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Collateral Circulation and Outcome after Basilar Artery Thrombolysis

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BACKGROUND AND PURPOSE: **This study was undertaken to examine the relationship between collateral flow and outcome after local intraarterial thrombolytic treatment for basilar artery thrombosis.**

METHODS: **Twenty-four patients with symptomatic basilar thrombosis were treated with intraarterial urokinase. Angiograms at the time of treatment were analyzed to characterize collateral flow. The number of posterior communicating arteries (PCoAs) and the degree of collateral filling of the basilar artery were then compared with symptom duration before treatment, with Glasgow Coma Scale (GCS) score at the time of treatment, with 90-day modified Rankin score, and with 90-day survival status.**

RESULTS: **Of the 20 patients who had carotid artery injections at the time of the thrombolytic procedure, two had no PCoA, eight had one PCoA, and 10 had two PCoAs. Nine had no collateral opacification of the basilar artery, six had collateral opacification of the distal basilar artery, and five had collateral opacification of the distal and proximal basilar artery. Ninety-day survival was 38%; 25% of patients had good neurologic outcomes. No correlation was found between the number of PCoAs and symptom duration, pretreatment GCS score, survival, or neurologic outcome. Duration of symptoms before treatment was longer in patients with collateral flow to the basilar artery. Basilar artery collateral flow did not correlate with survival, but it did correlate with neurologic outcome for the 12 patients with middle or distal basilar artery thrombus in whom collateral flow to the basilar artery was assessed (83% with collateral flow had good neurologic outcomes, but only 17% without collateral flow had good outcomes). All six patients with proximal basilar artery thrombus in whom collateral flow was assessed died, independent of the collateral flow observed.**

CONCLUSION: **In symptomatic acute basilar artery thrombosis, neurologic outcome was better after intraarterial thrombolysis in patients who had collateral filling of the basilar artery, except in cases of proximal basilar thrombosis. Patients with collateral filling of the basilar artery also tolerated longer symptom duration.**

Basilar artery thrombosis, most often fatal with conservative therapy (1–9), has been treated in recent years with intraarterial thrombolytic agents in an attempt to improve survival. Nonrandomized series (10–16) have suggested that survival is improved by basilar artery recanalization. In a previous study (15), we found that survival was higher in patients with thrombus confined to the distal basilar artery, but we found no other reliable predictors of outcome. Studies of patients treated for giant basilar artery aneurysms have shown that neurologic outcome was related to the number and size of the posterior communicating arteries (PCoAs) after ligation of the basilar artery (17, 18). We did not have sufficient data to comment upon the effect of collateral circulation in our prior report on basilar thrombolysis, but we have accumulated additional cases since that study. Given that outcome and collateral circulation are probably influenced by many factors, the present study was undertaken to determine the relationship between collateral circulation and outcome after intraarterial thrombolysis for basilar artery thrombosis.

Methods

Twenty-four of 26 patients who presented with symptomatic, angiographically confirmed acute basilar artery thrombosis were treated with local intraarterial urokinase. Two patients

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were excluded from treatment, one in whom a craniotomy had been performed within the previous 48 hours and one who was in a coma, with extensive brain stem infarction evident on brain CT scans. The method for local intraarterial thrombolysis using urokinase was described in a previous report (15). Bilateral common carotid angiography was usually performed in conjunction with the thrombolytic procedure.

Angiograms at the time of treatment were reviewed for 19 of the 24 patients whose films were still available. In the five patients for whom films were unavailable (cases 1, 8, 12, 17, and 18), the original angiographic reports and angiographic data from the prior basilar thrombolytic study were reviewed. Basilar artery clot locations were determined: distal basilar clots were defined as those that obstructed the basilar tip, the posterior cerebral origins, or the superior cerebellar origins but did not extend inferiorly to the level of the anterior inferior cerebellar artery; midbasilar clots were defined as those that obstructed the basilar artery at or about the level of the anterior inferior cerebellar arteries; and proximal clots were defined as those at or below the vertebrobasilar junction. This definition did not necessarily reflect the length of clot but rather defined location by the clot's most proximal or inferior extent. For each case in which the carotid arteries were injected during the basilar thrombolytic procedure, the time of the carotid artery injections was determined (before or after urokinase infusion in the basilar artery), as were the number and size of the PCoAs and the extent of basilar artery filling by collateral vessels. As the studies were digitally imaged in varying degrees of magnification, PCoAs were defined as large if they were approximately the size of the posterior cerebral artery, as medium if they were approximately half the size of the posterior cerebral artery, and as small if they were approximately one fourth the size of the posterior cerebral artery or smaller. Collateral flow that opacified the basilar artery from the tip inferiorly to the level of the superior cerebellar arteries was termed distal, while collateral flow that opacified the basilar artery from the tip to below the level of the anterior inferior cerebellar arteries was termed distal and proximal.

