THE PATHOLOGIC ANATOMY OF RUPTURED CEREBRAL ANEURYSMS

GEORGE WILSON, M.D., HELENA E. RIGGS, M.D., AND CHARLES RUPP, M.D.
Laboratory of Neuropathology, Philadelphia General Hospital, Philadelphia, Pennsylvania

(Received for publication July 17, 1953)

INCREASING interest in the surgical treatment of cerebral aneurysms suggests that an evaluation of the potentialities of this form of therapy might be aided by a review of some of the anatomic findings in 143 autopsied cases of subarachnoid hemorrhage caused by leaking miliary or berry aneurysms of the circle of Willis.

The sex and age distribution in this group differed significantly from that in cases of apoplexy previously reported. Women predominated 91 to 52; 31 per cent of the patients were under 40 years of age as compared to 9 per cent in the apoplectic group. The youngest patient was 14 and the oldest 94.

The aneurysms were all located above the carotid siphon on the circle of Willis, its peripheral branches or its basivertebral component. Forty-eight involved the anterior communicating artery, 51 the internal carotid, 30 the branches of the anterior, middle or posterior cerebral arteries, and 14 the basivertebral stems. The left half of the circle was involved in 54 cases, the right in 51 and the midline in 38 (Fig. 1).

The aneurysmal sac originated at arterial branchings, although not necessarily at a bifurcation, in 101 cases and by outpouchings from the vascular stem in 42. When the aneurysms involved the origin of an artery, particularly the anterior choroidal from the internal carotid, the distal portion of the vessel was frequently continuous with the fundus of the sac.

Multiple Aneurysms. Multiple aneurysms were present in 27 patients, bilaterally placed in 13 and in the midline as well as on one or both sides in 7. Ruptured and unruptured sacs were found on the same artery in 2, on the

* Read in part at the First International Congress of Neuropathology, Rome, September 13, 1952.
same side of the carotid stem in 7 and on the basivertebral branches in 7.

Meningeal Hemorrhage. Bleeding into the subarachnoid space was found at necropsy in 137 patients. Evidence of ancient leakage was noted in 6 patients dying of causes unrelated to aneurysmal rupture. Bleeding into the basal cisterns occurred in the majority of cases but was confined to the convexity on the side of the aneurysm in 12, to the median fissure in 7 and localized to the immediate region of the sac in 10. Subdural extension occurred in 22 cases.

Continued leakage or recurrent hemorrhage from the aneurysmal sac apparently occurred in 121 patients. Resolution of the bleeding, manifested by staining of the meninges and hemolyzed blood around the basal structures, was found in only 6 patients, 2 of whom died within 5 days after the onset. Recent bleeding or fresh hemorrhage superimposed on older clots was present in 26 patients who had been hospitalized longer than 1 week and 12 with symptoms of 2 to 6 weeks’ duration. Signs of older leakage, obviously antedating the episode of acute bleeding, could be demonstrated in many instances. Bronzing of the meninges, infiltration of tissue around the sac by blood pigment and collections of similar pigment along the basal vessels were noted in 13; envelopment of the aneurysm, wholly or partially, by a pseudo sac in 22; and dense adhesions between the sac and the arteries or adjacent structures in 25.

Parenchymatous Lesions. Destruction of brain tissue, more extensive than erosion or hemorrhage around the sac, complicated the rupture of the aneurysm in 94 cases. Frank hemorrhage occurred in 29 cases, as a subcortical hematoma contiguous to the aneurysm in 21, and deep within the brain structures at a distance from the sac in 8. Focal hemorrhagic or anaemic infarction involving both cortical and deep structures, frequently in more than one area, was present in 65.

Disregarding the nature of the parenchymatous destruction (hemorrhage or infarction), the site of the lesion, in the majority of cases, could be correlated with the location of the aneurysm on the cerebral vascular tree.

Aneurysms arising from both the anterior cerebral and the anterior communicating arteries were associated with damage to the medial portion of the frontal lobe and the corpus callosum, with additional involvement of the orbital portion of the frontal lobe and the caudate nucleus in cases of aneurysms of the anterior communicating artery. The cortex and subcortex of the convexity were involved with aneurysms of the middle cerebral in the Sylvian fissure. Lesions in this area, as well as of the lenticular region and the subependymal tissue around the inferior horn of the ventricle were present with aneurysms of the internal carotid. Destruction within the cerebellar hemisphere and damage to the retro-olivary portion of the medulla, respectively, was noted with two aneurysms arising from the basivertebral stem, one at the origin of the superior cerebellar and the other at the origin of the posterior inferior cerebellar. Lesions of the midbrain and upper pons occurred with aneurysms of the divisional branches of the basilar.

In general, the parenchymatous damage associated with leaking aneu-