THE SURGICAL TREATMENT OF ARTERIOVENOUS MALFORMATIONS OF THE BRAIN

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The methods used in the treatment of arteriovenous malformations of the brain have recently been summarized by Norlén,7 who reported a series of 10 cases in which the lesion was totally excised. Falconer3 and Bassett2 have each reported several cases in which treatment consisted of occluding the arterial supply without excising the malformation. Jaeger6 described a case in which he ligated the middle cerebral artery just distal to its origin from the carotid for a vascular malformation in the distribution of the middle cerebral. Occlusion of one or more afferent arteries was used by French and Peyton4 in 3 patients with arteriovenous malformations in the region of the vein of Galen.

Between March 1953 and February 1955, 9 arteriovenous malformations of the brain were seen in the Department of Neurological Surgery at Walter Reed Army Hospital. Surgical treatment was performed in all cases and consisted of total removal in 6 with various other methods used in the remainder. All of these patients except 1 were examined postoperatively by carotid arteriography. The choice of the operative procedure varied depending upon several factors.

In 2 of our patients, treatment consisted of occluding a single afferent vessel supplying the lesion. Arteriographic study showed the vascular malformations in these patients to be deeply seated in the frontal and parietal lobes where surgical approach directed at complete excision of the lesion would have resulted in extensive destruction of brain tissue. The blood supply in each case was seen on the arteriograms as a single vessel readily accessible for ligation. In the parietal lobe malformation (Case 1), this vessel was the terminal portion of the pericallosal artery. After ligation of this vessel, the possibility of some blood supply from the posterior cerebral still remained. However, we felt that the malformation was sufficiently removed from the distribution of the posterior cerebral to make this very improbable. The postoperative carotid arteriograms showed no evidence of filling in the malformation. The frontal lobe lesion (Case 2) was supplied by a large branch of the callosal marginal artery, and the arteriograms, following liga-

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tion, showed slight mottling of contrast medium only in the late venous phase. This method of treatment appears in such selected cases to be effective and obviates or minimizes the destruction of brain tissue that would result from total removal.

Obstructive hydrocephalus is a common complication of arteriovenous malformations in the region of the third ventricle. One such lesion (Case 9) was encountered in our series and the Torkildsen operation was used effectively in treatment. This particular patient presented a history of headache of 2 months' duration with no neurological abnormalities except for bilateral papilledema. The ventriculograms were sufficiently indicative of a colloid cyst of the third ventricle to warrant surgical exploration. No evidence of a cyst was found, but a pulsating vessel estimated to be 3 or 4 mm. in diameter was clearly visualized passing transverse to the long axis of the ventricle just posterior to the massa intermedia. The vessel was doubly clipped. We felt this to represent unequivocal evidence of an arteriovenous malformation with block of the third ventricle and/or aqueduct of Sylvius. There was no evidence of the malformation on the postoperative carotid arteriograms; we presumed the lesion to be in the distribution of the posterior cerebral artery. The papilledema had almost completely disappeared at the end of the second postoperative week. Vertebral arteriograms were not made. Gillingham has reported treating an arteriovenous malformation of the midbrain with a ventriculocisternostomy.

In their series of 48 patients with arteriovenous malformations, 24 of whom were operated upon, Olivecrona and Riives cited 5 cases in which the external carotids were greatly dilated. They believed operative treatment was particularly hazardous when the external carotid contributed to the malformation. We encountered one particularly unusual case (Case 8) in which there were bilateral parietal lobe malformations in the parasagittal area, with the major portion of the blood supply being derived from tremendously enlarged external carotid branches on either side. The latter vessels produced two 7- to 8 mm.-wide channels in the skull on either side. The intracranial lesion communicated through a small channel in the skull with a pulsating angioma of the scalp overlying the region of the sagittal sinus. The intracranial lesions were completely excised after the preliminary ligation of the external carotid arteries. We feel the latter is an important measure.

Total removal was performed when the malformation presented on the convexity of the cerebral hemispheres, or when the lesion was below the surface but was accessible through the cavity remaining after the evacuation of an intracerebral hematoma which was the result of hemorrhage from the malformation. Four of our cases fall into the latter category.

The following are summaries of our cases, together with preoperative and postoperative arteriograms. Unless otherwise noted, arteriograms were made with 35 per cent Diodrast injected percutaneously, using local anesthesia.