A review of patients' records and telephone interviews was approved by the human studies committee. Each patient's chart was reviewed to determine the duration of symptoms before treatment (hours from symptom onset to initiation of urokinase delivery), the neurologic status at the time of treatment (Glasgow Coma Scale [GCS] score), and the neurologic status at the time of discharge or transfer. Telephone interviews or outpatient chart information were used to determine each patient's status at 90 days (modified Rankin score 0–6, where 0 = no symptoms, 1 = no significant disability, and 6 = dead).

The number of PCoAs and degree of basilar artery filling by collateral flow were correlated with clot location in the basilar artery, symptom duration prior to treatment, GCS score at the time of treatment, 90-day survival, and 90-day modified Rankin score. When tabulating results related to collateral flow, we considered only those patients who had carotid injections before basilar artery recanalization or those who had carotid injections after basilar thrombolysis without recanalization or those without PCoAs or P1 segments, so the collateral flow described was accurate for the time the basilar artery was obstructed. Tables were constructed for χ^2 statistical analysis using the Fisher exact test.

Results

Twenty patients had carotid artery injections in conjunction with basilar artery thrombolytic procedures and four did not. All 20 who had the carotid injections were included in the PCoA analysis. Of these 20 patients, 14 had the carotid artery injections before intraarterial urokinase infusion, and they were included in the analysis of collateral flow. The remaining six patients had the injections after termination of urokinase infusion; of these, two (cases 9 and 13) had no recanalization after thrombolysis and were included in the analysis of collateral flow and four (cases 4, 6, 15, and 22) had complete recanalization of the basilar artery after thrombolysis. Of the four with complete recanalization, one (case 15) had persistent fetal posterior cerebral arteries with no connection from either PCoA to the basilar artery, and one (case 4) had no PCoA; those two were also included in the analysis of collateral flow.

The table lists the collected data for all cases. In 10 patients (cases 3, 5, 9, 10, 13, 17, 19, 21, 23, and 24), the basilar thrombus formed on atherosclerotic plaque within the basilar artery or propagated directly from distal vertebral artery plaque or dissection. In the remaining 14 patients, the basilar clot was probably embolic, either from vertebral artery disease or from a more remote source. In five such instances (cases 1, 4, 6, 20, and 22), the basilar embolus occurred after cardiac catheterization procedures.

Of the 20 patients with carotid injections, two had no PCoA, eight had one PCoA, and 10 had two PCoAs. PCoA size could not be characterized for two patients whose films were missing or destroyed, but the number of PCoAs was known for all patients from descriptions in reports and data forms. Seven patients had no collateral opacification of the basilar artery (Figs 1 and 2), six had collateral opacification of the distal basilar artery (Fig 3), and five had collateral opacification of the distal and proximal basilar artery, with the proximal portion filling in a retrograde manner from the distal portion in all such cases (Figs 4 and 5). All patients with collateral opacification of the distal and proximal basilar artery had proximal basilar artery thrombosis.

While collateral flow from the posterior inferior cerebellar artery to the superior cerebellar artery was observed and pial collateral filling of the posterior cerebral arteries was noted, in no case were these collaterals alone sufficient to opacify the basilar artery. In one patient (case 10), there was combined filling of the basilar tip from a small PCoA and from pial retrograde filling of a posterior cerebral artery. In all other patients, basilar opacification by collateral flow was only through circle of Willis connections.

No significant correlation was found between the duration of symptoms before referral for thrombolysis and the number of PCoAs. Of the patients who were symptomatic for less than 10 hours, one had no PCoA, three had one PCoA, and two had two PCoAs. Of the patients who were symptomatic for 10 hours or longer, one had no PCoA, five had one PCoA, and eight had two PCoAs. Although data on PCoA size were not available for all patients, it did not appear to be a critical factor limiting survival. Three of the nine survivors had only small PCoAs, while five of the 15 who did not survive had at least one large PCoA.

