Eur Arch Psychiatr Neurol Sci (1988) 237:91–100

European Archives of Psychiatry and Neurological Sciences



# Basilar Artery Disease – Clinical Outcome and Doppler Sonographical Follow-Up*

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**Summary.** In the past 5 years we have investigated 29 patients with symptomatic basilar artery stenoses (14 cases) and occlusions (14) and a patent primitive trigeminal artery with thin-calibered basilar and vertebral arteries (1) using directional continuous-wave Doppler sonography of the vertebral arteries. A total of 19 patients survived, and 17 of them were clinically and sonographically reexamined after $40.4 \pm 15.8$ months (mean $\pm$ SD). Among the 8 patients with basilar stenoses, 6 – with no further transient ischemic attacks (TIAs) in the interval – exhibited an increase in the summed modified Pourcelot indices (relative end-diastolic flow velocities) of the vertebrals by $0.18 \pm 0.16$; the other 2 showed a decrease by 0.26 each, in 1 case temporally related to a TIA, in the 2nd case without further clinical deterioration. In the 8 survivors with basilar occlusions, 5 remained – by sonographic criteria – unchanged with summed modified Pourcelot indices of the vertebrals of 0.00, while 3 patients exhibited a slight increase in the summed modified Pourcelot indices of $0.13 \pm 0.03$. While the difference between the outcome of subsets of patients treated with regimens of 30,000–40,000 units heparin/day or phenprocoumon and less radical drugs were statistically not significant, the former regimen appeared clinically more efficacious in preventing further deterioration in approximately two-thirds of the patients affected. Due to the potential recurrence of neurological symptoms, a treatment period with phenprocoumon of 6 months after discharge from hospital appears justified. Due to these therapeutic efforts, approximately half of the patients initially affected survived with no or only a mild neurological deficit. Directional continuous-wave Doppler sonography is, in our opinion, a reliable technique for examining the short- and long-term changes in peripheral vascular resistance.

**Key words:** Basilar artery stenosis – Basilar artery occlusion – Continuous-wave Doppler sonography

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* Herrn Prof. Dr. H. Gänshirt anläßlich seiner Emeritierung in Dankbarkeit gewidmet

## Introduction

While complete occlusion of the basilar artery has for many years been generally considered as fatal or productive of severe neurological deficit, careful investigations by Fields et al. [5] and Caplan [3, 4] demonstrated that more patients than previously presumed survived this event without permanent neurological deficit. In the present study, we report on the clinical outcome of patients with basilar artery disease during the initial stay in the hospital depending on the therapeutic regimen administered and on the severity of the acquired deficit. Moreover, we examined the surviving patients by clinical and Doppler sonographic criteria after an average of 40 months.

## Patients and Methods

The present study refers to 29 patients investigated in our hospital between 1980 and 1984 [1] with the angiographical (bilateral retrograde brachial angiography) or angiographical and sonographical (directional continuous-wave Doppler sonography) diagnoses of a symptomatic basilar artery stenosis (14 cases), a basilar artery occlusion (14 cases), and a patent primitive trigeminal artery with thin-calibered basilar and vertebral arteries (1 case). All patients requiring assisted ventilation upon admission to hospital had to be referred to the department of internal medicine. Thus, an unknown figure of basilar

**Table 1.** Clinical outcome of patients with basilar artery stenoses regarding the severity of clinical symptoms upon admission and the therapeutic regimen administered. Unless stated otherwise, the deficit observed was attributable to the posterior circulation, and sonography and angiography revealed normal findings in the carotid circulation. In the event of a severe clinical deterioration, CT scan was reperformed to exclude cerebral hemorrhage. ASA = acetylsalicylic acid

