OCCLUSION of the basilar artery is generally considered a very serious event incompatible with normal survival. Kubik and Adams described 18 patients with brainstem infarction due to occlusion of the basilar artery discovered at postmortem examination and emphasized the abrupt onset and frequent fatal outcome. Marshall subsequently pointed out that many untreated patients with the clinical picture of posterior circulation vascular disease do not develop serious deficits; however, the underlying vascular pathology in this group of clinical patients was unassociated with a relatively benign clinical course. In occlusive disease of the posterior circulation, the critical period for deficit acquisition is at the time of occlusion. Extent of the deficit depends on the rapidity of development of adequate collateral circulation, and the presence of distal embolization at the time of occlusion. Some patients survive basilar occlusion without permanent deficit.

Occlusion of the Vertebral or Basilar Artery
Follow Up Analysis of Some Patients with Benign Outcome

LOUIS R. CAPLAN, M.D.

SUMMARY Ten patients with angiographically verified occlusion of the basilar or vertebral artery have been followed for an average of 2.75 years. None has developed further ischemia after the initial stroke, and 4 patients survived without any clinical deficit. In occlusive disease of the posterior circulation, the critical period for deficit acquisition is at the time of occlusion. Extent of the deficit depends on the rapidity of development of adequate collateral circulation, and the presence of distal embolization at the time of occlusion. Some patients survive basilar occlusion without permanent deficit.

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AICA branching). Four had an occluded vertebral artery. All basilar occlusion cases had collateral circulation involving the long circumferential cerebellar vessels (PCA, AICA, SCA) with late filling of the distal segment of the basilar artery. Patients with vertebral occlusion had a patent contralateral vertebral artery and basilar artery. Six patients had transient ischemic attacks (TIAs) prior to strokes. The time between the initial TIA and the stroke ranged from 1 week to 1 year (average 15 weeks). The last TIA always occurred within 1 month of the stroke (5 or 6 within 1 week). TIAs were usually multiple (range 1–30, average 11).

After onset of stroke, 8 patients had either progression of the deficit over a 2 or 3 day period, or had fluctuations of their clinical deficit during the first 2 weeks. In 4 of these 8 patients the clinical deficit fluctuated with alteration of position in bed (3 when elevated, 1 when turned to the left side). Two patients left the hospital without neurological abnormalities, 3 had slight deficits, 4 moderate and 1 severe.

Follow up ranged from ½ to 6 years (average 2.75 years). No patient developed a late increase in deficit referable to the posterior circulation, and in 6 patients clinical deficits improved during the period of follow up. Two patients with a slight deficit upon hospital discharge subsequently returned to normal. Six patients have been treated with long-term warfarin; 2 patients had temporary anticoagulant treatment (1 heparin for 2 weeks, 1 warfarin for 6 months). No patient has had a new stroke, but 1 patient died of a myocardial infarction 4 years after his pontine infarction.