Longer duration of symptoms before referral for thrombolysis was found in the group of patients with collateral filling of the basilar artery. Of patients with no collateral filling of the basilar artery, four (57%)

Information for 24 patients treated with intraarterial urokinase for basilar artery thrombosis

Patient	Site of Clot in Basilar Artery	Pretreatment GCS Score	Symptom Duration Before Treatment, hr	Carotid Injection	No. and Size of PCoM Arteries	Collateral Basilar Opacification	90-Day Survival; Rankin Score
1	Distal	9	7	Not done	Unknown	Unknown	Dead; 6
2	Proximal	11, T	14	Not done	Unknown	Unknown	Dead; 6
3	Proximal	6, T	10	Pre-UK	0	None	Dead; 6
4	Distal	14	4	Post-UK	0	None	Alive; 3
5	Proximal	7, T	27	Pre-UK	2 (M,S)	Distal and Proximal	Dead; 6
6	Middle	9, T	11	Post-UK	1 (S)	None	Dead; 6
7	Distal	3, T	27	Not done	Unknown	Unknown	Alive; 4
8	Distal	10	22	Pre-UK	2	Distal	Alive; 0
9	Proximal	11	14	Post-UK	1 (M)	Distal and Proximal	Dead; 6
10	Proximal	7	23	Pre-UK	1 (S)	Distal and Proximal	Dead; 6
11	Proximal	11, T	25.5	Pre-UK	2 (M,L)	Distal and Proximal	Dead; 6
12	Distal	7	1	Pre-UK	1 (L)	None	Dead; 6
13	Middle	11, T	8	Post-UK	1 (L)	Distal	Dead; 6
14	Distal	15	79	Pre-UK	1 (S)	Distal	Alive; 2
15	Middle	4	5.5	Post-UK	2 Fetal	None	Dead; 6
16	Middle	6	9.5	Pre-UK	1 (S)	None	Dead; 6
17	Middle	15	72	Pre-UK	2 (S)	None	Alive; 2
18	Distal	6	27	Pre-UK	2	Distal	Alive; 2
19	Middle	5	12	Not done	Unknown	Unknown	Dead; 6
20	Distal	6	24	Pre-UK	1 (S)	None	Alive; 3
21	Middle	9	36	Pre-UK	2 (M;S)	Distal	Alive; 1
22	Distal	6, T	8.3	Post-UK	2 (L;S)	None	Dead; 6
23	Middle	15	82	Pre-UK	2 (M;S)	Distal	Alive; 1
24	Proximal	10, T	24	Pre-UK	2 (L;S)	Distal and Proximal	Dead; 6

Note.—T indicates intubated; L, large; M, medium; S, small; Pre-UK, before urokinase infusion; Post-UK, after urokinase infusion.

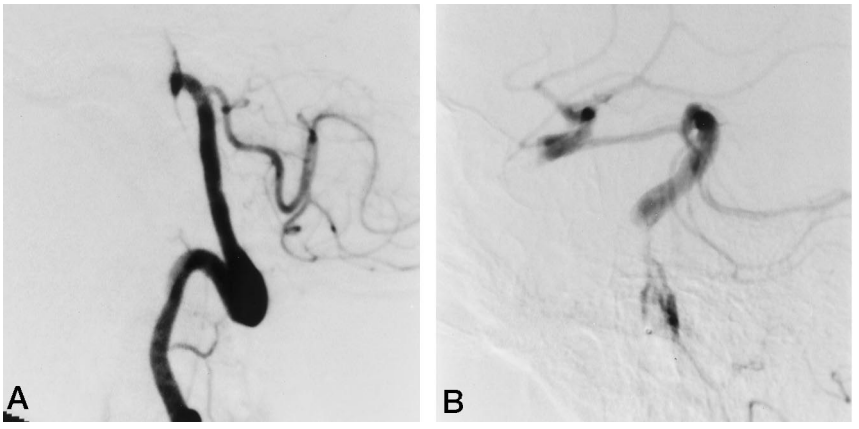

FIG 1. Case 3: proximal basilar thrombosis with no collateral flow to the basilar artery.

A, Initial left vertebral lateral projection shows an occlusion of the lower basilar artery.