| | Age (in years) upon admission and sex | Degree of basilar artery stenosis and additional angiographic/ sonographic findings | Clinical diagnosis upon admission | Therapeutic regimen | Clinical outcome upon discharge |
|---|---|---|---|---|---|
| 1. | 54, M | 50%–59% | Infarct with slight deficit | Heparin 7,500 units s.c. t.i.d., dextrans 500 ml/day; after 10 days, phenprocoumon | No deficit |
| 2. | 48, M | | Transient ischemic attack (TIA) | Phenprocoumon | No further TIAs |
| 3. | 59, M | 80% asymptomatic right internal carotid stenosis at origin | TIA and infarct with slight deficit | Phenprocoumon | Slight deficit, no further TIAs |
| 4. | 61, M | 60%–69% | Infarct with slight deficit | Phenprocoumon | Slight deficit |
| 5. | 71, F | | TIA | Dipyridamole 75 mg t.i.d.; phenprocoumon due to further TIAs | No further TIAs with phenprocoumon |
| 6. | 57, M | | TIA | Phenprocoumon | No further TIAs |
| 7. | 62, M | ⩾ 70% 90% right/80% left carotid stenosis at origin | TIA | High-dose heparin; phenprocoumon | No further TIAs; slight motor deficit of right face and arm due to previous infarction in left middle cerebral artery territory |
| 8. | 69, M | | TIA | Phenprocoumon | No further TIAs |
| 9. | 63, M | | Infarct with severe deficit | High-dose heparin; phenprocoumon | Slight deficit, moderate dementia |
| 10. | 47, M | | Infarct with slight deficit; severe dementia | Heparin 5,000 units s.c. t.i.d., pentoxifylline 400 mg t.i.d. | Unchanged (Buerger's disease) |
| 11. | 79, M | 80% asymptomatic stenosis of right internal carotid siphon proximal to ophthalmic artery | Infarct with slight to moderate deficit | Heparin 5,000 units s.c., dipyridamole 75 mg, and ASA 330 mg t.i.d. | Slight deficit |
| 12. | 63, F | | Infarct with severe deficit | Heparin 5,000 units s.c., dipyridamole 75 mg, and ASA 330 mg t.i.d.; phen-prcoumon due to further deterioration | Further deterioration and TIAs; almost complete recovery with phenprocoumon |

| | | | | | |
|---|---|---|---|---|---|
| 13. | 57, M | Occlusion of left internal carotid upon admission; upon deterioration, bilateral internal carotid occlusion | TIA and infarct with slight deficit | High-dose heparin | After 2 weeks, clinical deterioration; died of ischemic infarcts of left hemisphere, right middle/anterior cerebral artery territories, and brainstem |
| 14. | 76, F | | Infarct with slight deficit | Dextrans 500 ml/day, ASA 500 mg t.i.d. | Slight deficit; discharged with ASA 500 mg t.i.d.; after 10 days, severe bilateral motor deficits; died of ischemic brainstem infarct; no supratentorial lesions by CT scan |

artery thromboses with an initially severe neurological deficit requiring assisted ventilation were excluded from this study.

We report retrospectively on the clinical outcome of the patients upon discharge from the hospital with respect to the therapeutic regimen administered, and longitudinally on the course of the disease in the same patients by clinical and sonographical criteria after $40.4 \pm 15.8$ months (mean $\pm$ SD). The sonographical criteria in the evaluation of the carotid and vertebral arteries developed by Büdingen et al. [2, 9] were applied as previously described [1].

The therapeutic regimen of the initial study consisted of 5,000 units heparin i.v., followed by a continuous i.v. infusion of 30,000–40,000 units heparin/day for 10 days. The subsequent dosage of heparin was adjusted according to the activated partial thromboplastin time at twice normal (range 1.5 to 2.5). Some patients with the clinical diagnoses of transient ischemic attack (TIA) and ischemic stroke with mild neurological deficit were initially treated with phenprocoumon (Marcumar). The treatment with heparin was followed by oral administration of phenprocoumon for approximately 6 months unless one of the following contraindications argued against such a regimen: history of stomach or duodenal ulcer or bleeding disorder, hypertension not adequately treatable, or dementia with resultant unreliability.

High doses of heparin were initiated on the day of admission if Doppler sonography suggested a severely compromised basilar artery flow as described above, and after computerized tomography (CT) scan had excluded a cerebral hemorrhage. The CT scan was repeated in the event of a drastic clinical deterioration to exclude cerebral hemorrhage. In the cases with a residual end-diastolic flow component in at least one vertebral artery by Doppler sonography, and on weekends with no sonographically experienced physician on call (cases 2,6, and 12 of Table 1 and case 11 of Table 2), the corresponding high-dose heparin therapy was delayed up to 66 h until angiography revealed basilar artery disease.