**Table 1** Vertebrobasilar Occlusion-Deficit Profile

<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>Onset and course</th>
<th>Clinical signs</th>
<th>Deficit at hospital discharge</th>
<th>Severity of deficit at follow up</th>
<th>Follow up length</th>
<th>Angiographic lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Group I)</td>
<td></td>
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</tr>
<tr>
<td>1. 65, M</td>
<td>TIA 1 year before; 6 TIA’s within 2 wks., fluctuating deficit 1 week</td>
<td>dysarthria, clumsy right arm, intermittent INO</td>
<td>slight</td>
<td>none</td>
<td>2½ yrs.</td>
<td>Proximal basilar occlusion</td>
</tr>
<tr>
<td>2. 60, M</td>
<td>5 mos. before-3 TIA’s, 2 mos.-prolonged TIA, awakened with sudden deficit</td>
<td>left hemiplegia, pain in right eye</td>
<td>mod.</td>
<td>mod.</td>
<td>6 yrs.</td>
<td>Midbasilar occlusion</td>
</tr>
<tr>
<td>3. 59, M</td>
<td>many TIA’s over wks; fluctuating deficit for 2 wks.</td>
<td>bilat. VI, R VII, fluctuating level of consciousness</td>
<td>none</td>
<td>none</td>
<td>4 yrs.</td>
<td>Midbasilar occlusion beyond AICA</td>
</tr>
<tr>
<td>4. 46, M</td>
<td>30 or more TIA’s over 3 mos., grad. onset of deficit over hrs. with fluctuation for 2 weeks</td>
<td>transient quadriplegia and dysarthria</td>
<td>none</td>
<td>none</td>
<td>2½ yrs.</td>
<td>Proximal basilar occlusion</td>
</tr>
<tr>
<td>5. 26, M</td>
<td>4 TIA’s in 2 wks; sudden deficit</td>
<td>R hemiparesis, L ataxia, dysarthria, dysphagia</td>
<td>mod.</td>
<td>slight</td>
<td>1½ yrs.</td>
<td>Right vertebral occlusion; embolic occlusion of superior cerebellar artery and narrowing of PCA</td>
</tr>
<tr>
<td>6. 71, M</td>
<td>TIA 1 week before sudden deficit which fluctuated for 1 week</td>
<td>nystagmus, R VII, R ataxia, transient L hemiparesis</td>
<td>slight</td>
<td>none</td>
<td>1½ yrs.</td>
<td>Right vertebral occlusion at C1 level</td>
</tr>
<tr>
<td>(Group II)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. 44, F</td>
<td>Sudden deficit; later progressing over 48 hours</td>
<td>R Horner’s R palatal paralysis, diminished pain and temp. sensation R face and L body, R arm ataxia</td>
<td>mod.</td>
<td>mod.</td>
<td>3 yrs.</td>
<td>Right vertebral artery occluded intracranially</td>
</tr>
<tr>
<td>8. 58, M</td>
<td>2 abrupt deficits then fluctuated for one week</td>
<td>L VI, L ataxia, bilateral upgoing toes</td>
<td>mod.</td>
<td>slight</td>
<td>4½ yrs.</td>
<td>Left vertebral occlusion neck</td>
</tr>
<tr>
<td>9. 38, M</td>
<td>sudden deficit with fluctuation for 2 weeks</td>
<td>quadripareisis upgoing toes, pseudobulbar</td>
<td>slight</td>
<td>slight</td>
<td>6 mos.</td>
<td>Occlusion midbasilar artery; no filling of right vertebral artery</td>
</tr>
<tr>
<td>(Group III)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. 55, M</td>
<td>gradual evolution over 72 hours</td>
<td>quadriplegia, bilat. facial and tongue weakness</td>
<td>severe</td>
<td>severe</td>
<td>4 yrs.</td>
<td>Midbasilar occlusion beyond AICA</td>
</tr>
</tbody>
</table>
The patients fitted into 3 different patterns of onset and course (table). 1) The majority (6) (patients 1–6) had TIAs prior to stroke. The TIAs were usually multiple and increased in frequency as the stroke approached, with 5 of 6 patients having a TIA within a week of the stroke. The stroke usually began in the morning on rising, and symptoms and signs of brainstem, cerebellar and posterior cerebral artery territory dysfunction fluctuated for a period ranging between 2 days and 3 weeks. Clinical fluctuations were very sensitive to postural change. After a period of 2–3 weeks, the clinical deficit stabilized and no further deterioration occurred. 2) A second group of 3 patients (patients 7–9) had sudden onset deficits with only minor subsequent fluctuations over a period of 2–3 days. All had unilateral vertebral occlusions angiographically. 3) One patient (No. 10) had a progressive course over 3 days without TIAs or sudden onset.

