SURGICAL EXPERIENCES WITH ARTERIOVENOUS ANOMALIES OF THE BRAIN*

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It is a matter of record that there is no more formidable lesion dealt with by the neurologic surgeon than the arteriovenous anomalies encountered in the brain. In the first comprehensive work on this subject, Cushing and Bailey were extremely dubious as to whether or not these lesions would ever be amenable to direct surgical therapy. However, as better methods of hemostasis were developed through the evolution of the electro-surgical unit, reports appeared demonstrating the feasibility of therapeutic attack. Dandy, Norlén, Olivecrona, Pilcher, Ray, and Trupp and Sachs have made available their experience with the angiomas.

These abnormal remnants of the rapidly changing embryonic circulation of the developing brain usually come to the attention of the clinician during the late second and third decades of life although they do occasionally become symptomatic before that time. The usual story is one of the development of focal fits which gradually increase in frequency and severity, becoming generalized and often uncontrolled with more than adequate medical therapy. The development of headaches of increasing frequency and severity and, in our experience, repetitive episodes of subarachnoid hemorrhage are the next most common symptoms and signs in that order. Hemorrhage may also be intracerebral and in such an instance usually produces irreversible damage.

The greatest factor facilitating surgical treatment of these lesions has been the introduction and development of angiographic techniques. Precise localization and, most important of all, demonstration of major afferent arterial components are usually clearly defined. This is particularly important when the angioma is situated in the dominant hemisphere or in the depths of the brain in a position not favoring direct attack upon the lesion. Routine roentgenograms of the skull may frequently demonstrate an associated anomalous blood supply to the skull itself associated with the underlying vascular anomaly of the cortex (Fig. 1). These lesions also have a characteristic defect demonstrable by pneumography. Olivecrona has pointed out the importance of focal atrophy demonstrated by air study in the absence of other roentgen findings, particularly when the evidence for atrophy is associated with focal neurological signs compatible with such a lesion. In this group of cases air study in most instances was undertaken without the true nature of the lesion being suspected. Aside from the atrophy

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as mentioned, these lesions present a typical bizarre type of deformity (Figs. 4, 5, 10, and 13).

Our experience with the arteriovenous anomalies of the brain is represented by the following 18 cases.

REPORT OF CASES

Case 1. C.P. was a 30-year-old white male admitted to the University Hospital on April 25, 1939 with the complaints of headache increasing in frequency and severity and Jacksonian fits involving the left hand and leg, the latter having begun 1 year previously, increasing in frequency and severity. Pertinent findings were bruit over the right parietal eminence and low-grade papilledema. A right parietal flap was turned down revealing an extensive angiomatous deformity on the surface of the right parietal lobe. Multiple surface ligations were done with silk and silver clips. Electrocoagulation of the available vessels was carried out. No attempt was made to dissect out the anomaly. A left hemiparesis was present postoperatively; however, the bruit was entirely gone and was still absent 2 months later. The hemiparesis completely disappeared and has not recurred. He continues to have an occasional seizure.

Case 2. B.C. was an 8-year-old white female admitted on Oct. 6, 1939 with a story of persistent headaches and vomiting since infancy. There was a bluish discoloration over the left frontal bossa. There had been numerous nosebleeds. One year previously the patient had had a subarachnoid hemorrhage. Two weeks prior to admission she had onset of severe, almost constant, right Jacksonian fits, recurrence of severe nosebleeds, and gradual lowering of consciousness and development of a right hemiparesis. There was a definite tumeescence of the left frontal bossa, the eyelids, and the left side of the nose. A tangle of abnormal vessels was demonstrable in the retinal circulation on the left. There was a right hemiparesis. On Oct. 26, 1939 ligation of the left common and external carotid arteries was done and on Nov. 13, 1939 the left middle cerebral artery was partially occluded with silver clips. Her symptoms remained more or less unabated. She became progressively worse. Her hemiparesis became a total hemiplegia, headaches increased and fits persisted. She had become more or less vegetative as far as her mentality was concerned. She was last seen on Dec. 16, 1946, a helpless invalid.

Case 3. C.W. was a 37-year-old white male admitted on Oct. 29, 1941 with the story that he had had headaches as long as he could remember and during the 3 preceding years had had onset of left Jacksonian attacks involving both the arm and leg and visual disturbances in the left homonymous field. The fits could not be controlled with medication. Pertinent findings were a mild left hemiparesis, a definite bruit over the right occipital parietal region, and a left homonymous hemianopsia. Routine x-rays of the skull demonstrated a typical calcified lesion (Fig. 1). On Oct. 30, 1941, a total ligation of the right internal carotid artery was done. He was last seen on Dec. 6, 1941. There was a marked diminution of bruit; hemianopsia and hemiparesis were still present. Headaches were absent and the fits were better controlled on medication. Attempts at follow-up have been unsuccessful.

Case 4. W.W. was a 30-year-old white male admitted on Dec. 17, 1942, having