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ANGIOGRAPHIC IMPLICATIONS IN DIAGNOSIS AND PROGNOSIS OF BASILAR ARTERY OCCLUSION*

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BASILAR artery occlusion often presents a confusing clinical picture and the diagnosis rests upon angiographic findings. Angiographic examinations were made, without complication, on 9 patients who had total occlusion of the basilar artery.

This report compares the angiographic findings with the clinical data and evaluates these findings as prognostic indicators. Complete occlusion of the basilar artery does not necessarily result in death. Six of the 9 patients in this study have survived.

CLINICAL MATERIAL AND OBSERVATIONS

Five of the 9 patients were men, 4 were women. Age range was from 10 to 74 years with an average age of 49 years. The duration of symptoms preceding angiography was from a few hours to 10 years. Five of the patients had symptoms suggesting basilar artery occlusion for over 1 year. In each of these cases the patients had had complete recovery from initial symptoms. Three other patients had transient symptoms for several weeks prior to angiography. One patient underwent angiographic examination after an episode of unconsciousness and died the same day.

The most common symptoms were alteration in consciousness, vertigo, ataxia, dizziness, dysarthria, hemiparesis or hemiplegia, and visual disturbances. All 9 patients were hemiparetic or hemiplegic at some time during the course of their symptoms. Eight patients had speech difficulty and visual dysfunction. Seven patients complained of vertigo or dizziness. According to the clinical records, despite the character of the symptoms, basilar artery occlusion was unsuspected prior to angiography in 7 of the 9 cases.

Through the follow-up period of 3 months to 5 years, 6 of our patients have survived—some show only minor neurologic deficit. One patient, mentioned above, died the day of angiography. Another, whose condition became progressively worse, died 3 weeks after angiography. The third patient died from a brain abscess secondary to meningitis, 2 months after angiography (Fig. 1). In each instance necropsy confirmed the roentgenographic finding of basilar artery occlusion.

ROENTGENOGRAPHIC FINDINGS

Angiography of 1 vertebral artery and both carotid arteries was performed for all but 1 patient. In those instances in which no reflux could be achieved into the opposite vertebral artery, both vertebral arteries were injected. Because of her poor condition, the vertebral study was not performed in 1 patient.

In all but 2 patients (Fig. 2, *A* and *B*), the site of occlusion was distal to the origin of both posterior inferior cerebellar arteries. In these 2 patients, one of the posterior inferior cerebellar arteries was involved in the occlusion. Occlusion was distal to the origin of the anterior inferior cerebellar arteries in 1 patient (Fig. 3). In all instances, the occlusion extended to a site just proximal to the origin of the superior cerebellar arteries.

The posterior cerebral and superior cerebellar arteries were opacified from various pathways in all 9 patients. When carotid arteriography was performed in 7 patients,

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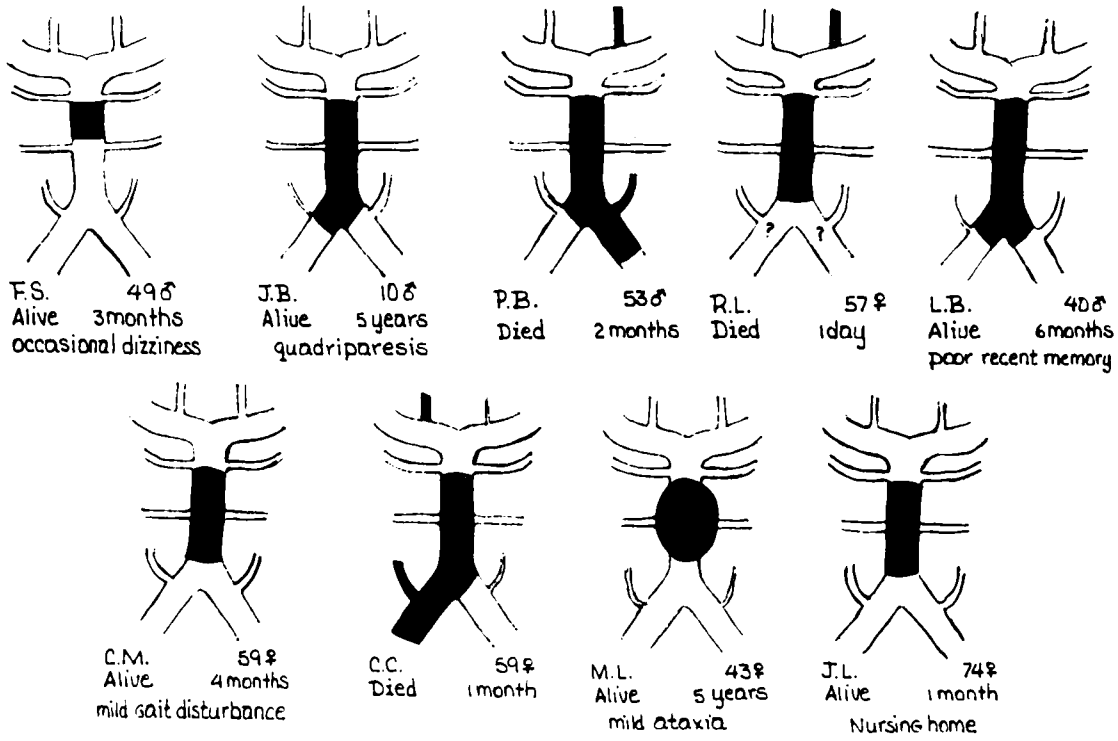

