduction for a junior medical student starting a neurosurgical or neurology rotation.