Of the 29 patients, 9 were in due course treated with i.v. dextrans (40,000 average molecular weight, Rheomacrodex), low-dose heparin (5000 units s.c. t.i.d.), acetylsalicylic acid p.o., dipyridamole p.o., pentoxifylline p.o., or combinations thereof – for reasons not unequivocally evident retrospectively. The other 20 patients received the standard therapy with heparin and/or phenprocoumon referred to previously.

In order to standardize the results concerning changes in the neurological picture, we divided the observed neurological deficits into three categories of severity without revealing the details: slight (no significant reduction in mobility), moderate (evident reduction in mobility without dependence on others in daily life), and severe (needing care).

## Results

### *Acute Course of the Disease*

In Tables 1 (basilar artery stenoses) and 2 (basilar artery occlusions), the results regarding the clinical symptoms, the therapeutic regimens administered, and the clinical outcomes upon dismissal from the hospital are listed according to the sequence of cases in the original publication concerning the Doppler sonographic diagnosis of basilar artery disease [1].

Table 3a refers to the cases with basilar artery stenoses treated with the standard therapeutic regi-

**Table 2.** Clinical outcome of patients with bilateral distal vertebral and basilar artery occlusions regarding the severity of clinical symptoms upon admission and the therapeutic regimen administered. Additional explanations analogous to Table 1

| | Age (in years) upon admission and sex | Localization of basilar artery occlusion and additional angiographic/ sonographic findings | Clinical diagnosis upon admission | Therapeutic regimen | Clinical outcome upon discharge |
|---|---|---|---|---|---|
| 1. | 70, M | Bilateral occlusion of distal vertebrals | Infarct with moderate deficit | High-dose heparin; phenprocoumon | Slight deficit |
| 2. | 64, F | | TIA | Phenprocoumon | No further TIAs |
| 3. | 45, M | Occlusion of proximal third | TIA | High-dose heparin; phenprocoumon | No further TIAs |
| 4. | 63, F | | Infarct with severe deficit | High-dose heparin; phenprocoumon | Moderate deficit |
| 5. | 60, M | | Infarct with moderate deficit | High-dose heparin; phenprocoumon | Almost complete recovery |
| 6. | 59, M | Occlusion of proximal and middle thirds | Infarct with moderate deficit | Dextrans 500 ml/day | Further deterioration; died of ischemic brainstem infarct after 10 days; no supratentorial infarct and hemorrhage detected by CT scan |
| 7. | 77, M | | Infarct with moderate deficit | High-dose heparin; phenprocoumon | Almost complete recovery |
| 8. | 66, F | | Infarct with severe deficit | High-dose heparin; phenprocoumon | Moderate deficit |
| 9. | 51, M | | Infarct with moderate deficit | Heparin 5,000 units s.c. t.i.d., dextrans 500 ml/day | Slight deficit |
| 10. | 35, M | | Infarct with severe deficit | High-dose heparin | Locked-in syndrome |
| 11. | 53, M | Left supraclinoid internal carotid occlusion proximal to ophthalmic artery | Infarct with severe deficit | Heparin 5,000 units s.c. t.i.d., dextrans 500 ml/day | Died of ischemic brainstem infarct after 2 days; additionally supraclinoid internal carotid occlusion without detectable supratentorial lesion by CT scan on 1st day |
| 12. | 76, F | | Infarct with severe deficit | High-dose heparin | Died of ischemic infarct of midbrain (CT scan) after 5 days |
| 13. | 74, F | Occlusion of middle third | Infarct with moderate deficit | Heparin 7,500 units s.c. t.i.d. | Unchanged |
| 14. | 67, M | | TIA | Heparin 5,000 units s.c. t.i.d., dextrans 500 ml/day, phenprocoumon | No further TIAs |

**Table 3a.** Clinical outcome of patients with basilar artery stenoses receiving the therapeutic regimens of 30,000–40,000 units heparin/day (partial thromboplastin time maintained at twice normal) and/or phenprocoumon

| Symptoms upon admission | | | | | | |
|---|---|---|---|---|---|---|
| TIA | | 4 | | | | |
| Infarct with | | | | | | |
| slight | | 1 | 2 | | | 1 |
| moderate | | | | | | |
| severe | | 1 | 1 | | | |
| neurological deficit | | | | | | |
| | TIA | no | slight | moderate | severe | lethal outcome |
| | | | Neurological deficit | | | |
| | | | | | | Clinical outcome upon discharge |