FIG. 1. Schematic drawing indicates the site and extent of occlusions of the basilar artery in 9 patients. Follow-up periods are noted.

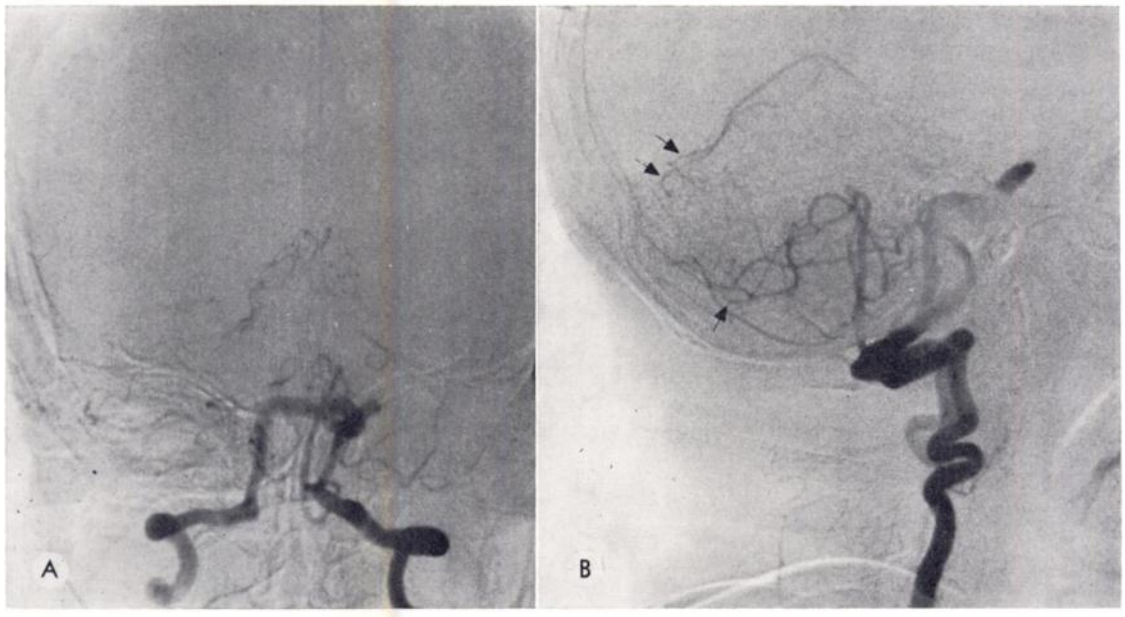

FIG. 2. Left vertebral angiograms. (*A*) Anteroposterior and (*B*) lateral views show reflux into the distal, right vertebral artery. The basilar artery is occluded just distal to the origins of the posterior inferior cerebellar arteries. Note collateral filling of the superior cerebellar artery *via* vermian branches of the posterior inferior cerebellar artery (arrows).

