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Arterial Enhancement in Acute Cerebral Ischemia: Clinical and Angiographic Correlation

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PURPOSE: To investigate the cause and clinical significance of arterial enhancement (AE) in contrast-enhanced T1-weighted MR examinations after acute cerebral ischemia. **METHODS:** Contrast MR examinations and conventional angiograms of 17 patients studied following an acute ischemic event or an internal carotid occlusion were retrospectively reviewed. MR and angiographic studies were performed within 1 day of each other. The presence of AE was correlated with both angiographic findings and patient clinical status. **RESULTS:** AE was not confined to patients with angiographic evidence of complete arterial occlusion. Only 64% of patients demonstrating AE had complete occlusion angiographically. Complete arterial occlusion did not always correlate with AE. In two of nine patients with complete occlusion, no AE was identified. In five of 10 patients with AE, angiographic slow flow was identified. In patients without AE, no angiographic slow flow was identified. In the 64% of patients with AE, significant symptoms were identified. Patients without AE were either asymptomatic or had mild symptoms at the time of the MR study. **CONCLUSIONS:** Our data support the hypothesis that arterial slowing is the cause of AE, which appears to be an indicator of decreased brain perfusion. Such MR findings may add important supplemental information to those provided by conventional angiography.

Index terms: Arteries, magnetic resonance; Brain, ischemia

Although magnetic resonance (MR) findings of arterial enhancement (AE) during acute cerebral ischemia have been reported recently (1–4), the mechanism of AE and clinical significance of the phenomenon, to our knowledge, have not been well established. We retrospectively correlated the presence or absence of AE with angiographic examinations and the clinical status of the patient at the time of the MR examination to investigate the clinical significance of AE and the hemodynamic factors that are represented by AE.

Materials and Methods

Seventeen patients (11 men, six women; age range, 18–74 years; average age, 51 years) were studied with contrast-enhanced MR imaging and conventional angiography following an acute cerebral vascular event or a balloon occlusion of the internal carotid artery. The procedures were performed within 1 day of each other. Fifteen of the 17 patients were studied after acute supratentorial cerebral vascular events. Two patients were studied immediately after balloon occlusion of the internal carotid artery. Each patient's medical record was retrospectively reviewed with respect to the time between the vascular event and MR examination, the time between the MR and angiographic study, the clinical severity of symptoms at the time of the MR examination, and the location of the ischemic region as defined by the discharge diagnosis. The day of the cerebral vascular event was designated as day 0. Only patients imaged from day 0 through day 4 were included in the study.

Imaging was performed with both medium-field (0.5 T, six patients) and high-field (1.5 T, 11 patients) strength scanners. In each patient, after an intravenous injection of 0.1 mmol/kg of gadopentetate dimeglumine, axial and coronal T1-weighted, 350-750/15-24/1-2 (TR/TE/excitations), images were acquired. In 15 of the 17 patients, precontrast T1-weighted examinations in at least one plane were obtained. T2-weighted, 2000-2350/90–100/1-2, im-

TABLE 1: AE vs arterial stenosis

	No. of Patients with Complete Obstruction	No. of Patients with Stenosis
AE (n = 11)	7	2 severe 1 moderate 1 mild
No AE (n = 6)	2	2 moderate 2 mild

Note.—AE = arterial enhancement.

ages were obtained in 16 cases. Abnormal AE was defined as asymmetric vascular enhancement in the involved vascular distribution after contrast administration.

Cerebral angiography was performed with conventional biplane cut film technique in 15 cases. Two patients underwent digital angiography. All cerebral angiograms were evaluated for occlusion or stenosis of vessels in the involved vascular distribution. Complete occlusion was defined as absence of antegrade fill of vessels distal to an obstruction. Severe stenosis was defined as greater than 75% narrowing in at least one plane with antegrade fill of vessels distal to the stenosis. Moderate stenosis was defined as narrowing greater than 50% but less than or equal to 75%. Lesser degrees of narrowing were defined as mild.

Angiograms were evaluated for evidence of slow arterial blood flow. Because of the retrospective nature of this study and the variability of filming rates, an accurate circulation-time calculation could not be made. The angiographic criterion for decreased flow was either 1) complete arterial obstruction with retrograde fill of vessels by collateral circulation or 2) severe stenosis causing a delay in antegrade fill of more distal vessels when compared with flow in nonobstructed vessels. Because of the lack of rapid sequential filming, we were unable to evaluate arterial slowing in the two patients studied with digital angiography.