**Table 3b.** Clinical outcome of patients with basilar artery stenoses receiving therapeutic regimens such as low-dose heparin, low molecular weight dextrans, acetylsalicylic acid, dipyridamole, pentoxifylline, and combinations thereof

| Symptoms upon admission | | | | | | |
|---|---|---|---|---|---|---|
| TIA | 1 | | | | | |
| Infarct with | | | | | | |
| slight | | | 2 | | | 1 |
| moderate | | | | | | |
| severe | | | | | | |
| neurological deficit | | | | | | |
| | TIA | no | slight | moderate | severe | lethal outcome |
| | | | Neurological deficit | | | |
| | | | | | | Clinical outcome upon discharge |

men, and Table 3b to the cases receiving divergent therapies (low molecular weight dextrans, low-dose heparin, acetylsalicylic acid, etc.). Tables 4a and 4b demonstrate the corresponding figures for basilar artery occlusions.

Of the 14 patients with basilar artery stenoses, 5 clinically exhibited TIAs, which recurred in 1 case who was receiving dipyridamole (75 mg t.i.d.). In this group, 7 patients demonstrated a slight neurological deficit upon admission to hospital that remained unchanged until discharge in 4 cases and subsided in another case. Of these 7 patients, 2 showed further clinical deterioration and died despite the therapeutic efforts, 1 in the group receiving standard therapy, and 1 receiving low molecular weight dextrans (500 ml/day) and acetylsalicylic acid (500 mg t.i.d.). The 2 cases with an initially severe neurological deficit partially improved up to the time of hospital discharge: in 1 case a slight, and in the 2nd case, a moderate neurological deficit persisted. The case with a symptomatic patent primitive trigeminal artery revealed a slight neurological deficit upon admission that further improved up to the time of discharge.

Ten of the 14 cases with basilar artery stenoses and the case with a patent primitive trigeminal artery exhibited normal findings in the internal carotid arteries by Doppler sonography and right retrograde brachial angiography – the left internal carotid artery had only been judged sonographically in these cases. Two patients (cases 3 and 11 in Table 1) each exhibited an 80% stenosis (reduction in lumen diameter) of one internal carotid artery, one located at its origin and one in the carotid siphon proximal to the origin of the ophthalmic artery. Both stenoses were detected by Doppler sonography, and were asymptomatic by CT scan parameters. Case 7 of Table 1 had bilateral tight stenosis of the internal carotid arteries with a slight motor deficit known for years while the acute symptoms were unequivocally attributable to the posterior circulation. Only in case 13 of Table 1 was the lethal outcome related to a bilateral internal carotid occlusion that developed in the course of the disease.

The 2 cases with bilateral distal vertebral artery occlusions and the 12 cases with basilar artery occlusions corresponded clinically upon admission to TIAs

**Table 4a.** Clinical outcome of patients with bilateral distal vertebral and basilar artery occlusions receiving the therapeutic regimens of 30,000–40,000 units heparin/day and/or phenprocoumon

| Symptoms upon admission | | | | | |
|---|---|---|---|---|---|
| TIA | 2 | | | | |
| Infarct with slight | | | | | |
| moderate | | 3 | | | |
| severe | | | 2 | | 2 |
| neurological deficit | | | | | |
| | no | slight | moderate | severe | locked-in syndrome, lethal outcome |
| | | Neurological deficit | | | |
| | | | | | Clinical outcome upon discharge |

**Table 4b.** Clinical outcome of patients with bilateral distal vertebral and basilar artery occlusions receiving therapeutic regimens analogous to those described in Table 3b

| Symptoms upon admission | | | | | |
|---|---|---|---|---|---|
| TIA | 1 | | | | |
| Infarct with slight | | | | | |
| moderate | | 1 | 1 | | 1 |
| severe | | | | | 1 |
| neurological deficit | | | | | |
| | no | slight | moderate | severe | lethal outcome |
| | | Neurological deficit | | | |
| | | | | | Clinical outcome upon discharge |