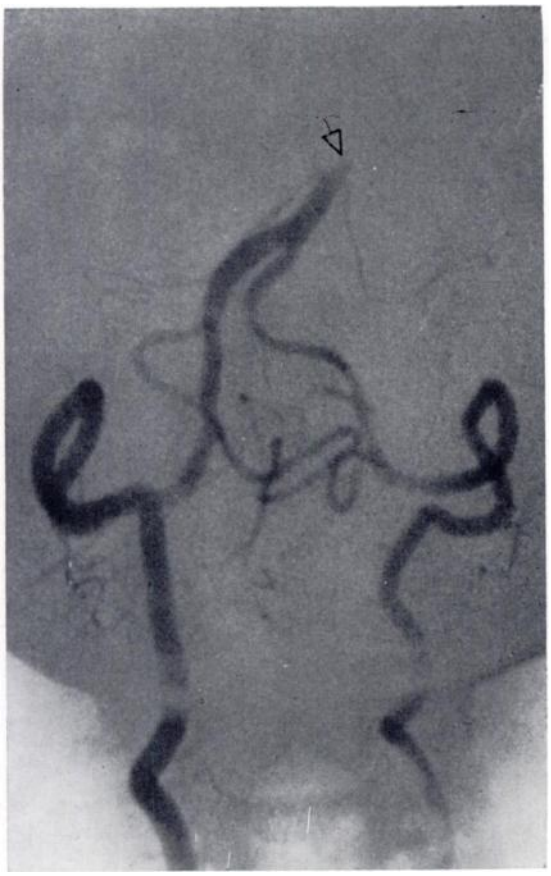

FIG. 3. Right vertebral angiogram shows occlusion (arrow) of the basilar artery immediately distal to the origins of the anterior inferior cerebellar arteries.

both posterior cerebral arteries were filled by patent posterior communicating arteries. The superior cerebellar arteries subsequently filled *via* the tip of the basilar artery (Fig. 4, *A* and *B*). In those 2 patients in whom the posterior communicating arteries carried no flow, the posterior cerebral arteries were filled in a retrograde fashion *via* pial anastomoses between posterior branches of the middle cerebral artery and posterior branches of the posterior cerebral artery (Fig. 5, *A–D*). As in the other 7 patients, the superior cerebellar artery was demonstrated after the contrast medium reached the basilar tip.

In 5 of 9 patients, filling of the superior cerebellar arteries was achieved on the vertebral angiogram as a result of anastomoses between vermian and hemispheric branches of the posterior inferior cerebellar artery and corresponding branches of the superior cerebellar artery (Fig. 2, *A* and *B*). Posterior meningeal branches of the vertebral artery contributed to collateral flow in 3 patients (Fig. 6). In 1 patient (Fig. 7), an hypertrophied tentorial branch of the meningohypophyseal trunk added to the flow. This same patient had an enlarged anterior spinal artery which provided an additional supply of collateral blood (Fig. 8).

Two patients had pneumoencephalography. One showed a large calcified aneurysm in the region of the midportion of the basilar artery (Fig. 9). The thrombosed aneurysm did not fill at angiography. The other patient had enlargement of the prepontine cistern, indicating atrophy of the pons.

REPORT OF CASES

CASE I. A 59 year old woman was admitted to the hospital after having been found comatose by her husband. She had been hospitalized twice in the preceding 18 months for episodes of stupor, unsteady gait, and slurred speech. Despite multiple tests no diagnosis had been reached. Since her previous admission, she had done well and had returned to work complaining only of occasional dizzy spells.

At physical examination her eyes were conjugatively deviated to the left. Corneal reflexes were absent. Although the patient was able to open and close her eyes on command, she was unable to move her extremities. Sustained ankle and knee clonus were elicited. Babinski's reflex was exhibited bilaterally. The face, trunk, and extremities showed reaction to noxious stimuli.

Cerebral angiography indicated complete occlusion of the basilar artery.

While in the hospital the patient showed progressive improvement. Follow-up examination 4 months after angiography showed only minor disturbance in gait. The patient was otherwise asymptomatic and neurologically normal.

