Drake and colleagues in several hundred patients with Grade I aneurysm. In the present study, 4 new postoperative deficits appeared after rupture with forced vascular clipping, vasospasm, or edema. These postoperative deficits might have been exacerbated by hypotension, which was sometimes protracted.

In this study, EEG did not identify intraoperative local ischemia in 3 patients with immediate postoperative focal deficits. Several reasons may explain the failure of EEG to predict these deficits. Most importantly, infarction caused by hypotension was not identified in these patients, and thus we lacked a clear-cut hypotensive stress for detection by EEG surveillance. Moreover, standard EEG electrodes could be placed only in the area over the unoperated scalp and, therefore, the area of potential maximum vulnerability was not monitored.

EEG with scalp electrodes near but outside the surgical site does not seem helpful for monitoring cerebral function in the region of aneurysm surgery. Preliminary studies in our operating room suggest that electrocorticography over the operative area will be more revealing.

References
the patient appeared chronically ill, was incontinent of urine, and exhibited moderate bilateral lower extremity weakness and hyperreflexia. He was oriented to person only and had great difficulty in following simple commands. No disturbance of cranial nerve function was noted, although his mental state made the sensory examination difficult. The blood pressure was 155/88 mm Hg.

Lumbar puncture obtained xanthochromic fluid with an opening pressure of 120 mm water, 2100 red blood cells per cubic millimeter, and protein and glucose concentrations of 1000 mgm and 44 mgm per deciliter respectively. A left retrograde brachial arteriogram suggested a preaxial mass displacing the basilar artery posteriorly; the luminal profile of the artery was only slightly irregular and no aneurysm was appreciated (fig. 1). A CT scan disclosed only mild bilateral ventricular dilatation, but, with contrast enhancement, revealed a 1.5 X 3 cm vascular blush in the region of the brain stem (fig. 2).

Because an extra-axial neoplasm, such as a meningioma, was suspected, the prepontine region was approached surgically. A fibrous-walled mass filled with clotted blood was encountered. Postoperatively, the patient did poorly and died 3 days later.

Autopsy Findings

Extensive atherosclerosis thickened and yellowed the large vessels at the base of the brain. The rostral basilar artery shared in this process, but was otherwise unremarkable. Caudally, however, at a point one centimeter distal to its origin, this vessel expanded abruptly into a discrete, brown, globoid mass which incorporated the terminal segments of both vertebral arteries. Positioned slightly to the right of the midline, it flattened the anterior medulla and bilaterally displaced cranial nerves IX through XII (fig. 3). A cut section of the mass made apparent its constitution of clotted blood, and the preservation within of the eccentrically placed lumen of the basilar artery. The latter was normal in caliber, but occluded by fresh thrombus (figs. 4 and 5).

There was a diffuse, intense, rusty color of the leptomeninges and superficial 1 mm of parenchyma of the basal surface of the cerebrum, brain stem, and cerebellum (fig. 3). The entire spinal cord, and cranial nerves III–XII, were characterized by a similar, but circumferential, involvement. The lateral ventricles were slightly dilated.
Multiple histologic sections through the basilar mass disclosed the hematoma positioned between the adventitia and the intima. Fragments of medial smooth muscle were at one point external to, at another internal to, and elsewhere within, the mass of blood (fig. 5). No point of communication was apparent between the intraluminal thrombus, which was acute, and the intramural hematoma, which was subacute. Serial sectioning, however, was not done. The hematoma was not vascularized.

A large infarct of several days duration softened the base of the pons. There was compression atrophy of both medullary pyramids, especially the right, and Wallerian degeneration of the lateral corticospinal tracts which was most intense on the left. Only occasional senile plaques were noted in the frontal cortex and hippocampus.

Microscopically, the superficial rind of discoloration of the spinal cord and the base of the brain was apparent as an extensive deposition of hemosiderin (fig. 6). In addition to this granular iron pigment within the cytoplasm of macrophages and astrocytes, there was also a diffuse agranular background iron staining. Structural alterations which accompanied this pigmentation included moderate spongiosis, slight astrogliosis, and scattered spheroids and ovoid bodies. Moderate neuronal loss was noted in the heavily pigmented basilar regions of the cerebral and cerebellar cortices. A sprinkling of lymphocytes populated the thickened spinal leptomeninges.

**Patient 2**

Clinical History

This 30-year-old white male had been in good health except for a 15-year history of migraine headaches which were characterized by either right or left hemicranial pain associated with nausea and vomiting and preceded by scintillating scotomas. Twenty-four hours prior to admission, he developed a headache typical of those previously experienced, left work early, and was discovered at home 3 hours later in a semiconscious state.