in 3 cases, and to a moderate neurological deficit in 6 cases – with partial recovery to a slight deficit in 4 cases, of persistent character in 1 case, and with lethal outcome in another case. The 5 patients with a severe neurological deficit upon admission recovered partially under the standard therapeutic regimen to a moderate deficit in 2 cases; 2 patients died, and 1 deteriorated to a locked-in syndrome. Among these 14 patients with bilateral distal vertebral and basilar artery occlusions, 13 exhibited normal Doppler sonographic and angiographic findings in the internal carotid arteries; 3 of these patients underwent percutaneous left carotid angiography to visualize the left posterior communicating artery without demonstrating pathological findings. Only case 11 of Table 2 showed a left supraclinoid internal carotid occlusion that was also detected by Doppler sonography. He died 2 days after admission to hospital due to ischemic infarctions in the brainstem and the left middle cerebral artery territory as demonstrated at autopsy. Two further patients (cases 6 and 12 of Table 2) died of ischemic brainstem infarctions within the initial treatment period after 10 and 5 days, respectively.

Due to the low figure of patients in the group receiving low molecular weight dextrans, low-dose heparin, etc., there was no statistically significant difference detectable compared to the cases receiving high-dose anticoagulant therapy regarding the clinical outcome ($\chi^2$ test, $P > 0.05$). However, if one combines all cases with moderate and severe deficits upon discharge, and all deaths under the initial therapeutic regimen administered, the outcome of the group not receiving high-dose anticoagulant therapy appeared unfavorable: 4 of the 9 cases of this group fell into this category, while the corresponding figure in the group receiving high-dose anticoagulant therapy amounted to only 6 out of 20 cases. It is notable that the patients with moderate and severe neurological deficits upon admission were equally distributed in both therapeutic categories: 4 out of 9, and 9 out of 20 cases, respectively.

**Table 5.** Long-term clinical and sonographic results obtained from patients with basilar artery stenoses

| | Time interval (months) | Therapeutic regimen during interval | Modified Pourcelot index right/left vertebral artery (upon reexamination) | Change of summed modified Pourcelot indices |
|---|---|---|---|---|
| 1. | 49 | Phenprocoumon for 1 week; since then pentoxifylline 400 mg b.i.d. | 0.00<br>0.37 | +0.05 |
| 2. | 55 | Phenprocoumon for 1 year; since then dipyridamole 75 mg and ASA 330 mg b.i.d. | 0.00<br>0.23 | −0.26 |
| 3. | Patient could not be contacted | | | |
| 4. | 52 | Phenprocoumon of unknown duration | Patient died of ischemic stroke due to basilar artery thrombosis as proved by autopsy | |
| 5. | 35 | Dipyridamole 75 mg t.i.d.for 2 weeks; since then phenprocoumon | 0.33<br>0.34 | +0.47 |
| 6. | 32 | Phenprocoumon for 3 months; since then dipyridamole 75 mg and ASA 330 mg t.i.d. | 0.00<br>0.24 | +0.08 |
| 7. | 44 | Phenprocoumon for 6 months; since then no medication. Bilateral carotid surgery | 0.00<br>0.31 | +0.18 |
| 8. | 45 | Phenprocoumon of unknown duration | Patient died of myocardial infarct | |
| 9. | 15 | Phenprocoumon for 7 months; TIA after discontinuation; since then phenprocoumon | 0.00<br>0.00 | −0.26 |
| 10. | 6 | Pentoxifylline 400 mg t.i.d. | Patient died of Buerger's disease | |
| 11. | 54 | Dipyridamole 75 mg and ASA 330 mg t.i.d. for 1 year; since then no medication | 0.16<br>0.00 | +0.08 |
| 12. | 61 | Phenprocoumon 6 months; since then dipyridamole 75 mg and ASA 330 mg t.i.d. | 0.33<br>0.00 | +0.21 |
| 13. | Patient died in hospital | | | |
| 14. | 0.75 | ASA 500 mg b.i.d. | Patient died of basilar artery thrombosis in hospital after readmission | |

In 4 cases, the diagnostic procedure and hence the institution of adequate therapy was delayed due to a weekend situation without a sonographically experienced physician on call. In 2 of these patients (cases 2 and 6 of Table 1), the delay was not related to a worsening, while in cases 12 of Table 1 and 11 of Table 2, the clinical situation further deteriorated, in the latter case with fatal outcome.

*Long-Term Follow-up*

In Tables 5 und 6, the results of the long-term follow-up of the patients with basilar artery stenoses (Table 5) and basilar artery occlusions (Table 6) are listed according to the sequence of Tables 1 and 2.