CASE II. A 49 year old man was admitted to the hospital with right hemiparesis and right-sided numbness, dizziness—with falling to the right, and dysarthria. He had been well until

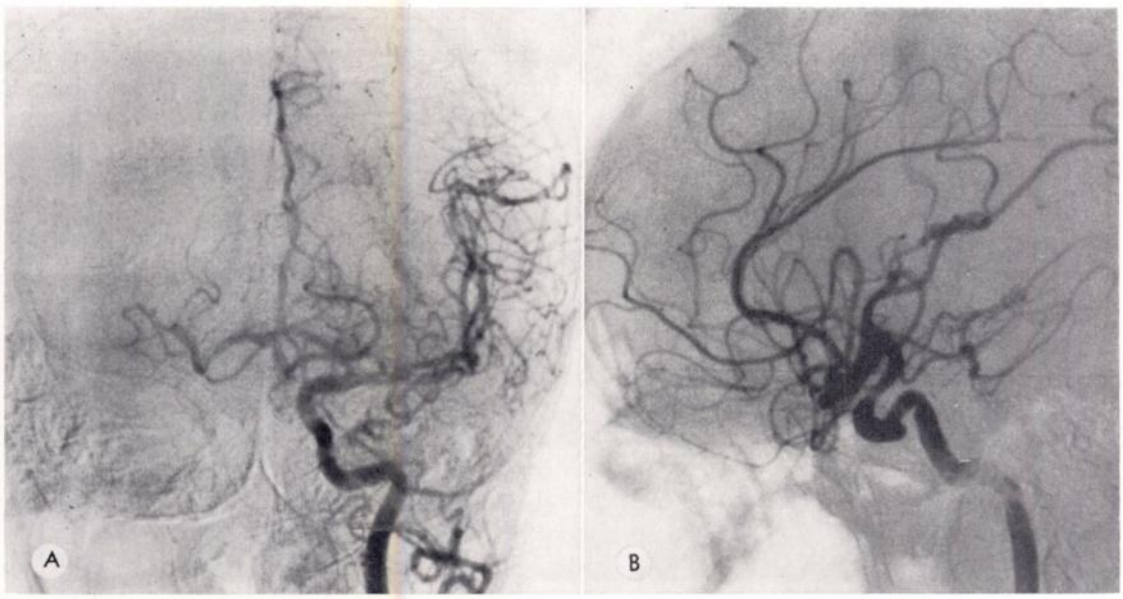

FIG. 4. Left carotid angiograms. (*A*) Anteroposterior and (*B*) lateral views demonstrate filling of posterior cerebral artery and superior cerebellar artery from the posterior communicating artery.