Nine of the 17 cases have been reported in an earlier publication (1). These patients were reevaluated independently and further characterized with respect to degree of arterial stenosis, arterial slowing, and clinical status at the time of the study.

Results

Correlation of Angiographic Findings with MR Evidence of AE

The relationship between the degree of arterial occlusion identified angiographically and the presence or absence of AE is summarized in Table 1. AE was identified in six patients in which the affected vessel was severely stenosed or occluded distal to the circle of Willis (Figs. 1 and 2). Complete vascular occlusion was not a requirement for AE. Complete occlusion could be demonstrated in only seven of the 11 patients in whom AE was identified. Two of the four patients with complete occlusion of the internal carotid artery did not demonstrate AE (Fig. 3).

The relationship between angiographic evidence of slow arterial flow and AE in the 15 patients studied with conventional angiography is summarized in Table 2. Angiographic evidence of slow flow was always associated with AE (Fig. 1) and was not identified in patients without AE. In five patients with AE, slow flow could not be documented. In one of these patients, complete proximal intracranial occlusion was present, but collateral flow could not be demonstrated angiographically. In another patient with complete occlusion of the internal carotid, the middle cerebral artery was opacified prominently by collaterals via the ophthalmic artery. The middle cerebral was not opacified during injection of the contralateral carotid (Fig. 4).

In one instance, there was irregularity of a single small frontal branch of the middle cerebral artery. This was disproportionate to the much larger focus of AE. Therefore, it was thought that this obstruction did not account for the AE, and the degree of stenosis was classified as mild.

AE versus Clinical Symptoms at the Time of MR Examination

Patients with AE tended to have more severe clinical symptoms than those without AE (Table 3). The six patients without AE were either asymptomatic (three patients) or had mild symptoms of clumsiness or weakness (three patients) at the time of the MR study.

Seven of the 11 patients demonstrating AE manifested significant symptoms (aphasia, hemiparesis, nystagmus, visual field defects, sensory deficits) at the time of the MR study.

Fig. 1. Radiologic study of a 37-year-old man who underwent an MR examination 3 days after an acute ischemic event. Physical examination, at the time of the MR study, identified an altered mental status, hemiparetic gait, and a left visual field defect.

A, Right common carotid cerebral angiogram. Absence of filling in the insular vessels secondary to occlusion of the middle cerebral artery.
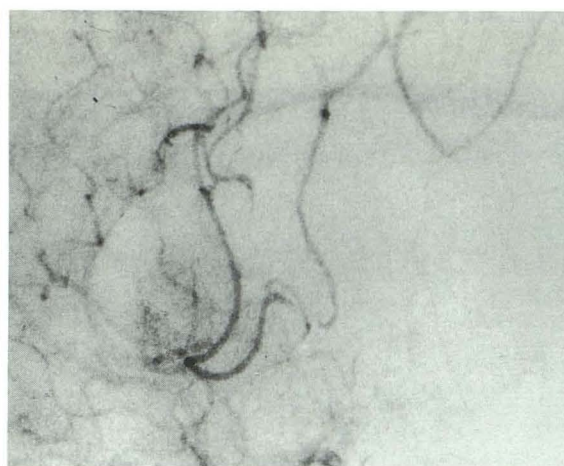
B, Same injection as *A*. Delayed opacification of insular vessels.