Illustrative Cases

1. Patient 4

A 46-year-old man with known coronary artery disease had 30 or more episodes of vertigo and diplopia during a 3 month period. He awakened during December, 1975, with poor hearing and slurred speech and staggered when he attempted to walk. Recovery occurred within hours, but later the same day he became mute and quadriplegic while awaiting examination in the office of an otologist. Neurological examination within minutes revealed ocular bobbing and no voluntary or reflex horizontal gaze. There was severe facial, palatal and lingual paralysis bilaterally. The left limbs were flaccid, but the patient could feebly lift his right arm and right leg from the bed for a few seconds. He was placed in the Trendelenburg position and an intravenous infusion of heparin was begun. By the next morning, a right internuclear ophthalmoplegia (INO) and slight left hemiparesis remained; by day 7, examination showed return to normal. Angiography revealed occlusion of the proximal basilar artery; the left posterior inferior cerebellar artery (PICA) filled the left superior cerebellar artery (SCA) by branches coursing over the cerebellar hemispheres, leading to delayed filling of the distal basilar artery (figs. 1 and 2). A transient brief deficit (right INO and left hemiparesis) followed angiography. During hospitalization the patient was treated with intravenous heparin, subsequently changed to warfarin. He has had no further attacks or central nervous system deficits in the 2½ years since hospitalization. After 1 year, warfarin was discontinued. He has been maintained on dipyridamole 150 mg, aspirin 20 grains and clofibrate 2 gm daily.

2. Patient 1

A 65-year-old male with a remote myocardial infarction (20 years) complained of intermittent claudication of his legs. A year before hospitalization, he had a brief episode of dizziness with diplopia. Two weeks before admission an episode of diplopia lasted 5 minutes. During the week before hospitalization, there were 4 brief spells of dizziness with diplopia; on one other occasion he suddenly slumped to the ground without paralysis. An ophthalmologist whom he consulted discovered weakness of the right lateral rectus muscle. On the morning of admission in December, 1974, he noted dizziness, slurred speech and numbness, and weakness of his right limbs. A left
Horner's syndrome, left horizontal rotatory nystagmus, and right limb ataxia were present on examination. During the initial 3 weeks of hospitalization there was considerable fluctuation of his symptoms and signs, often after elevation of his head. On day 5, transfemoral vertebral angiography demonstrated complete occlusion of the proximal basilar artery with reflux from the left vertebral artery filling the right vertebral. Both PICAs were opacified. Treatment with intravenous heparin had been instituted shortly after hospitalization, but had to be stopped on day 14 when a large retroperitoneal hemorrhage developed. Upon hospital discharge at 4 weeks, the patient's only residual neurological deficit was slight instability of gait. He has had no transient episodes or strokes during the 3½ years since hospitalization and has been able to return to work in a restaurant. He has been maintained on aspirin 15 grains daily.

3. Patient 5

A 26-year-old man had several poorly defined episodes of dizziness, occasionally accompanied by weakness of the face or legs. Two weeks later (January, 1977) he awakened with dizziness and ringing in his right ear, and vomited. Later in the morning, he became unable to walk; his vision was distorted especially to the right; and he could not swallow. Blood pressure was 145/85. Present on examination were a left Horner's syndrome, left horizontal rotatory nystagmus, bifacial weakness, left greater than right, palatal and lingual weakness with severe dysarthria, moderate right hemiparesis, clumsiness of the left hand and increased deep tendon reflexes, right greater than left. CAT scan was normal. Angiography the same day revealed occlusion of the right vertebral artery intracranially. Injection of the left vertebral artery led to good basilar opacification but no filling of the left superior cerebellar artery; a narrowing was apparent at the orifice of the left posterior cerebral artery (figs. 3 and 4). Treatment with heparin was begun and was later changed to warfarin.

Several months after the stroke, the patient developed transient unilateral spells of numbness, first in his right, then left limbs. These were migratory, associated with headache, and disappeared after diphenylhydantoin was begun. The clinical impression was that the spells were migrainous. In April, 1977, he developed severe chest pain and an electrocardiogram revealed an acute myocardial infarction. Coronary arteriography revealed occlusion of the left anterior descending coronary artery, but no embolic source was identified by cardiac catheterization. Blood lipids were normal. A temporal artery biopsy was normal. He has had no further neurological symptoms 18 months after his stroke. Residual deficit consisted of slight slurring of speech and clumsiness of his left arm. He has been maintained on warfarin.