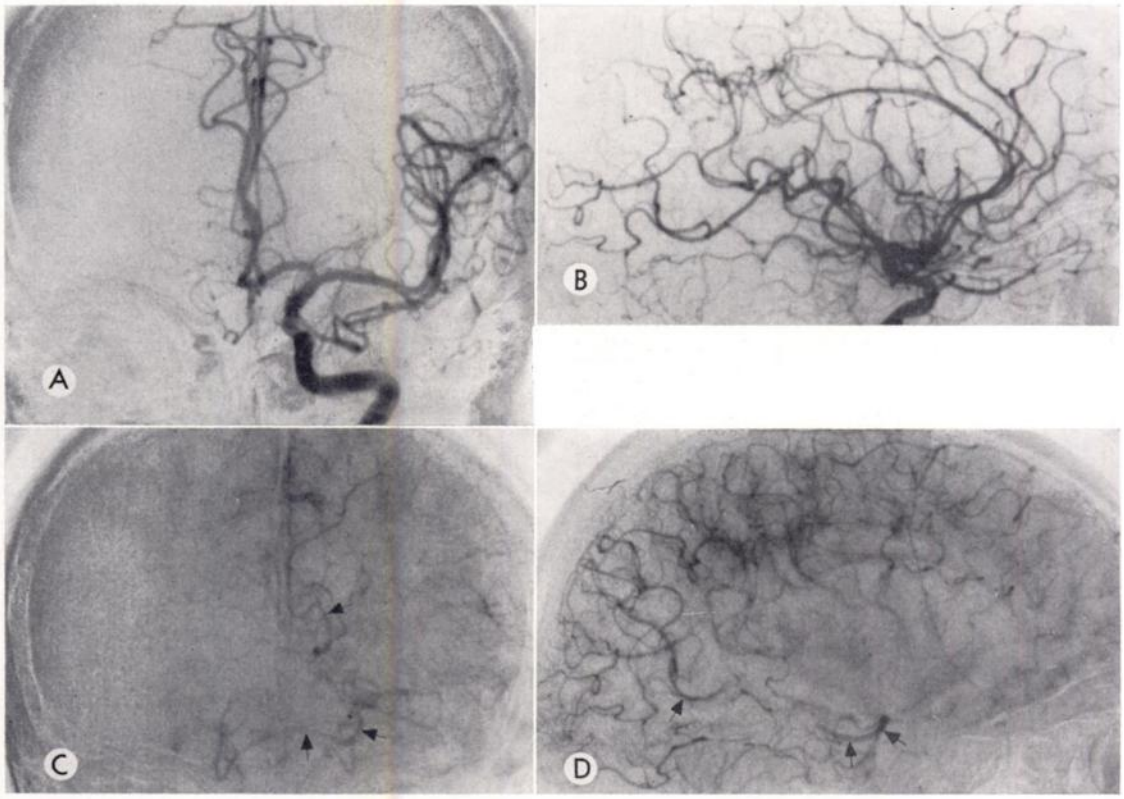

FIG. 5. (*A*) Anteroposterior and (*B*) lateral left carotid angiograms in early arterial phase show no filling of posterior cerebral artery from the injection of the carotid artery. (*C*) Anteroposterior and (*D*) lateral left carotid angiograms, late arterial phase, indicate retrograde filling of posterior cerebral artery (arrows) from posterior branches of the middle cerebral artery. The superior cerebellar artery fills after opacification of the basilar tip.

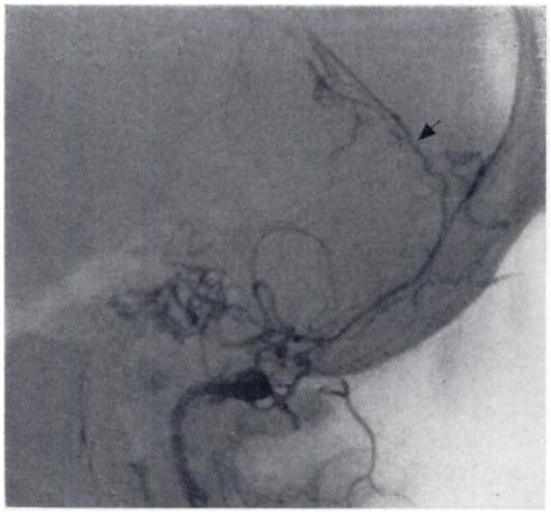

Fig. 6. Right vertebral angiogram shows proximal occlusion of the basilar artery. There is poor filling of the posterior inferior cerebellar artery. Collateral supply to the superior vermian branch of the superior cerebellar artery by means of the posterior meningeal branch of the vertebral artery is demonstrated (arrow).

3 weeks before admission when he had a sudden onset of dizziness, nausea, vomiting, diplopia, and left-sided weakness. This episode lasted only a few minutes, then completely cleared. In the interval between the onset of symptoms and admission, he had had several such transient episodes with alternating hemiparesis.

Physical examination showed no abnormality. Neurologic examination revealed central facial weakness on the right and a mild weak-

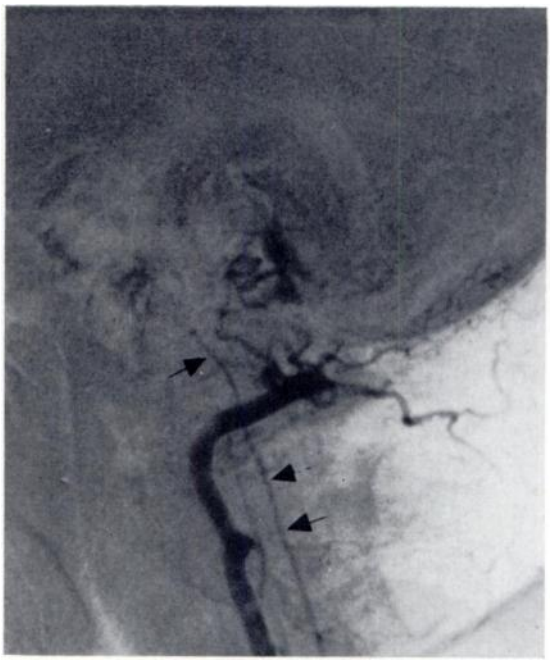

Fig. 8. Left vertebral arteriogram shows an enlarged anterior spinal artery (arrows) serving as collateral blood supply to the brain stem.