C, D, and *E*, Gadolinium-enhanced T1-weighted MR images. There is AE involving the right middle cerebral artery distribution.
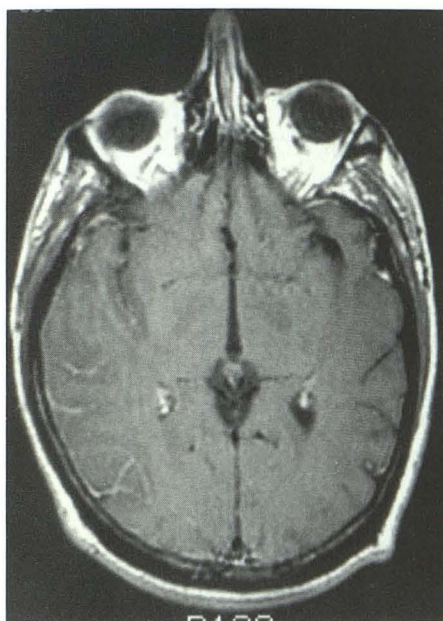
F, G, and *H*, Corresponding T2-weighted images demonstrating T2 prolongation in the adjacent brain parenchyma.
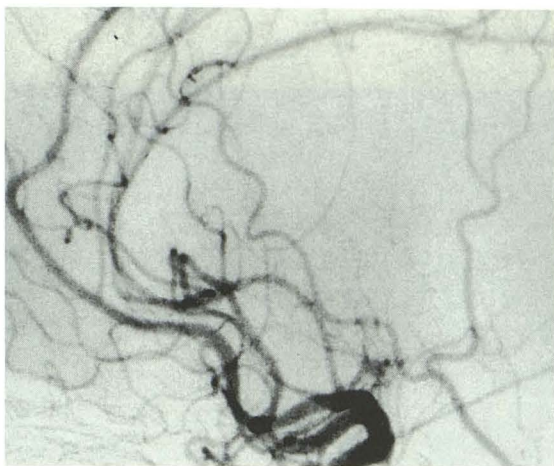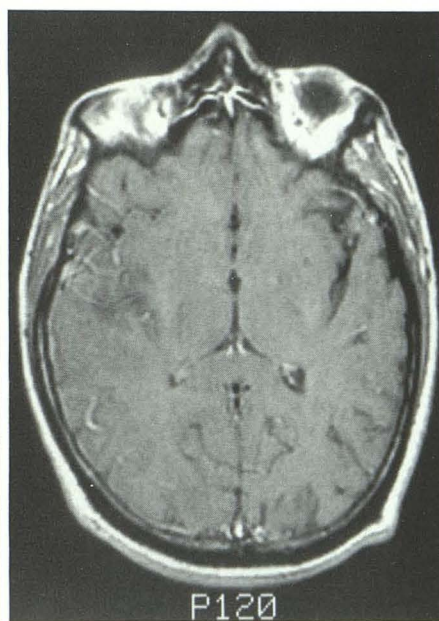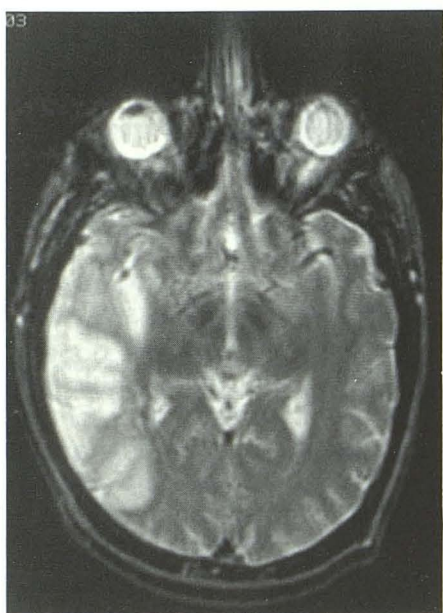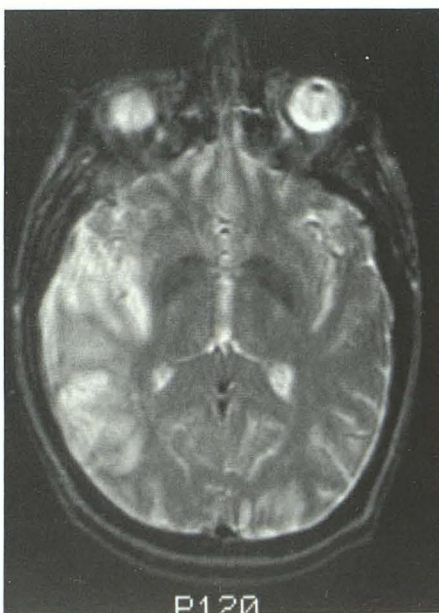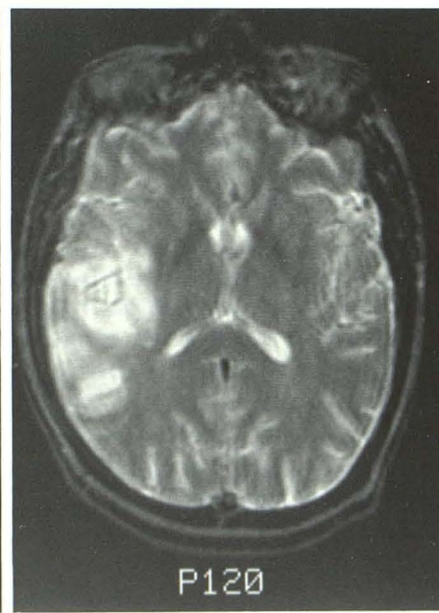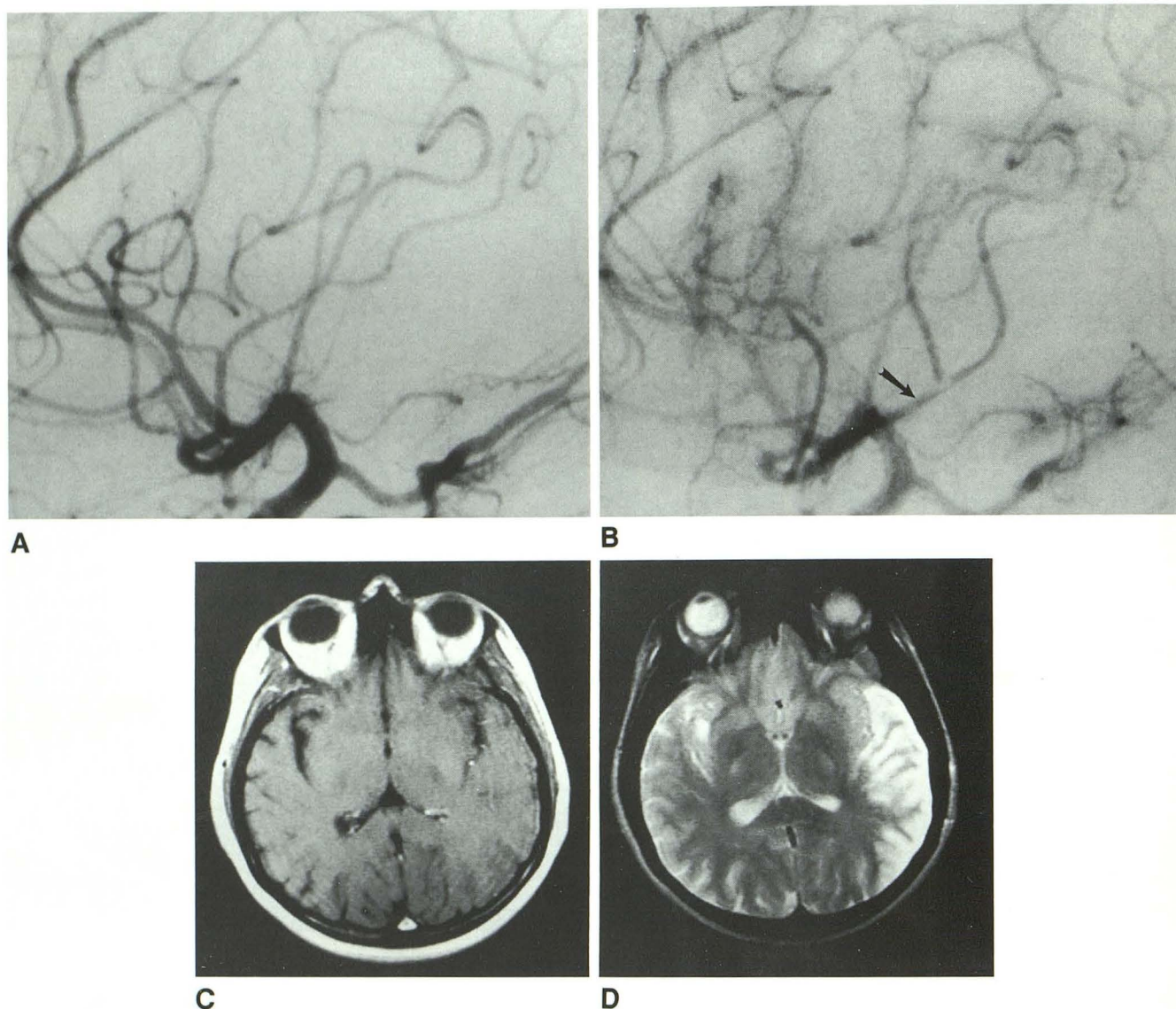
A

