The following is in response:

To the Editor:

I appreciate the interest of Drs. Struck, Rodnitzky, and Sloan in the increased incidence of ischemic stroke in the morning. Knowledge about the factors that precipitate ischemic stroke in patients at risk may provide insight that would lead to new therapeutic and preventive strategies. Endogenous dopamine and fibrinolytic activity may well be very important factors. If so, it will be a very challenging problem to design scientific studies capable of testing the hypothesis and determining the underlying mechanisms.

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Anterior Choroidal Artery Territory Infarction and Small-Vessel Disease

To the Editor:

The report of Bruno et al describes in detail 31 patients with anterior choroidal artery (AChA) territory infarction, diagnosed by demonstrating an appropriate area of infarction on computed tomographic (CT) scan. Arteriography or noninvasive carotid artery studies were available in 27 of the 31 patients and showed only mild or moderate stenosis in four patients. Intracranial arteriography in 17 patients demonstrated patency of the AChA in 15 patients and a nonvisible AChA in two patients. Based upon these observations, the authors conclude that extracranial arterial disease is rarely the cause of AChA territory stroke and that small vessel disease is the likelier etiology for most of these strokes.

We have encountered two patients with AChA territory infarcts who suggest that these conclusions may need to be reconsidered. Both patients suddenly developed a hemiparesis and incongruous homonymous hemianopsia. Additionally, one patient also had evidence of a hemisensory deficit. CT scan in each patient demonstrated an infarction in the AChA vascular territory.

The report of Bruno et al describes in detail 31 patients with anterior choroidal artery (AChA) territory infarction, diagnosed by demonstrating an appropriate area of infarction on computed tomographic (CT) scan. Arteriography or noninvasive carotid artery studies were available in 27 of the 31 patients and showed only mild or moderate stenosis in four patients. Intracranial arteriography in 17 patients demonstrated patency of the AChA in 15 patients and a nonvisible AChA in two patients. Based upon these observations, the authors conclude that extracranial arterial disease is rarely the cause of AChA territory stroke and that small vessel disease is the likelier etiology for most of these strokes. We have encountered two patients with AChA territory infarcts who suggest that these conclusions may need to be reconsidered. Both patients suddenly developed a hemiparesis and incongruous homonymous hemianopsia. Additionally, one patient also had evidence of a hemisensory deficit. CT scan in each patient demonstrated an infarction in the AChA vascular territory. Angiography in the first case revealed a large ulcerated plaque at the origin of the internal carotid artery and occlusion of the AChA just distal to its origin (Figure 1). Angiography in the second case demonstrated complete occlusion of the internal carotid artery at its origin. The ipsilateral middle and anterior cerebral arteries were visualized, but the AChA was not with injection of the contralateral carotid artery. Neither patient had a history of hypertension or a potential source for cardiogenic embolism, although one patient did have dietary-controlled diabetes mellitus.

These two patients suggest that AChA infarcts may occur in association with proximal internal carotid artery abnormalities. The AChA occlusion presumably occurred on the basis of artery-to-artery embolization, and the findings in the first case strongly support this hypothesis. These two cases suggest that not all AChA infarcts occur on the basis of small vessel disease and that at least a noninvasive carotid artery evaluation should be considered in these patients. Proximal extracranial arterial disorders are
being increasingly identified in patients with lenticulostriate artery lacunar syndromes, and it is not surprising that a percentage of AChA territory infarct patients will also harbor such lesions.2

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References


The following is in reply:

To the Editor:

We read with interest about the experience of Dr. Fisher and his colleagues regarding anterior choroidal artery (AChA) territory infarction. We reported carotid artery studies on all 31 of our patients with AChA territory infarction1 and not 27 as stated by Dr. Fisher in his letter. The first case of AChA territory infarction presented by Dr. Fisher suggests an embolic AChA occlusion. The second case is associated with ipsilateral internal carotid artery occlusion and nonvisibility of the AChA. Nonvisibility of the AChA on arteriography should not be interpreted as an abnormality since it occurs in about 5% of normal studies.2 Small size of the AChA and obscuration by middle cerebral artery branches are contributing factors. The abstract referenced by Dr. Fisher in his letter3 does not refer to the AChA and, therefore, is not pertinent to our article. We reported patients with brain infarction in a specific vascular territory and not lacunar infarction in general.

Based on our experience with 31 patients with AChA territory infarction1 and 55 additional patients in other reports,4–7 the two cases presented by Dr. Fisher are interesting and unusual, but they do not invalidate our conclusion that AChA territory infarction is usually caused by small vessel disease.

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Effect of Insulin on Acute experimental Cerebral Ischemia in Gerbils

To the Editor:

We would like to comment on the article by Fukuoka and coworkers1 in the March 1989 issue of Stroke regarding the effect of insulin after transient common carotid artery occlusion in Mongolian gerbils. The results showed that nonhypoglycemic animals treated with insulin had the most favorable outcome. In this study, glycemia after ischemia was not monitored, and the effects of plasma glucose concentration in the first hours of the experiment could have influenced the final results.

We compared the effects of serum glucose concentration on the neurologic outcome2 and mortality in a group of Mongolian gerbils subjected to unilateral common carotid artery occlusion. Group A was treated with 3.5 mg/kg of glucose intraperitoneally immediately after carotid occlusion and 1.75 mg/kg at subsequent intervals of 30, 60, and 180 minutes. Control Group B was treated with 0.9% saline injections and Control Group C was treated with 8.0% saline injections. After the first 210 minutes of the experiment, Group A animals demonstrated more severe neurological disability (p<0.00001). In a similar experiment2 with gerbils made hyperglycemic by administration of 3.5 mg/kg of glucose i.p. immediately after unilateral common carotid artery occlusion, we compared cerebral neuropathologic changes 24 hours later with a control treated with 0.9% saline injections. In the hyperglycemic gerbils, we found a greater degree of neuronal ischemic changes (p<0.05) and, unlike the normoglycemic gerbils, most brains exceeded the stage of selective neuronal necrosis (p<0.01).

We conclude that experiments dealing with the protective effects of insulin on cerebral ischemia should take into account the plasma glucose concentration during and immediately after ischemia.

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References

Anterior choroidal artery territory infarction and small-vessel disease.
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