THE ANEURYSMAL ORIGIN OF NONFATAL SUBARACHNOID HEMORRHAGE
AN ANGIOGRAPHIC SURVEY OF 53 CASES
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In 1947, an analysis was reported1 of 130 cases of spontaneous subarachnoid hemorrhage found in the records of the Buffalo General Hospital between the years of 1929 and 1945, inclusive. The cause of the bleeding was determined at autopsy or at operation in 47 cases. In 44 cases (93.6 per cent), the bleeding originated from intracranial aneurysms. The statement was made, “It seems reasonable to assume that in the unproved cases, the percentage due to aneurysms would parallel that of the proved cases.” Hyland2 also said that “the accumulated evidence . . . indicates a sufficiently high incidence of demonstrable aneurysms as the cause of this clinical syndrome to warrant consideration of the prognosis in unverified cases in terms of aneurysm.” Martland3 found that practically all cases of fatal subarachnoid hemorrhage are caused by rupture of intracranial aneurysms. He felt, however, that most of the cases of spontaneous subarachnoid hemorrhage with recovery (roughly 50 per cent) “are probably not due to ‘leaking’ or ruptured intracranial aneurysms, but rather to some unexplained infection or to some as yet unknown cause.”

Since our earlier report, cerebral angiography has been employed with increasing frequency in diagnosis of cases of subarachnoid hemorrhage not immediately fatal. It was considered interesting to determine to what degree this adjuvant was helpful in the diagnosis of subarachnoid hemorrhage.

In 324 patients subjected to cerebral angiography in the Buffalo General Hospital between 1947 and 1951, inclusive, were 53 in whom the test was made for spontaneous subarachnoid hemorrhage. Unfortunately, it was not at first realized that, to be as conclusive as possible, the entire intracerebral vascular tree must be visualized. The 53 tests were done by injection of both carotids and a vertebral artery in only 3 cases, of both carotids in 22, of 1 carotid in 26, of a single vertebral artery in 1 case, and in 1, no successful vascular visualization was obtained. In cases in which only one carotid artery was injected, the test was made on the side suggested by the patient’s clinical signs.

In the 53 cases of subarachnoid hemorrhage, angiograms disclosed aneurysms in 22 cases (41 per cent) and an arteriovenous malformation in 1 case. The aneurysms were on the carotid artery in 7 cases, carotid bifurcation (anterior cerebral-middle cerebral branching) in 2, anterior cerebral in 6, anterior communicating in 2, middle cerebral in 4 and several vessels were
involved in 1 case. In 4 other cases the angiograms did not indicate aneurysms that were found later at autopsy. These were on the posterior communicating artery in 1 case, and on the anterior cerebral-communicating complex in 3. In another case a unilateral carotid injection revealed 2 aneurysms on the left middle cerebral artery, and a third was found on the contralateral middle cerebral artery at autopsy.

In the 27 cases of aneurysms verified to date, 4 (15 per cent) were missed by angiography as it was employed in this series. It obviously is not possible to determine in how many other of the remaining 26 patients with subarachnoid hemorrhage, aneurysms were missed in like manner.

In the group of 27 patients having proved aneurysms, 10 of the angiograms were performed with bilateral and 17 with unilateral carotid injections. Three of the 4 unvisualized aneurysms were found in patients who had had bilateral injections. In the fourth case the injection was made on the side on which the proximal anterior cerebral artery was congenitally absent; the aneurysm was on the anterior communicating artery irrigated by the uninjected carotid. Three of the 4 unvisualized aneurysms involved the anterior communicating artery. The fourth was found at autopsy embedded in a cerebral peduncle, but its involved vessel was not identified. In this case both carotids and a vertebral artery were injected for angiography.

**DISCUSSION**

In a series of patients surviving subarachnoid hemorrhage, a smaller percentage of aneurysms (41 per cent) was disclosed by angiography than in a series studied at autopsy (93.6 per cent).

While other factors may be involved in the production of spontaneous subarachnoid hemorrhage, aneurysms can be demonstrated to be responsible for the bleeding in a high percentage of the patients who recover from a given episode, as well as in the fatal cases.

In this series subarachnoid hemorrhage was caused by aneurysms in 26 cases and by an arteriovenous malformation in 1 case.

Despite its shortcomings and its imperfect utilization in this series, angiography is the best adjuvant available at present for identification of the lesions responsible for subarachnoid hemorrhage.

Visualization of the entire intracranial vascular tree is necessary to disclose the largest possible number of aneurysms.

Even complete angiography does not insure that all aneurysms will be visualized. This is understandable on the basis of the dynamics of fluid flow in tortuous tubes, the size of some of the lesions and on the likelihood of partial thrombosis in some of the aneurysms. It is likely that repetition of the angiograms after an interval of time would visualize aneurysms in some cases.

In this series, aneurysms of the anterior communicating artery were the ones most frequently unvisualized by angiography.
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REFERENCES