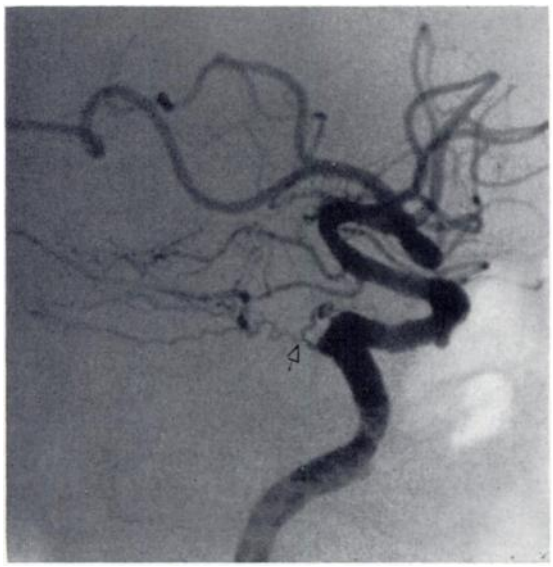

Fig. 7. Left internal carotid arteriogram shows a hypertrophied tentorial branch (arrow) of the meningohypophyseal trunk adding to the collateral flow.

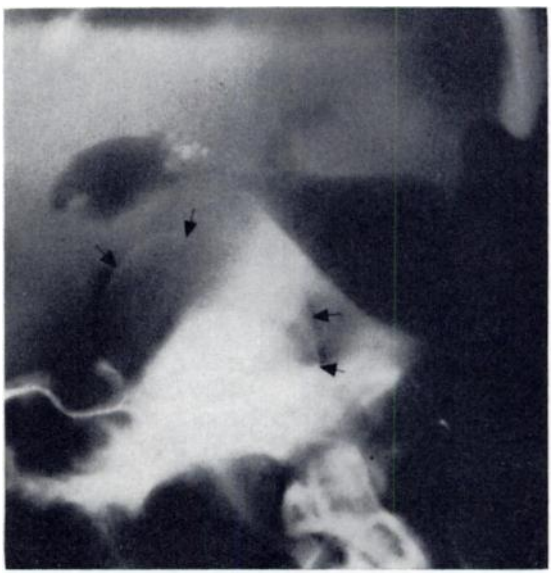

Fig. 9. Lateral pneumoencephalogram demonstrates a large calcified aneurysm of the basilar artery (arrows), which was totally occluded at angiography.

ness of the right extremities. The patient walked with a wide-based gait and tended to drift toward the right. Cerebral angiography disclosed complete occlusion of the basilar artery.

During his hospitalization the patient's symptoms cleared; he was discharged. Examination 6 months after angiography showed a mild, right hemiparesis. No other abnormalities were noted.

CASE III. A 47 year old man was admitted to the hospital for evaluation after a "blackout spell" occurred while he was driving his car. Seven years before he had been evaluated following 2 transient episodes of light-headedness and falling to the right side. No diagnosis was reached at that time.

Physical examination at the time of admittance indicated mild, left hemiparesis; no other neurologic abnormality was noted. Cerebral angiography indicated complete basilar artery occlusion.

Six months after angiography, the patient complained of slight clumsiness with his left arm and occasional dizziness. Physical examination was unrevealing.

DISCUSSION

Basilar artery occlusion and its accompanying clinical picture has been recognized for more than 100 years.[8,11] Before 1946, basilar artery occlusion was a retrospective diagnosis. Of 22 cases reported by Kubik and Adams in 1946,[10] 18 were of basilar artery occlusion with detailed correlation between symptoms and pathologic findings. The other 4 patients demonstrated characteristic symptoms; these patients survived. To our knowledge, however, the diagnosis was not confirmed. In 1956, Haugsted[7] made the angiographic diagnosis of occlusion of the basilar artery. Shortly thereafter (late 1950s) arteriographic findings for this condition were reported.[5,9,13] Until that time, it was generally accepted that basilar artery occlusion was catastrophic - usually fatal—and, if not fatal, capable of causing severe neurologic changes. In 1966 Fields and his coworkers[6] described 8 patients who survived basilar artery occlusion (follow-up period was 18 months).