B, Lateral basilar artery injection after thrombolysis shows recanalization, mid-basilar stenosis, and filling of the right middle cerebral artery from the basilar artery via the right posterior communicating artery. The right common carotid injection (*not shown*) showed occlusion of the right internal carotid artery. The left common carotid injection (*not shown*) showed no posterior communicating artery and no right A1 segment. The occlusion of the right internal carotid artery, absent right A1 segment, and absent left posterior communicating artery made the right middle cerebral artery dependent on the basilar artery and prevented collateral flow to the basilar artery from the carotid circulation via the circle of Willis. This patient died despite basilar artery recanalization.

were symptomatic for less than 10 hours and three (43%) were symptomatic for 10 hours or longer. Of patients with collateral filling of the basilar artery, one (9%) was symptomatic for less than 10 hours and 10 (91%) were symptomatic for 10 hours or more. This difference between groups was significant ($P = .05$).

No significant correlation was found between patients' neurologic status at the time of treatment and the number of PCoAs seen. Of patients with GCS scores of 3 to 10, one had no PCoA, four had one PCoA, and six had two PCoAs. Of patients with GCS scores of 11 to 15, one had no PCoA, three had one PCoA, and three had two PCoAs.

We found only a slight tendency for patients with collateral filling of the basilar artery to have higher GCS scores (thus, better neurologic status) at the time of treatment. Of patients with poor GCS scores (3 to 10), six (55%) had collateral flow to the basilar artery and five did not. Of patients with better GCS scores (11 to 15), five (71%) had collateral flow to the basilar artery and two did not ($P = .42$).

No significant correlation was found between the number of PCoAs and survival rate. Overall survival

was 38% at 3 months. Of the patients who survived, one had no PCoA, two had one PCoA, and five had two PCoAs. Of the patients who died, one had no PCoA, six had one PCoA, and five had two PCoAs.

No significant correlation was established between the number of PCoAs and neurologic outcome at 90 days. Of the patients with good neurologic outcomes (modified Rankin scores of 0 to 2), none had no PCoA, one had one PCoA, and five had two PCoAs. Of the patients with poor neurologic outcome (modified Rankin scores of 3 to 6), two had no PCoA, seven had one PCoA, and five had two PCoAs.

No significant correlation was found between the degree of collateral basilar artery filling and overall 90-day survival status. Of patients with collateral flow, five survived and six died. Of patients without collateral flow, three survived and four died. Even with the exclusion of patients with proximal basilar thrombosis, a 100% mortality group, no correlation was observed between collateral flow and survival. In patients with middle or distal basilar thrombosis with collateral flow, five survived and one died; and in patients without collateral flow, three survived and three died.

We did find a significant correlation between collateral flow to the basilar artery and 90-day neurologic outcome for the 12 patients without a clot in the proximal basilar artery. Of those with collateral filling of the distal basilar artery, five (83%) had a good neurologic outcome and one (17%) did not. Of those without collateral flow to the basilar artery, one (17%) had a good neurologic outcome and five (83%) did not ($P = .04$). Of the six patients with a clot in the proximal basilar artery, two had no collateral flow and four had distal and proximal collateral basilar artery filling; all six had a poor neurologic outcome.

Discussion

It is a common perception that collateral circulation to cerebral territories is beneficial in patients who have suffered thromboembolic stroke. This is supported by angiographic data in several studies of acute stroke (19–24). The PCoAs, when present, connect the internal carotid arteries to the posterior cerebral arteries. In cases of basilar thrombosis, if thrombus does not extend into the P1 segment (the portion of the posterior cerebral artery between the