On admission to Duke University Medical Center, the patient was comatose, respired apneustically, and responded with decerebrate posturing to painful stimuli. The deep tendon reflexes were bilaterally, symmetrically 2-3 plus; bilateral Babinski responses and sustained ankle clonus were observed. His eyes remained fixed in the midline when his head was rotated to either the right or the left; the miotic pupils reacted to light. Caloric stimulation increased lateral movements of the ipsilateral and contralateral eyes. There was frequent ocular bobbing. The remainder of the physical examination was within normal limits. The blood pressure was 140/80.
The cerebrospinal fluid was under a pressure of 165 mm H_2O and contained 3 lymphocytes per mm. The protein and glucose concentrations were 37 and 117 mgm/dl respectively. An EEG disclosed slowing to the delta and theta frequencies with suppression of the rhythms in the left hemisphere.

A percutaneous right carotid arteriogram was thought to be normal. In retrospect, an indirect sign suggestive of a low pressure circuit in the vertebrobasilar system was present in the form of paradoxical full contrast-filling of the right posterior cerebral artery from the internal carotid vessel via a tiny posterior communicating connective ("dangling posterior cerebral artery"). During the next 3 weeks, the patient's neurological status changed little. He expired of bronchopneumonia on the 21st day after entering the hospital.

Autopsy Findings

There was a thrombus-filled, hourglass-shaped expansion of the distal 1.5 cm of the basilar artery which incorporated the orifices of the pontine perforating vessels and the superior cerebellar arteries (fig. 7). Multiple infarcts about 3 weeks old were found in the rostral pons, midbrain, thalami, occipital lobes, and superior cerebellar hemispheres.

The expansion of the basilar artery was the consequence of a circumferential intramural hematoma between the intima and the adventitia. The lumen was acutely thrombosed. The remaining parenchyma and vasculature were unremarkable; there was no intravascular edema or fibrosis as had been observed in the intracerebral vessels of some patients with migraine.

Discussion

The basilar artery lesion in each of these 2 cases represents an intracranial "dissecting aneurysm" — the enlargement of a major cerebral blood vessel by an intramural hematoma rather than by expansion of the lumen. In contrast both clinically and pathologically to the bifurcational outpouching of a saccular aneurysm and the fusiform ectasia of an atherosclerotic vessel, the dissecting lesions are uncommon causes of stroke or intracranial mass effect. They are heterogeneous with respect to location, pathogenesis, and the age of the patient (6 months–69 years). They exhibit little sex preference.

The dissecting aneurysm is a fusiform or abrupt expansion of a major intracranial vessel in or near the circle of Willis. The internal carotid,7, 8, 10, 11, 14, 16, 19, 27, 29, 31, 34, 36, 38, 41, 43, 49, 54 and middle cerebral,1, 9, 10, 12, 13, 10, 10, 18, 23-26, 33, 35, 36, 43, 44, 48, 57 arteries have been the most common sites. Involvement of the vertebral,6, 11, 29, 48 posterior cerebral or anterior cerebral30, 32, 31, 57 vessels has occurred less frequently. Angiographic studies have disclosed variable effects of dissecting aneurysms. The lumens of the parent vessels have most often been stenosed22, 44, 48, 50, 53 or occluded by thrombus10, 18, 20-22, 28-30, 38, 39, 41-43, 47, 48, 51, 54 but displacement13 has also been noted. Double lumens are recorded.27

When studied postmortem, the hematoma of the dissecting aneurysm is a circumferential or eccentric mass variably positioned within the arterial wall. A few have been located within the media15, 25, 28, 53 or intima,18 but most are found between these 2 layers in the subintima.3, 9, 12, 14, 16, 18-24, 27, 30, 31, 35, 38, 39, 43-46, 49-57 Although the hematoma is often presumed to develop as a dissection of blood from the lumen, a communication between the hematoma and the lumen has been identified in only 2 cases.10, 54

The suggested pathogeneses of intracranial dissecting aneurysms are diverse and include trauma, surgery, arteriosclerosis, migraine, infection, homocystinuria, congenital medial defects, "panarteritis", fibromuscular dysplasia, and strenuous physical exertion. Unlike dissecting aneurysms of the aorta, the intracranial lesion has only rarely been associated with cystic medial necrosis.

