THE FATE OF PATIENTS WHO HAVE CEREBRAL ARTERIOVENOUS ANOMALIES WITHOUT DEFINITIVE SURGICAL TREATMENTS

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INDICATIONS for operation in any given condition must be drawn from the balance remaining between the risks attending surgery, not only to life but to function, on the one hand, as opposed to the risk to life and to function if surgery is denied. This probably is no more true than in the case of arteriovenous anomalies of the brain, in which the operative mortality rate is at least 11.7 per cent, and in which the risk to function is real because of the fact that these lesions so frequently lie in the sylvian and rolandic fissures.

The literature on the fate of these patients for whom no definitive surgical procedure has been carried out is scanty and conflicting. We have therefore reviewed our series of cases from the standpoint of natural history and course of the pathologic process.

Between 1930 and 1954, 51 patients with proved cerebral arteriovenous anomaly have been studied at the Mayo Clinic. The presence of the lesion was verified either by surgical exploration or by cerebral angiography. We rejected, for purposes of this review, a considerable number of cases in which there was strong reason to suspect that an arteriovenous anomaly was present, but in which adequate verification was not obtained. Of this group of 51 patients, 23 received no definitive treatment. The surviving patients of this group have either been re-examined during 1954 or have answered questionnaires regarding their present status and their histories during the interval. In those cases in which the patients’ answers by letter were not deemed satisfactory, the attending physician was consulted and he supplied the necessary information.

The analysis of these cases will be considered from the standpoint of the usual symptoms presented by these patients.

INTRACRANIAL BLEEDING

Olivecrona wrote that “in the end, probably most, if not all patients die of hemorrhage or are completely incapacitated.” He also wrote that almost

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half of his patients had a history of one or more hemorrhages with hemiplegia, which was often transient, although some degree of motor or speech defects usually remained. Dandy\(^2\) wrote that death resulted from cerebral hemorrhages in 40 per cent of these cases. In Mackenzie's\(^4\) series of 50 patients, 15 presented a history of intracranial hemorrhage. Bassett\(^1\) reported on 18 patients, 11 of whom had subarachnoid hemorrhages, some of them with recurrent bleeding, but he thought that the hemorrhages rarely were fatal and that they were not so devastating as those caused by rupture of an aneurysm. Gould and associates\(^3\) encountered subarachnoid bleeding in 15, and intracerebral bleeding in 9, of 41 patients (an incidence of intracranial bleeding of 56 per cent). The mortality rate from hemorrhage was 20 per cent of the entire series.

In our series of 23 patients, 9 (39.1 per cent) have had intracranial bleeding. Seven of these patients have had subarachnoid hemorrhages and 2 had what appeared historically and clinically to have been intracerebral hematomas.

Of the 7 patients who have had subarachnoid bleeding, 2 have died, both in their first attack; one of these patients (Case 13) had had temporal-lobe attacks for 2 years, while the other (Case 19) had had jacksonian convulsions on the average of two times a year for 22 years. The mortality rate from intracranial bleeding in this group of 23 patients is thus 8.7 per cent.

One patient (Case 4) has had three attacks of subarachnoid bleeding, the first one occurring as the first symptom of her disorder in 1932. The last episode of bleeding occurred in 1942. None of these attacks produced crippling symptoms, and at the time of this report she was in good physical and mental health. In 2 patients some degree of partial hemiparesis developed with their attacks of subarachnoid bleeding. One of these (Case 23) 5 years later had only slight hemiparesis and was working full time, as this report was written. The other (Case 22) became moderately hemiparetic at the time of his subarachnoid hemorrhage, and 5 years later wrote that his hemiparesis was no worse and that he was in good health, except for this disability. The other 2 living patients (Cases 5 and 20) both sustained the bleeding in 1941, and had not had subsequent hemorrhages as this was written. Both were working full time and were in good health, except for occasional episodes of convulsions.

Thus, of the 5 patients who survived subarachnoid hemorrhage, 3 have no residual paralytic or speech defects and 2 have some degree of residual hemiparesis. Examination of Table 1, in which are depicted the locations and approximate sizes of these lesions, suggests that in each instance extirpation of the lesion very probably would have been accompanied by rather marked hemiparesis, with speech defects, in addition, in 2 instances (Cases 4, 5, 20, 22 and 23).

Two patients, as judged on the basis of examination and findings, had intracerebral hemorrhages. One of these patients (Case 29) had had headaches for 21 years before examination in 1941. At that time she had impairment of memory and bilaterally choked optic disks. She was last examined