B

C

D

E

F

G

H

Fig. 2. A 32-year-old woman was studied by MR on the day following a left hemispheric cerebral infarction. On the day of examination, the patient was described as disoriented with incoherent speech. The final diagnosis was severe Wernicke-type aphasia.

A and *B*, Left common carotid angiogram. There is delayed opacification of middle cerebral branches (*arrow*).

C, Gadolinium-enhanced T1-weighted MR study demonstrates AE.

D, Corresponding T2-weighted image. There is T2 prolongation in the adjacent brain parenchyma.

Discussion

Several reports have evaluated MR findings after cerebral ischemia (1–6). Recently, AE has been reported in contrast-enhanced MR studies obtained during the acute phase of cerebral infarction (1–4). Although slow flow has been considered as a possible underlying cause, the exact mechanism of AE and its clinical significance remain uncertain. Our study suggests that the presence of AE is more consistent with slow arterial flow than with the degree of arterial stenosis or occlusion.

As has been demonstrated by Bradley et al (7) and Hinks and Quencer (8), high vascular flow rates result in signal loss. In our study, none of the patients without AE had angiographic evidence of slow flow. In contrast, 50% of the patients with AE demonstrated slow flow angiographically.

The rationale for the lack of angiographic slow flow in the remaining cases demonstrating AE is uncertain. In three of these five patients, the angiograms were performed on the day after the MR study. It is possible that a change in the hemodynamic status of the patient occurred dur-
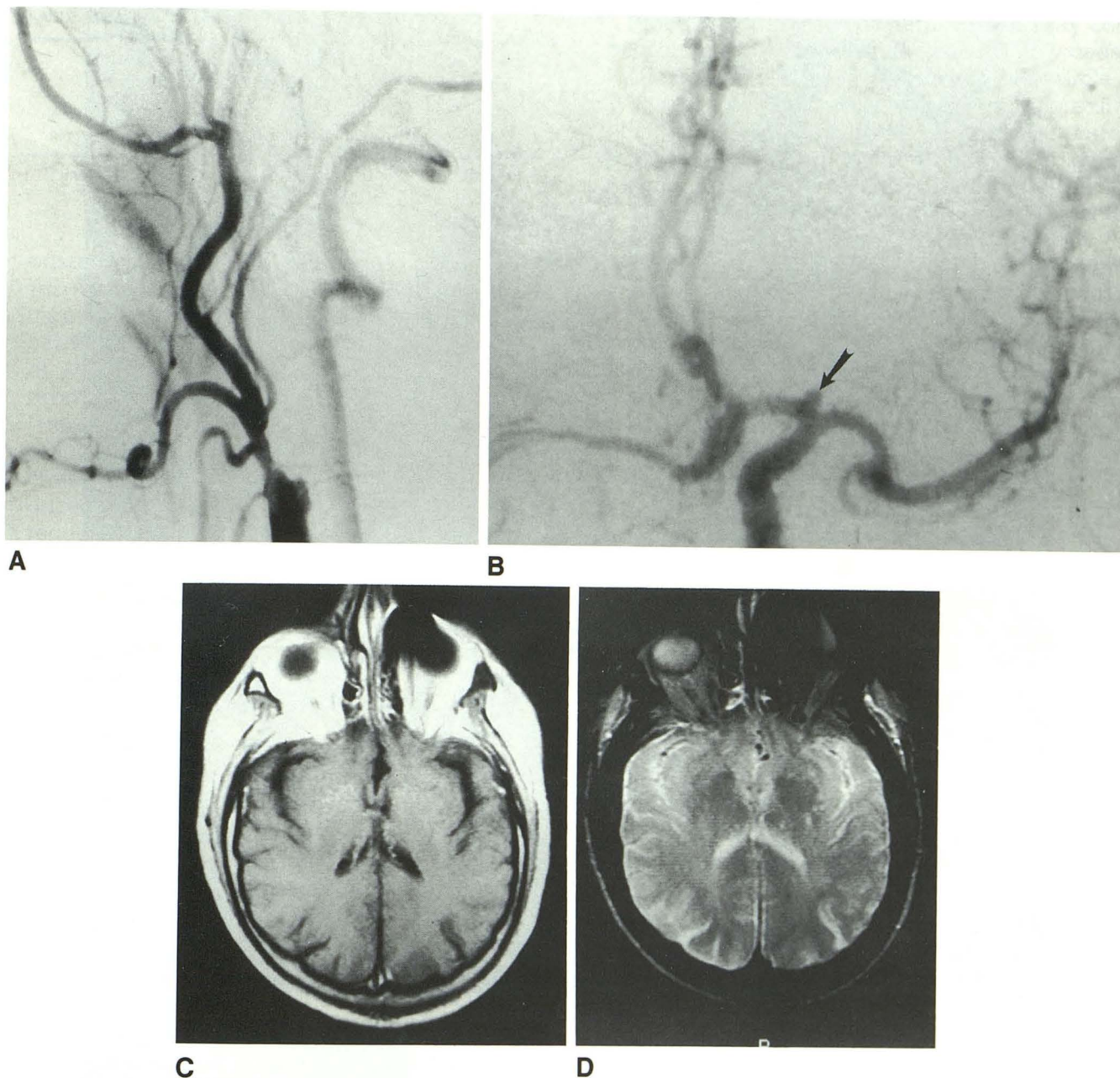
A

