Total excision of an arteriovenous malformation of the corpus callosum

Case report

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Cerebral arteriovenous malformations involving the corpus callosum are usually regarded as inoperable because of their location. The first publication of a surgical attempt on such a lesion seems to be that of Basset in 1951. Recently, Seljeskog, et al., described the complete removal of a similar lesion which involved the inferior sagittal sinus in an infant. Dany, et al., reported in the French literature three cases operated on with good results. In only one, however, was the angiomatous malformation removed completely; in the other two, control postoperative angiography disclosed fistulous vessels remaining. We are reporting the total excision, under operative angiographic control, of an arteriovenous malformation of the corpus callosum.

Case Report

A 7½-year-old boy was referred for surgery on March 3, 1968, with the diagnosis of pericallosal arteriovenous malformation. One year earlier he had been admitted to this hospital in deep coma. Large subhyaloid hemorrhages were then observed in the ocular fundi; the pupils were maximally dilated and non-reacting; there was decerebrate rigidity, which later on resolved into left spastic hemiplegia in extension. The cerebrospinal fluid (CSF) was grossly hemorrhagic. Right cerebral angiography demonstrated a shift of the anterior cerebral artery to the left and a cluster of vessels at the anterior part of the pericallosal artery (Fig. 1). Left carotid and vertebral angiography excluded alternative blood supply to the malformation. The boy was in coma for 2 days and subsequently remained lethargic for 3 weeks. He then gradually recovered consciousness. He was discharged on April 3, 1967, with markedly spastic left hemiparesis. Since that time he had complained occasionally of severe headaches and blurred vision, sometimes diplopia, but not further episodes of loss of consciousness.

Examination. On admission to our depart-

Fig. 1. Right cerebral angiogram, lateral view, showing shift of the anterior cerebral artery to the left and a cluster of vessels at the anterior part of the pericallosal artery (March 2, 1967).
ment the child was frail and pallid; he responded to questioning with lucid and intelligent answers. He dragged the left leg heavily and complained of dizziness and headaches, particularly when walking. There was a severe left spastic hemiparesis with sustained ankle clonus and a left Babinski response. There was papilledema but no hemorrhages or exudates on the right; the left ocular fundus was normal. The visual fields were full and the visual acuity corrected was 6/6 for each eye. The general physical examination was noncontributory. Blood pressure was 110/70 mm Hg. The electrocardiogram was normal. Skull and chest x-ray films revealed no abnormality. The electroencephalogram was noticeably disturbed, showing bursts of sharp waves over a background of slow wave abnormal activity over the right hemisphere. Combined air and angiographic studies were performed on March 12. Cerebral angiography now showed a noticeable growth of the arteriovenous malformation (Fig. 2). In addition, a large paramedian porencephalic cavity communicating with the lateral ventricle was demonstrated on the pneumoencephalogram (Fig. 3).

Operation. On March 12, a right frontotemporal flap was turned down under endotracheal anesthesia. A Polyethylene P.E. 160 catheter was advanced into the internal carotid artery for angiographic control curing operation. Controlled respiration and Mannitol were used but no hypothermia or hypo-