Occlusion of the basilar artery is not rare. It was noted to appear in 1 of every 300 necropsy specimens at the Boston City and Massachusetts General Hospitals.[10] Of 4,834 necropsy examinations conducted at Bellevue Hospital in New York from 1938–1958, 0.2 per cent of the patients had had complete occlusion of the basilar artery.[3] Over a 5 year period at the Mayo Clinic, 1 of about every 450 necropsies revealed basilar artery occlusion.[15]

Although basilar artery occlusion has been reported to occur in all age groups, the majority of patients are over the age of 50. Men are afflicted slightly more often than women. The youngest recorded case is that of a 6 year old boy who had typical symptoms; the cause was undetermined.[4] We have demonstrated basilar artery occlusion in a 10 year old child. Schechter and Zingesser[14] reported basilar artery occlusion to have occurred in a 16 year old person.

The great majority of occlusions of the basilar artery are due to atherosclerosis. Occurrence of basilar artery occlusion due to embolus has been reported in various percentages. Kubik and Adams[10] reported 7 of 18 cases of basilar artery occlusion to be due to embolus; Cravioto *et al.*[3] reported 2 of 14. Many early-reported cases were caused by syphilitic endarteritis.[13] Other causes such as trauma and infections have been noted as well. One case of phycomycotic thrombosis of the basilar artery in a 23 year old patient with lymphoblastic leukemia has been reported.[2] Upon the death of one of our patients, necropsy disclosed thrombosis secondary to meningitis. Loop and his co-workers[12] described a basilar artery trapped and occluded in a fracture of the clivus. Thrombosis in a patient with polycythemia has been described, as well as has accidental ligation during a surgical procedure.[2]

Aneurysms of the basilar artery are not uncommon. No case could be found in the literature, however, of survival following complete occlusion of the basilar artery secondary to thrombosis of a basilar artery aneurysm. One of our patients, a 43 year

old woman, has survived for 5 years after the diagnosis of thrombosed aneurysm of the basilar artery.

Patients with basilar artery occlusion fall basically into 2 categories: (1) those with abrupt onset of coma and rapid progression to death; and (2) those with transient symptoms of brain stem ischemia. Patients in the latter group often present a problem in diagnosis and are of particular concern to radiologists.

The acuteness with which occlusion occurs is directly reflected in the outcome of the condition. In the case reported to be due to accidental ligation, the patient died within hours after the surgical operation.[1] In the instance in which the basilar artery was trapped within a fractured clivus, the patient quickly died.[12] The 7 patients of Kubik and Adams[10] who had embolic occlusion died much sooner than those who had atherosclerotic thrombosis. One of our patients who had suddenly become unconscious without antecedent symptoms died just hours after angiography.

To our knowledge no relation between angiographic findings and prognosis has been previously reported. Well established collaterals through the circle of Willis and anastomosis between branches of the basilar artery, however, certainly favor survival. In our series, the 2 patients in whom the occlusion involved one of the posterior inferior cerebellar arteries died within 2 months after angiography. Significantly, these 2 patients also had no flow through one of their posterior communicating arteries; this absence of flow necessitated retrograde flow from the middle cerebral artery to fill the posterior cerebral and superior cerebellar arteries. Our third patient who died had no vertebral angiogram; she too, however, had only one, small patent posterior communicating artery. The surviving 6 patients, had all major collateral pathways available. These 9 cases support the contention that a direct relationship exists between the availability of collateral pathways and survival.

SUMMARY

Angiographic examinations were made of 9 patients who had complete occlusion of the basilar artery. Six are alive 3 months to 5 years after diagnosis.

In 7 of the patients the diagnosis was not suspected prior to angiography. The commonest site of occlusion was just distal to the origins of the posterior inferior cerebellar arteries. Various patterns of collateral flow were observed.

A direct relationship is indicated between patency of collateral pathways and prognosis.

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