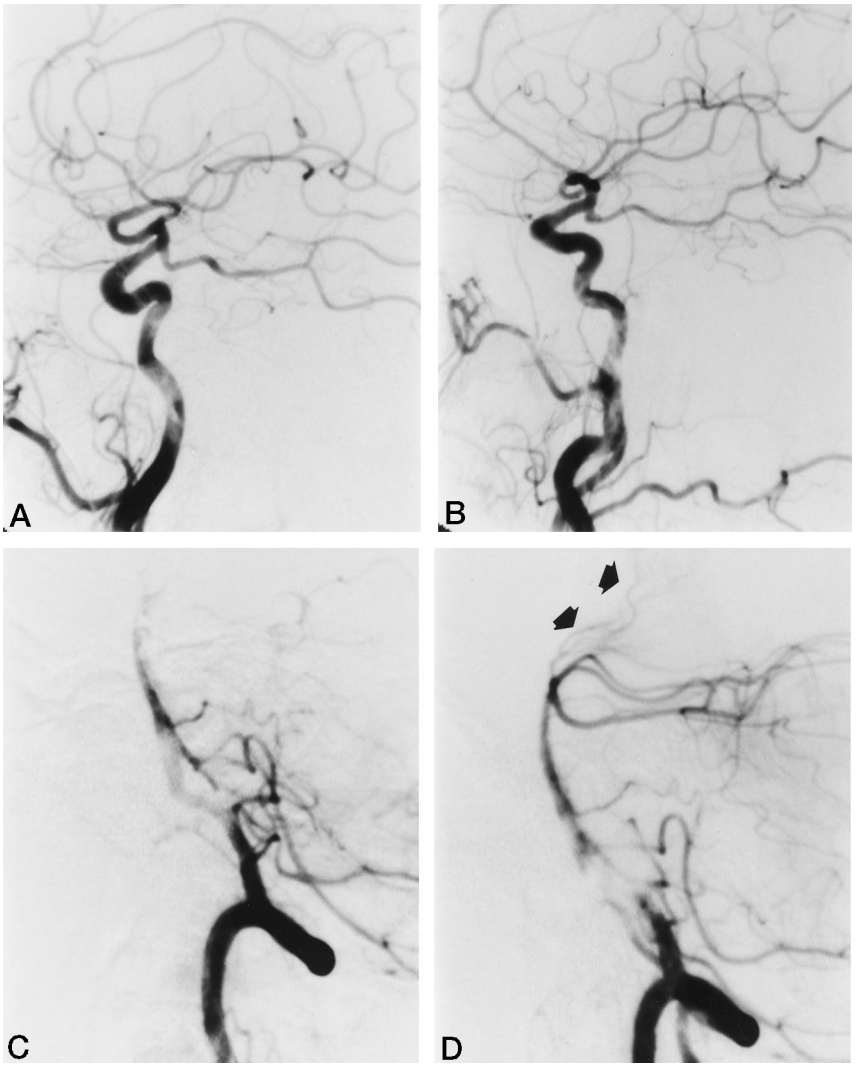

FIG 2. Case 15: middle basilar artery thrombus with no basilar artery collaterals.

A and *B*, The right (*A*) and left (*B*) posterior cerebral arteries are of direct carotid (persistent fetal) origin. Without P1 segments, there is no collateral filling of the basilar artery, but the posterior cerebral arteries fill normally. Note that no thalamoperforating arteries are visible.

C and *D*, Left vertebral injections before (*C*) and after (*D*) recanalization show that the thalamoperforating arteries (*arrows, D*) fill from the basilar tip, even though there are no P1 segments to fill the posterior cerebral arteries. This patient died despite basilar artery recanalization.