Of the original 14 patients with basilar artery stenoses – 13 on the basis of atherosclerosis, 1 due to an angitis (Buerger's disease) – 8 were reexamined by clinical and Doppler sonographic criteria after $43 \pm 15.1$ months. Five patients had died in the meantime, 2 of them soon after the initial deficit acquisition, and 1 due to a further basilar artery thrombosis as proved at autopsy. Of the 5 deaths, 4 were related to the initial pathological deficit, and only 1 patient died of a myocardial infarction. One patient could not be contacted.

In 6 of the 8 patients reexamined, we found an increase in the summed modified Pourcelot indices of the vertebral arteries by $0.18 \pm 0.16$ (range 0.05–0.47) by Doppler sonography. According to the histories of these patients, no further TIAs occurred in the elapsed interval, nor did we find any neurological deficit beyond those observed at the time of discharge from hospital.

In the remaining 2 cases with basilar artery stenoses, we found a decrease in the summed modified Pourcelot indices of the vertebrals of 0.26 each at the time of the follow-up examination, in 1 case (case 2 of Table 5) without a corresponding neurological deficit. In this latter case, Doppler sonography –

**Table 6.** Long-term clinical and sonographic results obtained from patients with bilateral distal vertebral and basilar artery occlusions

| | Time interval (months) | Therapeutic regimen during interval | Modified Pourcelot index right/left vertebral artery (upon reexamination) | Change of summed modified Pourcelot indices |
|---|---|---|---|---|
| 1. | 76 | Phenprocoumon for 4 months; since then ASA 500 mg b.i.d. | 0.00<br>0.00 | ±0.00 |
| 2. | 35 | Phenprocoumon discontinued; ASA 500 mg and pentoxifylline 400 mg b.i.d. | 0.00<br>0.00 | ±0.00 |
| 3. | 30 | Phenprocoumon for 6 months; TIA after discontinuation, phenprocoumon for another 6 months; since then ASA 330 mg and dipyridamole 75 mg t.i.d. | 0.00<br>0.30 | +0.17 |
| 4. | 16 | Phenprocoumon for 3 months; since then ASA 330 mg and dipyridamole 75 mg b.i.d. | 0.00<br>0.11 | +0.11 |
| 5. | 36 | Phenprocoumon for 9 months; since then ASA 500 mg/day | 0.00<br>0.00 | ±0.00 |
| 6. | Patient died in hospital | | | |
| 7. | 33 | Phenprocoumon for 3 months; since then no medication | 0.00<br>not detectable | ±0.00 |
| 8. | 41 | Phenprocoumon for 3 years; since then ASA 500 mg b.i.d. | 0.00<br>0.00 | ±0.00 |
| 9. | 71 | ASA 500 mg/day | Patient living in USA, not reexamined by Doppler sonography | |
| 10. | 3 | No medication | Patient died due to complications of locked-in syndrome | |
| 11. | Patient died in hospital | | | |
| 12. | Patient died in hospital | | | |
| 13. | 1 | Heparin 7,500 units s.c. t.i.d.; pentoxifylline 300 mg i.v. t.i.d. | Patient died, probably of further stroke, in a rehabilitation center (no CT scan performed) | |
| 14. | 35 | Phenprocoumon for 3 months; since then ASA 330 mg and dipyridamole 75 mg/day | 0.16<br>0.21 | +0.12 |

with a unilateral loss of end-diastolic flow velocity in one vertebral artery – indicated severely compromised intracranial flow of the corresponding vertebral artery (possibly an intracranial occlusion) ensuing in the interval without a clinically comprehensible deficit. The 2nd patient (case 9 of Table 5) suffered from a further TIA after discontinuation of 7 months treatment with phenprocoumon. This event required a second admission to hospital; at that time, Doppler sonography suggested a basilar artery occlusion due to the bilateral absence of an end-diastolic flow component in both vertebral arteries. We did not find a neurological deficit at the time of the follow-up examination beyond that at dismissal from hospital, but did find severe dementia.

The case with the patent primitive trigeminal artery revealed no further deterioration according to clinical and sonographical criteria after an interval of 22 months.