The cases incited by trauma7, 8, 30, 35, 39, 48, 50 have followed closed-head injury without fracture and presented with symptoms which were acute,7, 8, 50 or delayed,20, 23, 25, 29, 21 or relapsing.48 Surgical trauma has been identified as a pathogenetic factor in several cases.10, 18, 25 In one, surgical manipulation of the internal carotid artery during tonsillectomy was presumed to have initiated the dissection.18 In another, it occurred after ligation of a saccular aneurysm on the middle cerebral artery.13

Arteriosclerosis has been implicated as a promotor of intramural dissection in 4 cases3, 9, 11, 19, 54 and in our first case. The subdivisions of arteriosclerosis represented included "mucoid degeneration in the middle coat,"7 "arteriosclerosis with cystic medial degeneration,"9 "sclerotic left vertebral artery,"11 "segmental alteration of atherosclerotic origin,"13 and arteriosclerosis as in Patient 1 above.

The association of migraine and dissecting intracranial aneurysm was first made by Sinclair, who suggested that recurrent attacks of migraine produce medial edema and fibrosis which predispose to subintimal dissection.13 In our second patient, the temporal relationship between an attack of migraine and the terminal dissection supports the possible occasional relationship between these 2 disease states.

Other conditions that weaken or stress the arterial walls such as infection,1, 4-6 homocystinuria,15, 50 congenital medial defects,16, 24 "panarteritis,"48 and fibromuscular dysplasia,48 have been recorded in isolated cases. The association between strenuous physical exertion and dissection may be an expression of elevated arterial pressure.7, 16, 32, 42

Clinical manifestations of dissecting aneurysms appear most often in young adults of either sex as acute hemorrhage or thrombosis, with symptoms of the latter referable to the distribution of the occluded vessel7, 3, 5, 14, 17, 19, 27, 28, 30, 33, 35, 38, 39, 43, 46, 49-57 Patient 2 represents such a case.

Patient 1, on the other hand, is atypical because the symptoms express 1) the lesion's mass effect by compression of the pyramidal system and 2) the
consequence of chronic subarachnoid hemorrhage. Ventricular dilatation was apparent both pre- and postmortem, so that the patient's intellectual deterioration, ataxia, and incontinence could be attributed to a low grade communicating hydrocephalus. The leptomeningeal fibrosis, which could account for this obstruction, was associated with a heavy meningeal and parenchymal deposition of iron, or "superficial hemosiderosis", and raises the possibility that these symptoms, as well as the hearing loss, could reflect the parenchymal alterations presumed to result from the deposition of this iron or other plasma substances.24 This uncommon entity of siderosis has complicated hemorrhage from a variety of intracranial lesions including saccular aneurysms, neoplasms, vascular malformations, the Sturge-Weber syndrome, and hemispherectomy. Characteristic are superficial deposits of iron pigment with local gliosis and neuronal loss.25, 26

The diagnosis of the dissecting aneurysm depends on awareness of the entity. In Patient 1, this lesion was unsuspected because of the deceptive appearance of a preserved basilar artery lumen and an apparent preaxial mass. The CT scan, furthermore, suggested a lesion intrinsic to the neuraxis because of the blush in the compartment normally occupied by the posteriorly displaced brain stem. In Patient 2, the basilar artery occlusion by the dissection could have been visualized by vertebral angiography.

References

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ALTERATIONS OF VISION have been reported by 10–15% of patients afflicted with migraine. Visual disturbances may occur immediately prior to or during the episode of severe headache. Symptoms described include homonymous hemianopsia, unilateral or bilateral scintillating scotomata, micropsia, or macropsia, bilateral amaurosis or monocular amaurosis. Symptoms are often stereotyped and related to specific ocular or brain arterial territories.

While some have questioned the relationship of pure monocular amaurosis to migraine, adequate evidence has been presented in the literature confirming the existence of this relationship. The visual symptoms are almost always transient. However, several reports have emphasized permanent visual dysfunction in association with migraine and actual occlusions of the central retinal artery and its branches, as well as secondary partial or complete optic atrophy.

A variety of drugs have been employed to prevent migrainous head pain with reasonable success in most patients. Little attention, however, has been paid to the question of prevention of the associated visual symptoms, in part because they are usually transient and much less disturbing to the patient than the headache. Some patients are disturbed primarily by the transient visual symptoms and prevention of permanent sequellae, such as hemianopsia, may be a major therapeutic concern. This report describes 4 patients with classical migraine and associated transient visual disturbances. Treatment of all these patients with isoproterenol at the onset of each subsequent episode prevented further visual dysfunction in 3 and reduced the incidence of visual disturbance in the fourth. Two additional untreated patients suffered permanent hemianopsia. The pathophysiology and treatment of this serious complication of migraine is discussed.

Transient Visual Disturbances

Patient 1. A 53-year-old white female was seen at the Bellevue Hospital with complaint of 2 episodes of vision loss in the left eye during the previous week. Each episode began with throbbing pain in the left temporal area followed by gradual loss of vision moving from...
Dissecting aneurysms of the basilar artery in 2 patients.
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