B

C   D

Fig. 3. Radiologic study of a 70-year-old man who underwent an MR examination 2 days after an episode of left-handed numbness. The symptoms had resolved at the time of the MR study.

A, Right common carotid angiogram. The internal carotid is occluded. There is reflux into the vertebral artery.

B, Right anterior and middle cerebral arteries are opacified during a left common carotid angiogram. An aneurysm (*arrow*) is identified near the left internal carotid bifurcation.

*C*, Gadolinium-enhanced T1-weighted MR study. There is no AE.

D, Corresponding T2-weighted image. No evidence of parenchymal abnormality was noted.

TABLE 2: AE vs angiographic arterial slowing

	No. of Patients with Angiographic Slow Flow	No. of Patients without Angiographic Slow Flow
AE (n = 10)	5	5
No AE (n = 5)	0	5

Note.—The two patients who underwent digital angiography were not included in this analysis. AE = arterial enhancement.

ing this interval. Alternatively, AE may be more sensitive to decreased flow rate than the angiographic criteria used in this study. The data from two patients who demonstrated AE support this conclusion. In the first patient, a complete intracranial occlusion was identified angiographically. Slow arterial flow would be expected distal to the occlusion; however, collateral flow was not dem-
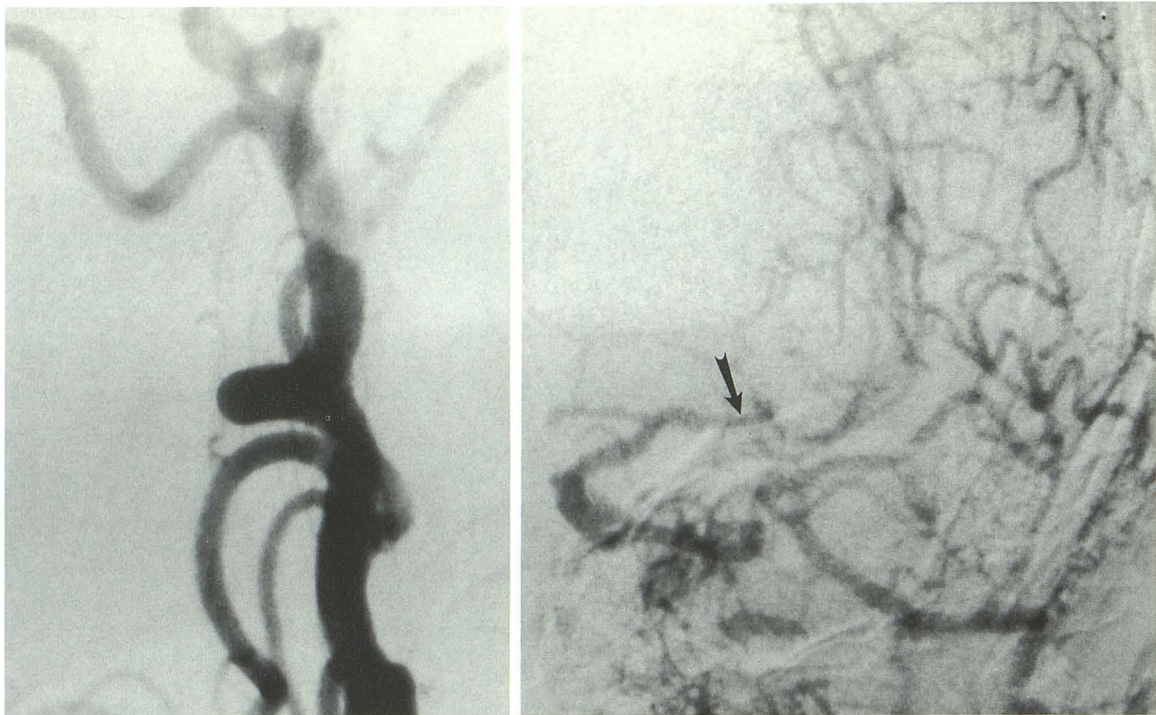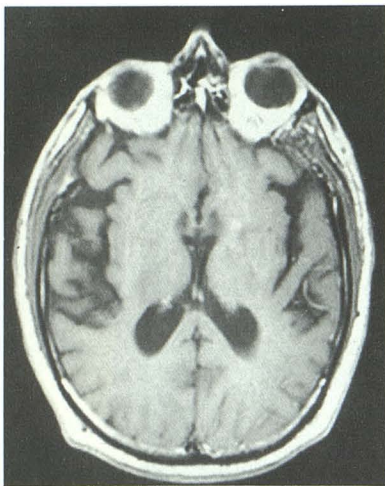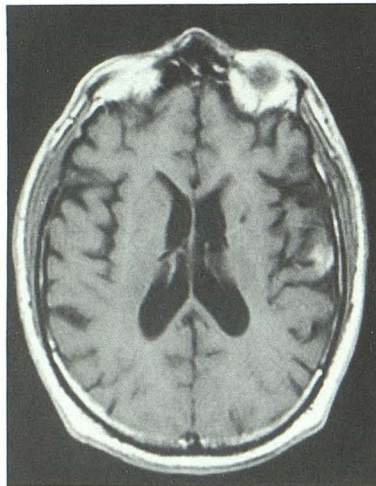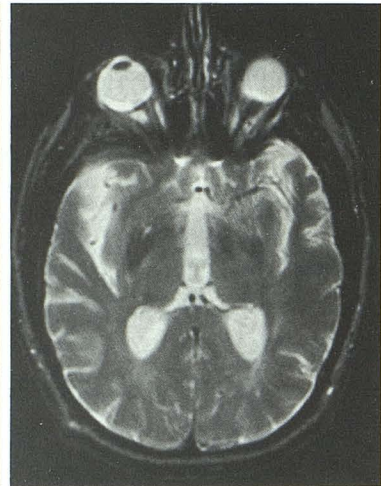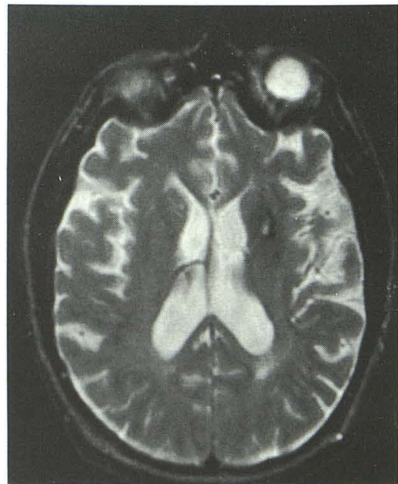
Fig. 4. A 61-year-old man with a history of transient ischemic attacks, the most recent being an episode of aphasia on the day before admission. There were no apparent speech deficits at the time of examination.

A and *B*, Left carotid angiogram. There is complete occlusion of the internal carotid artery. The middle cerebral artery (*arrow*) fills by external carotid collaterals.

C and *D*, T1-weighted MR study following gadolinium administration. There is abnormal AE as well as parenchymal enhancement, indicative of ischemia.

E and *F*, Corresponding T2-weighted images. No apparent parenchymal abnormality was noted.