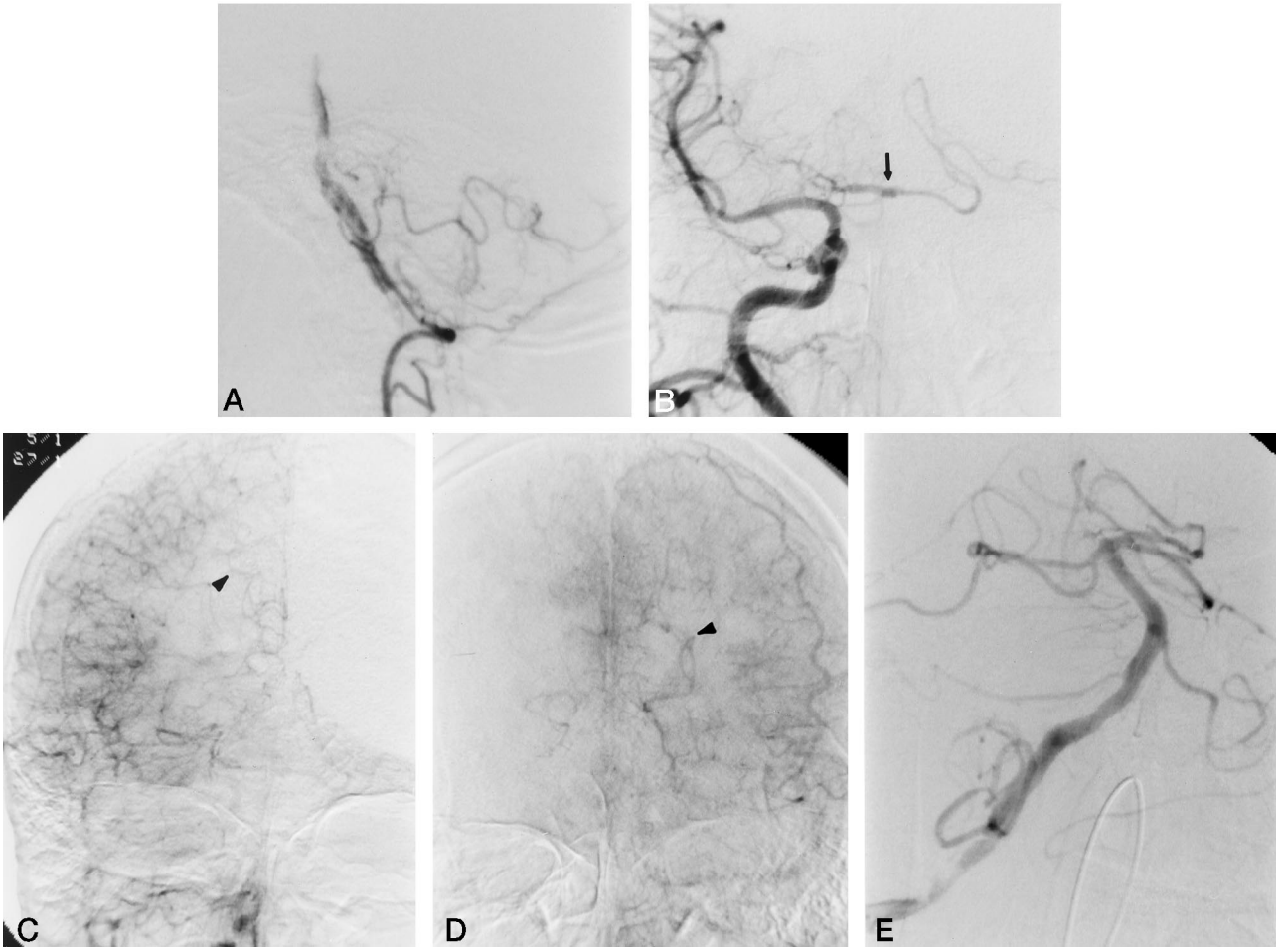

FIG 3. Case 14: distal basilar thrombosis with collateral filling of the basilar tip.

A, Lateral projection of the initial left vertebral injection shows an occlusion of the distal basilar artery.

B, Frontal projection of the right common carotid injection shows a small right posterior communicating artery filling the basilar tip (*arrow*), the right and left superior cerebellar arteries, and the proximal portion of the right posterior cerebral artery.

C, Later in the same injection, the distal right posterior cerebral artery fills in a retrograde direction via pial collaterals (*arrowhead*).

D, There is no left posterior communicating artery, and the left posterior cerebral artery also fills in a retrograde direction via pial collaterals (*arrowhead*).

E, After thrombolysis, all distal basilar branches fill in an anterograde direction. This patient survived.

PCoA and the basilar artery) and if it does not fully obstruct the basilar tip, there is a route for collateral flow to reach the basilar artery from the carotid artery. We sought to determine whether such collateral flow was beneficial in a setting in which brain stem perforating artery origins were likely to be obstructed by basilar artery thrombus.

We included data on angiographically visible collateral filling of the posterior communicating and basilar arteries only if such collaterals had been evaluated by carotid artery injections performed before successful thrombolysis or if thrombolysis had been unsuccessful in recanalizing the clot. We excluded data from patients who had carotid artery injections performed only after successful thrombolysis, since collateral flow to the basilar artery would be underestimated in that group.

Cerebellar artery–to–cerebellar artery collateral flow and pial collateral filling of the posterior cerebral arteries, although observed in several cases, did not opacify the basilar artery, except in one patient in whom PCoA collateral flow also opacified the basilar artery. Distal collateral routes such as these, while probably protective for the occipital lobes and cerebellum, did not appear to provide protection for the brain stem by themselves, so circle of Willis collaterals were judged to be more important to survival.

Distal basilar thrombosis tended to be embolic, while middle and proximal basilar thrombosis tended to be the result of thrombosis on atherosclerotic lesions. While the extent and degree of basilar filling by collateral flow may have been dependent on the location of the clot and hence on the underlying pathogenesis of the clot, we examined the relationship between clot location and outcome in our prior study and confined our analysis in this study to the relationship between collateral filling of the basilar artery and outcome. In view of our mortality statistics in patients with proximal clot, the clot location and its underlying pathogenesis may play a more important role in predicting survival than does collateral flow.