Discussion

Meyer et al. described the angiographic findings in 35 patients with occlusive disease of the posterior circulation and emphasized the frequency of abnor-
malities in the basilar artery itself. Clinical course and follow up data were not included in the report, which excluded “critically ill patients” or those with severe brainstem infarction. Nonetheless, angiography in 2 patients revealed clinically unsuspected complete occlusion of the basilar artery. Archer and Horenstein discussed the angiographic findings in 20 patients with basilar occlusion. Their clinical group consisted of patients with severe clinical deficits in contrast to Meyer’s criteria for selection. Fifteen of Archer and Horenstein’s patients were stuporous or comatose at the time of angiography and 2 were “locked-in”; 15 patients died, 4 were severely disabled and 1 patient had a moderately severe deficit (hemiparesis). These authors defined the locus of occlusion and the pathways of collateral circulation and sought to correlate the anatomy of the clinical deficit with angiographic findings. Caplan and Rosenbaum pointed out the utility of vertebrobasilar angiography in differentiating severe obstructive disease of the vertebral or basilar artery from “small vessel” disease within the posterior circulation, and emphasized the frequent benign outcome of many patients with clinical vertebrobasilar symptoms who had no major vascular lesions seen angiographically. Caplan and Rosenbaum also pointed out that patients with known basilar occlusion may survive without disabling neurological sequelae. The present report seeks to extend and explain that finding.

The initial period of TIAs, in group 1 patients, likely represents diminished flow referable to vascular stenosis. When occlusion of the vertebral or basilar artery becomes complete, a hemodynamically unstable situation develops in which collateral circulation must develop quickly or there will occur irreversible ischemia of brainstem, cerebellum, or posterior cerebral territory hemispheric tissue. During this period of development of collateral circulation (1-21 days), hemodynamic changes (arrhythmia, bleeding, or hypotension) and alteration of position may be critical. The slight changes in cerebral blood flow which occur on sitting may be enough in these patients with marginal vascular compensation to produce clinical ischemia. Positional ischemia has been limited to patients with basilar or bilateral vertebral occlusion and has not been seen in patients with unilateral vertebral occlusion, presumably because of an adequate contralateral vertebral supply. As thrombosis occurs, embolization distally within the vertebrobasilar system may lead to sudden posterior circulation clinical deficit. Embolization distally is the presumed mechanism of presentation in our group 2 patients with sudden onset deficits, and in patient 5 with a sudden deficit following TIAs in whom nonfilling of the left superior cerebellar artery (a clinically affected vascular territory) favored an embolic mechanism. Embolization in the posterior circulation has been documented on postmortem examination by others. Meyer et al., and Sundt, Whisnant et al. have also emphasized the role of emboli arising from proximal vertebrobasilar occlusion. After a period of several weeks, a stable collateral circulation develops (sometimes after death of cerebral tissue, i.e., stroke) which is generally resistant to further hemodynamic crises. In addition, stabilization of the clot (days to weeks) is usually associated with a cessation of late embolization arising from the region of thrombosis.

In some patients, e.g., group 3 (patient 10) thrombosis may produce a progressive clinical course over days without preceding TIAs. In our experience, in patients with progressing stroke due to lacunar infarction or internal carotid artery occlusion, the prognosis is poorer than for those patients with TIAs or fluctuating course. The recovery phase of a TIA or fluctuating thrombotic stroke may be due, at least partially, to the presence of adequate collateral circulation. Progressing stroke may indicate poor collateral potential, and so a worse prognosis. The single patient in group 3 in this report had the most severe deficit of any of our 10 patients.

Jones, Millikan and Sandok described the temporal profile of 37 patients with a clinical diagnosis of vertebrobasilar system infarction. None of the patients had angiographic definition of the underlying vascular pathology, but all 10 patients who died had occlusion of a vertebral or basilar artery at postmortem examination. Twenty-two of their 37 patients had reached maximum deficit within 24 hours, and some other patients progressed over a period of 4 days. No patient had progression of signs after 1 week, and there were no known late exacerbations.