Of the 14 patients with a bilateral distal vertebral or a basilar artery occlusion, 9 were still alive at the time of reexamination after 37.8 ± 17.1 months, and 8 of them were reinvestigated. One of them, living in the United States, kindly sent us his neurologist's report (without any deficit) but we could not reexamine him using Doppler sonography. In 1 case (case 3 of Table 6), a further TIA occurred after discontinuation of 6 months phenprocoumon treatment; the other 7 patients had no further TIAs or neurological deterioration upon reexamination.

We found a bilateral absence of an end-diastolic flow component in both vertebrals – indicating the persistence of basilar artery occlusion – in 5 of the 8 patients reinvestigated by Doppler sonography.

Three patients exhibited an increase in the summed modified Pourcelot indices of the vertebrals by $0.13 \pm 0.03$, probably indicating improved collateral pathways, e.g., through rete mirabile anastomoses.

The 5 deaths were closely related to the initial deficit acquisition, and in 3 cases occurred during the initial treatment period in hospital (cases 6, 11, and 12 of Table 2). One death occurred due to complications of the locked-in syndrome that developed early in the course of the disease (case 10 of Table 2). The last patient (case 13 of Table 2) died 1 month after her transfer to a rehabilitation hospital, probably due to a further stroke.

## Discussion

In 1946, Kubik and Adams [6] described 18 patients with brainstem infarction due to occlusion of the basilar artery discovered at postmortem examination and emphasized the abrupt onset and the frequent fatal outcome. Marshall [7] subsequently pointed out that many untreated patients with the clinical symptomatology of posterior circulation vascular disease do not develop serious deficits; however, the underlying vascular pathology in this group of clinical patients was unknown. Angiographically controlled studies by Meyer et al. [8], Fields et al. [5], and Caplan [3, 4] on the clinical outcome of selected patients with basilar stenoses and occlusions provided evidence that more patients than previously presumed survive this event without permanent deficit. The present publication addresses the short- and long-term outcome (40 months on average) of all patients with angiographically proven diagnoses of basilar artery and/or bilateral distal vertebral artery disease treated in our institution over a 5-year period. All patients requiring assisted ventilation upon primary admission were excluded, a fact that might in part explain the favorable outcome of the patients reported.

Our conclusions:

1. In comparison to other conservative regimens (with low molecular weight dextrans, low-dose heparin, acetylsalicylic acid, dipyridamole, pentoxifylline, and combinations thereof), the therapeutic regimen of 30,000–40,000 units heparin/day – while not statistically significant – tended to lessen the severity of the residual neurological deficit and the number of lethal outcomes in acutely symptomatic patients with basilar artery disease. Phenprocoumon appeared to be equally adequate in patients with TIAs due to basilar artery disease. A regimen using local fibrinolysis with streptokinase [10] has not been administered in our institution.

2. All but two deaths observed during the follow-up period – approximately one-third of all patients affected – occurred with a close temporal and causal relationship to the initiation of deficit acquisition. Only in one case was death causally related to concomitant carotid artery disease.

3. Approximately half of the patients affected survived with no or only a mild neurological deficit.

4. A period of 6 months for the treatment with phenprocoumon appears justified to prevent further clinical deterioration; in the event of the reappearance of TIAs after phenprocoumon discontinuation, longer treatment with phenprocoumon may be necessary. Under this regimen, the probability of suffering from further TIAs or strokes was fairly low when the first few months had been survived. Nevertheless, in a few cases of basilar artery disease, less radical – and thereby less dangerous – regimens proved to be equally effective, possibly representing the natural course of the disease in these cases.

5. Further progression of atheromatous disease, as suggested in two cases by Doppler sonography, did not necessarily induce further neurological deterioration.

6. As judged by Doppler sonography, peripheral vascular resistance decreased in the long-term, probably due to a decrease in the stenotic lesion in 75% of the patients examined with basilar artery stenoses, and due to the probable development of collateral pathways in almost 40% of the patients with basilar artery occlusions.

7. Once the diagnosis of basilar artery disease has been confirmed by angiography, Doppler sonography constitutes a reliable technique for the noninvasive follow-up by semiquantitatively estimating the peripheral vascular resistance. Even cases of basilar artery disease below a 60% reduction in lumen diameter that cannot be detected by Doppler sonography [1] can be followed for a possible increase in the peripheral resistance by calculating the modified Pourcelot indices of both vertebral arteries.

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Received January 31, 1987