The data in this series indicate that while the num-

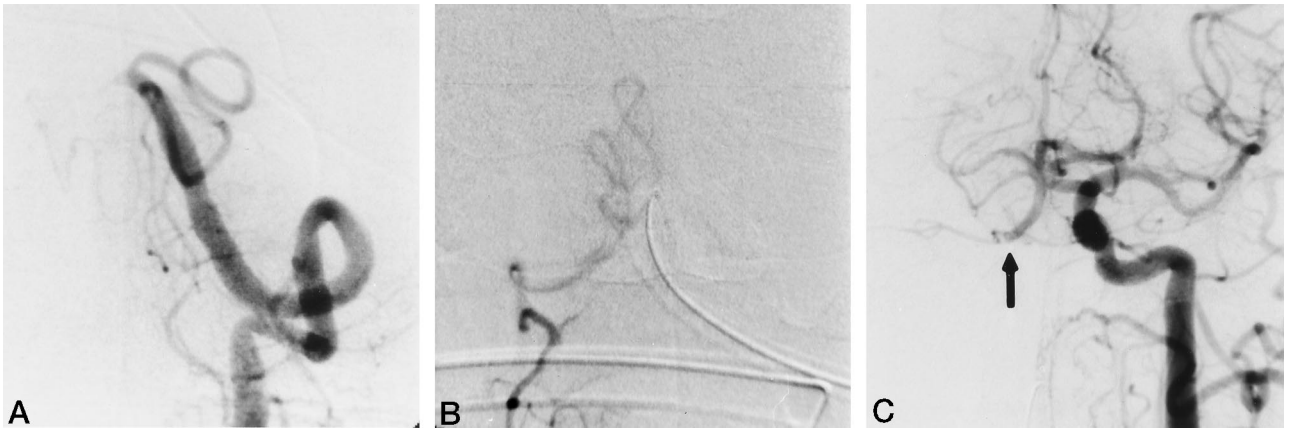

FIG 4. Case 11: proximal basilar thrombosis with collateral filling of the distal and proximal basilar artery via large circle of Willis connections.

A and *B*, Initial left and right vertebral injections show occlusions immediately distal to the posterior inferior cerebellar arteries.

C, Left carotid injection shows collateral filling of the basilar artery via a large left posterior communicating artery. The basilar artery fills in a retrograde direction to a point just proximal to the anterior inferior cerebellar arteries (*arrow*). The right posterior communicating artery was of moderate size and filled the right posterior cerebral artery on the right carotid injection (*not shown*). Recanalization with intraarterial urokinase was unsuccessful, and this patient died despite the extensive collateral flow to the basilar artery.