Since little data exist concerning the clinical tempo of angiographically verified vertebrobasilar occlusion, it will be useful to compare the clinical course of our patients with that of patients with known occlusion of the internal carotid artery (a vessel of comparable size and length). Fourteen patients, personally examined, with angiographically documented occlusion of the internal carotid artery were followed an average of 3 years. Eight had TIAs, 2 sudden onset deficits, and 4 gradually progressive onset over days. Fluctuations of clinical deficit occurred during the first 2 weeks but the patients remained stable thereafter irrespective of treatment (8 untreated). Two of these patients had late (greater than 1 year post-stroke) episodes of amaurosis fugax; each, in addition to the carotid occlusion, had severe stenosis of the ipsilateral external carotid artery and 1 had, in addition, stenosis of the ophthalmic artery. No patient developed a late permanent or transient central nervous system deficit on the side of prior carotid occlusion. Barnett and Aldis specifically studied the question of delayed cerebral ischemia distal to occlusion of a major cerebral artery. Of 426 patients, there were only 12 occurrences of subsequent events, 4 within the first 2 weeks (1.9% late occurrence). Of the late occurrences 3 were amaurosis fugax alone, 6 were cerebral alone and 3 were amaurosis fugax and cerebral. One patient had common carotid stenosis ipsilateral to an internal carotid artery occlusion but episodes ceased after complete common carotid artery occlusion. In 3 patients documented hemodynamic changes occurred (2 postural hypotension and 1 ventricular arrhythmia). Barnett subsequently reported that 25 of 235 patients
enrolled in a cooperative study of antiplatelet aggregation drugs had cerebral ischemia subsequent to known occlusion. One of these patients had a basilar artery occlusion, and another a bilateral vertebral artery occlusion in the neck. Barnett and Aldis and Barnett expressed the belief that late episodes represented embolic material breaking off from the top of the prior occlusion, or, more commonly, embolization from diseased external or common carotid collateral supply. In other series of patients with carotid occlusion reported in the literature late episodes occasionally occurred but insufficient details were given regarding the nature and locus of the late ischemia.

The patient population reported herein represents a selected group of patients with vertebrobasilar disease — all were angiographically studied with documented occlusion of the vertebral or basilar arteries and all survived long enough for follow up. During the same period, 5 patients with angiographically documented disease died, and 10 patients with severe brainstem infarction died without angiography. In addition, our patients' course does not represent the natural course of illness since 8 patients have received temporary or long term anticoagulation. Nevertheless, 2 conclusions seem warranted from our clinical material:

1) Some patients with occlusion of the basilar artery survive with little or no deficit.

2) In occlusive disease of the posterior circulation, just as is true in patients with occlusion of the internal carotid artery, the critical period for acquisition of a central nervous system deficit is at the time of occlusion. The degree of deficit depends primarily on the development of adequate collateral circulation and the presence of distal embolization at the time of occlusion.

Our data suggest that in the group of patients with documented occlusion of the basilar artery a period of bedrest in the supine position, with maintenance of systemic blood pressure, is important. Occlusion of a vertebral artery, in the presence of a patent contralateral vertebral artery, usually is associated with a nonprogressive clinical course unless distal embolization occurs in the weeks after occlusion. Short term (weeks or a few months) anticoagulation may be hypothetically useful in preventing embolization or acute extension of the clot. A clinical trial of conservative medical therapy with short term anticoagulation or agents which decrease platelet agglutination (e.g., aspirin, dipyridamole, or sulfinpyrazone) seems worthy of consideration as opposed to the present practice in many centers of long term warfarin therapy. Intracranial bypass grafts (e.g., occipital to PICA shunts) might be reserved for those unusual patients with documented extensive stenosis of major vertebral or basilar vessels with repeated episodes of ischemia unresponsive to conservative treatment.

References

Occlusion of the vertebral or basilar artery. Follow up analysis of some patients with benign outcome.
L R Caplan

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