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Amnesia after Operations on Aneurysms of the Anterior Communicating Artery

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All patients with ruptured aneurysms admitted to neurosurgical departments in Denmark after April, 1978, are the subjects in a prospective study that includes neuropsychological examinations. Data from 48 patients with aneurysms of the anterior communicating artery have been analyzed. Trapping of the aneurysm was done in 11 patients and resulted in an amnesic syndrome in 9. Thirty-seven patients were operated upon by ligation of the neck of the aneurysm or similar procedures, resulting in 6 cases of amnesia. Trapping invariably disrupts blood supply through newly described dorsal perforating branches from the anterior communicating artery. These perforating branches may supply areas of vital importance to memory function.

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The six neurosurgical departments in Denmark have entered a collaborative study of intracranial ruptured aneurysms. Clinical data are recorded prospectively, and both preoperative and postoperative computerized tomographic (CT) scans and angiographic studies are available in most cases. The study includes neuropsychological examinations done three months and two years following operation.

The purpose of this paper is to discuss our finding of an amnesic syndrome in some patients operated on for aneurysms of the anterior communicating artery, and to present a possible explanation for its occurrence.

Materials, Methods, and Results

The present investigation comprises 48 consecutive patients operated on for aneurysms of the anterior communicating artery in four facilities during two years. Four additional patients were not available for neuropsychological examination.

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Based on the results from the examination done three months following operation, 15 of the 48 patients were judged as suffering from an amnesic syndrome, e.g., severe and relatively isolated defects of memory. This assessment was done without knowledge of the neurosurgical technique. The criteria for the diagnosis of amnesia were based on objective tests and included: (1) a normal or only slightly narrowed memory span; (2) defective verbal and/or visual learning capacity and memory; and (3) absolutely or relatively intact general level of intellectual function. Faulty orientation was considered typical but not mandatory for the diagnosis. The incidence of postoperative amnesia differed among the departments, ranging from 1 case of 12 from one department to 5 of 10 from another.

The surgical technique consisted of either ligation of the aneurysmal neck or trapping of the aneurysm (Fig. 1). Trapping was used in 11 cases and resulted in 9 cases of amnesic syndrome, whereas among the remaining 37 patients operated upon with ligation of the aneurysmal neck and similar procedures, only 6 cases of amnesia were encountered.

The 15 amnesic patients were extensively reexamined two years following operation. On formal testing, no improvement was seen, although some of the patients appeared better oriented and some obviously had better memory of personal affairs. Only 2 of 3 patients who were considered only mildly amnesic (memory impairment not incompatible with working capacity) had been able to return to gainful employment; 6 were moderately amnesic (very little learning in formal testing, variable orientation); and 6 were severely affected (unable to retain sufficient information for orientation in time and place).

Signs of more generalized intellectual impairment, including some reduction in abstract reasoning, were present in most patients, but usually to a mild degree. Among other deficits, lack of drive and initiative were common. Two patients showed signs of hypothalamic involvement. In 2 other patients a slight hemiparesis was present, whereas in the remaining 13 the neurological examination was essentially normal.

Discussion

The introduction of the operating microscope and microsurgical techniques to the treatment of intracranial aneurysms has reduced the associated mortality and mor-

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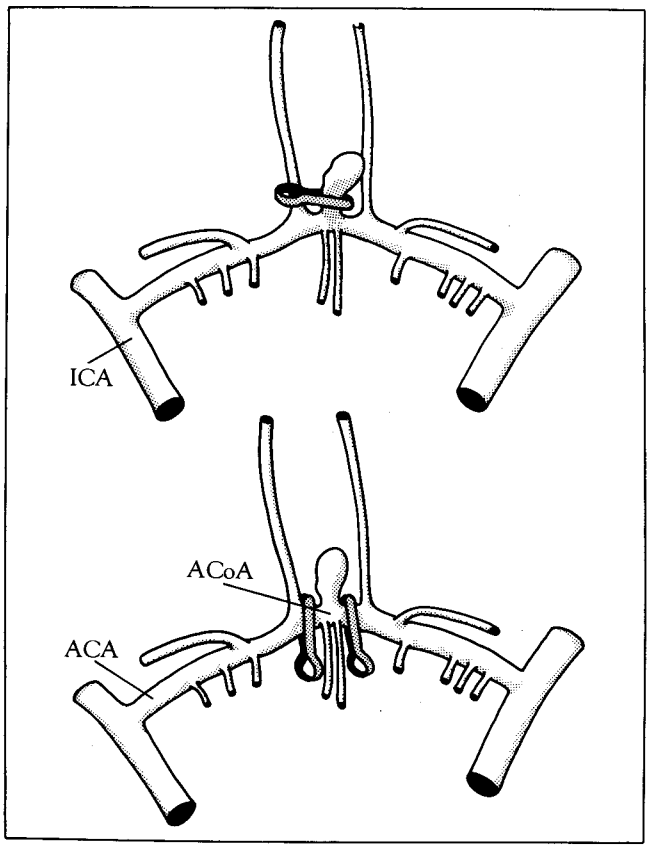