TABLE 3: AE vs clinical symptoms

	No. of Patients with Severe Symptoms	No. of Patients with Mild or No Symptoms
AE (n = 11)	7	4
No AE (n = 6)	0	6

Note.—AE = arterial enhancement.

onstrated angiographically. In the second patient, AE was identified in association with internal carotid artery occlusion. The ipsilateral middle cerebral artery did not fill via the anterior circle of Willis. The middle cerebral was, however, prominently opacified by ophthalmic artery collaterals. In five patients with a similar angiographic pattern, Powers et al (9), using positron emission tomography (PET), demonstrated reduced cerebral perfusion pressure in all five, and reduced flow in four. Their findings suggest that decreased flow that was not documented angiographically may have been present in our patient.

The cause of arterial slowing distal to a vascular lesion is probably related to increased total resistance to flow in the involved arterial circulation. This phenomenon can be explained by the following formula:

$$F = P/R$$

where F = flow, P = pressure, and R = resistance.

Although systemic blood pressure affects the entire cerebrovascular circulation, a focal increase in resistance will result in a decreased flow that is limited to the affected vessel and vessels that are supplied by it. Resistance is dependent on the effective diameter of the vessel lumen. It can be demonstrated that resistance is inversely proportional to the fourth power of the lumen radius (10). Thus, a severe stenosis or occlusion will greatly increase resistance. If collateral circulation cannot compensate adequately, a decreased flow rate through the affected vascular tree would be expected. Vascular narrowing may involve either proximal or distal vessels. Proximal occlusion or stenosis can occur secondary to thrombus, emboli, or plaque. Small emboli may occlude smaller, more distal arteries. Increased resistance may also occur at the level of the capillaries. The "no-reflow phenomenon" (11, 12) has been used to explain distal vascular obstruction. This theory suggests that compression of capillaries may occur as the result of edema involving endothelial cells and perivascular glial cells.

Blood flow through collateral vessels may compensate for the expected decrease in flow caused by increased vascular resistance. The circle of Willis can provide collateral flow around proximal obstructions. Pial collaterals on the brain surface offer a potential source of collateral blood supply for more distal obstructions (13, 14). If sufficient flow is maintained, AE would not be expected. Therefore, slow blood flow (as evidenced by AE) is reflective of both increased vascular resistance and the effectiveness of collateral circulation. Complete or severe stenosis distal to the circle of Willis resulted in AE in six patients. Circulation distal to the circle of Willis is dependent on residual flow through the obstructed vessel and collateral flow through pial vessels. The presence of AE suggests that residual flow through the stenotic focus and collateral flow through pial vessels were not adequate to maintain normal flow rate.

AE was identified in two of the four patients with complete occlusions proximal to the circle of Willis. Differences in hemodynamic response to internal carotid occlusion (9) may be responsible for the variability of AE seen in our four patients with carotid occlusion. We think that patients demonstrating AE had relatively reduced flow rates compared with those that did not demonstrate AE.

In one patient, no structural lesion could be identified angiographically to account for AE. We suspect that increased distal resistance either by emboli or perivascular edema may have been the cause of vascular slowing. Because of the small size of the vessels involved, these lesions would not be visualized angiographically. The distal nature of the obstruction would inhibit collateral flow.

Our study further suggests that a relationship exists between AE and the clinical status of the patient. Sixty-four percent of our patients with AE had significant symptoms at the time of the MR examination. In contrast, all patients without AE were either asymptomatic or had mild symptoms at the time of the MR study. These results suggest that AE is an indicator of diminished brain perfusion.

The significance of the AE identified in the four patients with mild or no symptoms is uncertain. Previous work using PET to study patients with a history of transient ischemic attacks demonstrated a subset of asymptomatic patients with decreased cerebral blood flow (15). In these individuals, oxygen metabolism was maintained by

increasing the percentage of oxygen extracted from the blood. It is possible that a similar mechanism was present in our patients. AE may have been indicative of decreased flow; however, increased oxygen extraction may have compensated.

In conclusion, our data support the hypothesis that arterial slowing is the cause of AE. The presence of AE depends on the degree of vascular stenosis and also the ability of other vessels to provide adequate collateral flow. AE appears to be an indicator of decreased cerebrovascular perfusion. This ability to assess the effectiveness of collateral flow by MR offers the potential to add important supplemental information to the findings provided by conventional angiography. A prospective study comparing MR and PET imaging would be of great value in further defining the relationship between AE and cerebral perfusion.

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