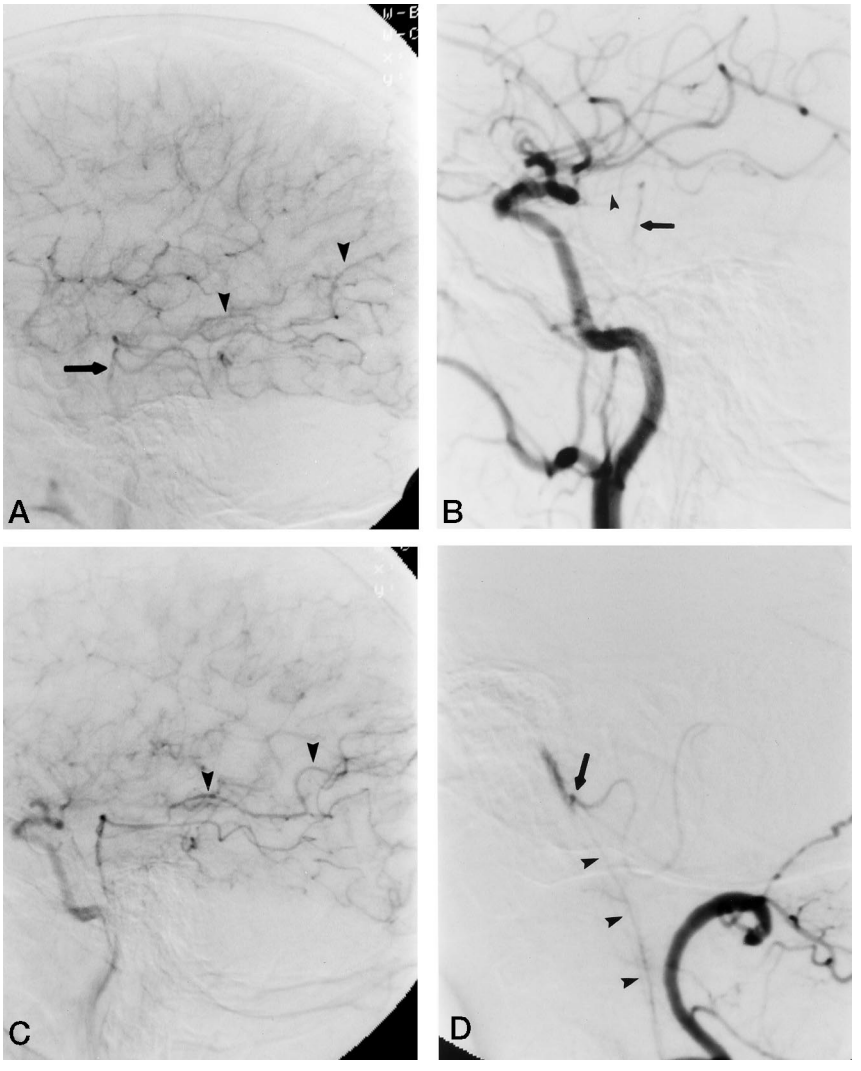

FIG 5. Case 10: proximal basilar thrombosis with collateral filling of the distal and proximal basilar artery via small and remote connections.

A, The right posterior communicating artery is not present on the right common carotid injection, but the right posterior cerebral artery (*arrowheads*) fills late in a retrograde direction via pial connections. The posterior cerebral artery, in turn, fills the basilar artery in a retrograde direction (*arrow*).

B, The left posterior communicating artery is tiny (*arrowhead*) on the left common carotid injection, but it contributes flow to the basilar artery (*arrow*).

C, The left posterior cerebral artery (*arrowheads*) fills later on the left carotid injection, predominantly via pial connections and in a retrograde direction.

D, The cervical right vertebral artery fills the anterior spinal artery (*arrowheads*), which in turn fills the contralateral distal vertebral artery and posterior inferior cerebellar artery (*arrow*) in this case of bilateral distal vertebral and proximal basilar occlusion. Limited circle of Willis connections led to recruitment of smaller and more remote collaterals. Recanalization was unsuccessful and this patient died.

ber of PCoAs (and possibly their size) was irrelevant, the degree to which the PCoAs actually filled the basilar artery was relevant to both clinical presentation and neurologic outcome after basilar artery thrombolysis. Patients with collateral filling of the basilar artery had longer duration of symptoms before referral for thrombolysis and did better after thrombolysis (83% survival, 83% good neurologic outcome) than did patients without collateral filling of the basilar artery (50% survival, 17% good neurologic outcome) if they were not in the high mortality subgroup with proximal basilar artery thrombus. These data support and supplement those of previous studies, such as two (17, 18) that found better outcomes after basilar artery ligation and one (14) that found better outcomes after basilar thrombosis when good PCoA collaterals were demonstrated.

Are the longer duration of symptoms before clinical deterioration and better outcome after thrombolysis solely the result of collateral filling of the basilar artery or do they reflect the fact that there is less obstructive thrombus when collateral filling of the basilar artery is present? Do PCoA collaterals to the basilar artery matter if important basilar artery branches are occluded at their origins by thrombus? These questions are not easily answered, but regardless of the underlying explanation, our findings lead to the conclusion that the presence of collateral filling of the basilar artery by PCoAs in cases of middle or distal basilar artery thrombosis is a favorable prognostic sign in patients undergoing thrombolysis. On the other hand, symptomatic patients with acute proximal basilar artery thrombosis had little chance of survival after thrombolysis, independent of the degree of collateral filling of the basilar artery.

Taken together with prior reports, it appears that the favorable prognostic factors for patients with symptomatic basilar artery thrombosis undergoing intraarterial thrombolysis include distal clot location, complete basilar artery recanalization, and collateral filling of the basilar artery. Performing carotid injections before the initiation of urokinase therapy for basilar thrombosis has advantages and disadvantages, offering helpful prognostic information but potentially delaying initiation of therapy for the symptomatic obstructive lesion. On the basis of these data, simply proving that favorable collateral flow to the basilar artery exists in a symptomatic patient with basilar thrombosis should not lead to the conclusion that such a patient would survive without thrombolysis. Brain stem signs and symptoms indicate a lack of adequate perfusion of basilar artery branches, regardless of the observed collateral flow, and all the patients with basilar thrombosis who survived in this series underwent intraarterial thrombolysis.

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