Fig. 1. Anterior part of the circle of Willis. Both drawings show the internal carotid arteries (ICA), *the anterior cerebral arteries* (ACA), *and an aneurysm located on the anterior communicating artery* (ACoA). *In the upper picture a ligature of the aneurysmal neck has been performed, sparing perforating branches from the anterior communicating artery. The lower picture shows trapping of the aneurysm and of the perforating branches from the anterior communicating artery. Nine of 11 patients operated upon by trapping developed the amnesic syndrome.*

bidity [25]. However, permanent cerebral damage due to bleeding, arterial spasm, or the surgical intervention, is common, and ruptured aneurysms remain a major challenge to neurosurgeons [18].

Roughly 30% of ruptured intracranial aneurysms originate from the anterior communicating artery [10, 18]. In a recent clinical analysis of a large series of patients, psychological symptoms were recorded as the most frequently observed postoperative complication. Such symptoms occurred almost three times more frequently with these aneurysms than with aneurysms of the internal carotid or middle cerebral arteries. Disorientation and amnesia accounted for the majority of the psychological symptoms [20].

An amnesic syndrome may occasionally follow a spontaneous subarachnoid hemorrhage, with a recorded incidence of 2 to 3% [15, 23]. The association between an amnesic syndrome and surgical treatment of anterior communicating artery aneurysms was first noted by Swedish neurosurgeons [14]. Lindqvist and Norlén [9] found in a later analysis of a consecutive series of 33 patients with such aneurysms that 17 were amnesic after the operation, in 5 cases with a chronically severe course. A similarly high incidence of postoperative amnesia was reported by Okawa and co-workers [16].

No comprehensive study of the nature or cause of this condition has been carried out. Talland and associates [21] followed 2 cases, with experimental studies of memory functions carried out for two to three years, and noted the similarity of the condition with the Wernicke-Korsakoff syndrome. These authors suspected that the damage in the 2 patients extended to the circuit of Papez, and argued that the lesions might result from bilateral ischemia involving tissue in the posterior inferior medial frontal areas. Luria [11] also reported 2 cases of severe amnesia after operations on anterior communicating artery aneurysms. The results from experimental neuropsychological studies of these patients indicated that the amnesia was global in nature. Although unclear concerning possible causes, Luria seemed to favor an explanation in terms of either arterial spasm or, in patients with permanently severe amnesia, hemorrhage spreading to the region of the floor of the third ventricle.

Other authors have implicated damage to frontal and cingulate cortical structures, either due to resection of the gyrus recti [13] or, more commonly, due to a circulatory deficiency in the territory supplied by the two anterior cerebral arteries [1, 2, 7]. However, in view of the known neuropathology of amnesia with other etiologies [12, 22] involving mesial temporal and periventricular structures, it is difficult to accept the suggestion that lesions of the prefrontal and cingulate cortices are responsible for amnesia after operations on aneurysms of the anterior communicating artery.

Older anatomical studies seem to indicate that the anterior communicating artery has no or only a single variable branch [3, 6]. Yaşargil and colleagues [25], however, using the operating microscope, found several branches, and this has been confirmed by anatomical studies [4, 5, 17, 24]. The majority of these branches originate from the dorsal surface of the anterior communicating artery, and thus are difficult to see during surgery, even when a microscope is used. Most of them enter the brain in the anterior perforated substance and in the suprachiasmatic area. The branches are shown in Figure 1 and further illustrations may be found in works from Rhoton's laboratory [19, 24]. Between 3 and 13 branches were found in each of 10 autopsy cases. Most branches were small, 50 to 250 μ, but at least 1 larger branch of 250 to 1,000 μ was present in every case [4]. It is important to note that the areas of supply seem to be structures along the anterior wall of the third ventricle (as shown in Figure 2), probably including the lamina terminalis, the anterior hypothalamus, the optic chiasm, the mesial part of the anterior commissure, the septal area, col-

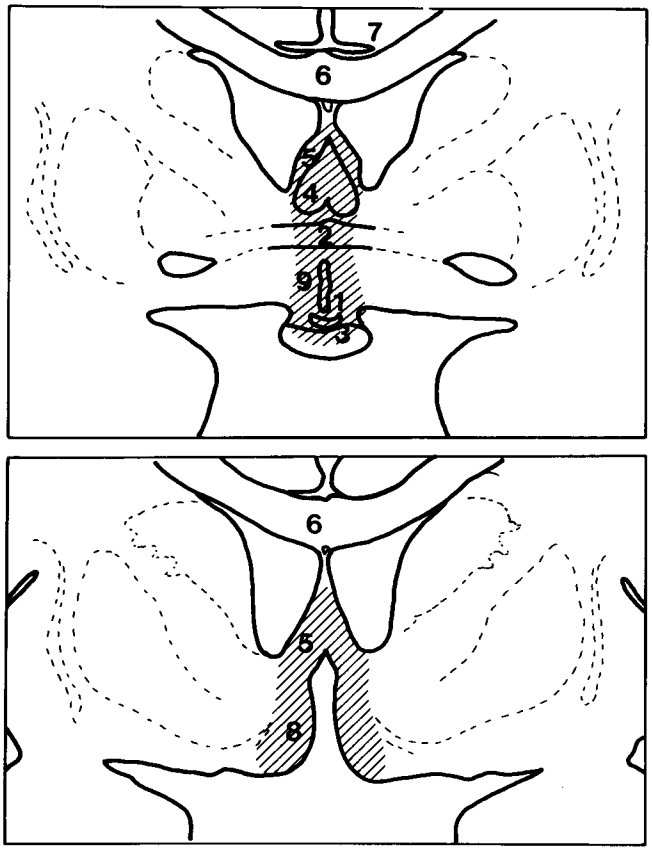

Fig. 2. Coronal sections of the brain at the level of the anterior commissure (above) and septal nuclei (below). The areas of supply from perforating branches of the anterior communicating artery, as determined by infusion of contrast agent [22], are indicated by crosshatching and are identified by numbers: (1) lamina terminalis; (2) mesial anterior commissure; (3) optic chiasm; (4) columns of the fornix; (5) septum; (6) corpus callosum; (7) cingulum; (8) subcallosal area; and (9) anterior hypothalamus. The supply is limited to midline structures.

umns of the fornix, the subcallosal areas, and possibly even parts of the corpus callosum and the cingulum [4, 5].

On the basis of this new anatomical evidence, I suspect that disruption of the blood supply through the perforating branches of the anterior communicating artery may cause the amnesic syndrome. Such disruption invariably occurs with the trapping procedure. Regrettably, previous published accounts of amnesia after operations on aneurysms of the anterior communicating artery contain no information on the surgical procedures [9, 16]. Yaşargil, however, reported 8 cases (of a series of 203 such aneurysms) of permanent psychoorganic brain syndrome, with confusion, disorientation, dementia, and lethargy, 4 of which had been treated by trapping [25]. In the series reported by Lindqvist and Norlén [9], 14 patients had been treated by trapping, resulting in 12 cases of amnesia, which was severe and permanent in 5 patients [8].

It is conceivable that the observed association between trapping and amnesia might be spurious. In the present series, arterial spasm appeared to be slightly more common in the 15 amnesic patients than in the 33 nonamnesic, yet the association was not close. Basal medial frontal areas showing low absorption on CT scans are equally common in amnesic and nonamnesic patients. These and other clinical and neuroradiological parameters of potential significance will be reported in later publications. So far, I have not been able to see or conceive of any other possible cause of amnesia in these patients than lack of blood supply through the branches of the anterior communicating artery. Obviously, it is of great theoretical interest to determine the nature of the lesion causing amnesia in these patients. Perforating arteries from other parts of the circle of Willis to the optic chiasm and anterior hypothalamus are numerous [5, 6], but little is known about possible alternative supply to other structures supplied by branches of the anterior communicating artery. The determination of which areas are critical in the development of amnesia after operations on aneurysms of the anterior communicating artery may require neuropathological studies.

The evidence for an association between damage to the perforating branches from the anterior communicating artery and disabling amnesia appears sufficiently strong to indicate that the procedure of trapping such aneurysms should be avoided when possible, and care must be taken when dissecting around the aneurysm to spare these important branches.

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