Carotid Angiography in Patients With Lacunar Infarction
A Prospective Study

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We prospectively studied the results of carotid angiography in 45 patients with transient or nondisabling neurologic deficits caused by lacunar infarction in the internal capsule or corona radiata and demonstrated by computed tomography. An ipsilateral stenosis at the bifurcation of the internal carotid artery was found in 14 patients (31%, 95% confidence limits 18–47%), seven of whom also had stenosis of the contralateral internal carotid artery. In previous studies an average of 65% of patients with transient hemispheric deficit had internal carotid artery stenosis or occlusion. Hypertension and hypertensive retinal vasculopathy assessed by fundus photographs were found in most patients, but not significantly more often in the patients without internal carotid artery stenosis. Our findings support the notion that small vessel disease rather than emboli from the carotid bifurcation is the most common cause of lacunar infarction. (Stroke 1988; 19:1093–1096)

Lacunar infarction constitutes between 10% and 23% of all symptomatic cerebral infarcts.1–5 This type of stroke is considered a separate entity not only because of its small size and deep location but also because of its characteristic clinical features and relatively good prognosis.6–10 However, dilemmas in management arise10 because more than one theory exists to explain the pathogenesis of lacunar infarction. Small vessel disease resulting from hypertension is generally thought to be the most common cause,11–14 but extracranial sources of emboli, such as the heart or the internal carotid bifurcation, have also been found in patients with lacunar infarction.2–5,14–17 Treatment of these extracranial lesions is controversial, especially since such potential sources of emboli might in fact be coincidental.18–20 It is unresolved whether carotid endarterectomy should be undertaken in patients who have had a transient ischemic attack or a minor stroke distal to an atherosclerotic lesion in the extracranial course of that artery, even if the location and size of the infarct are disregarded.21 Before carotid endarterectomy can be considered, selective catheterization of the neck vessels or at least of the aorta (intraarterial digital subtraction angiography) is necessary.

For all these reasons, it is unclear whether patients who recently suffered a lacunar infarction with transient or minor neurologic deficits should undergo carotid angiography with a view to carotid endarterectomy. We have therefore undertaken a prospective study of carotid angiography in patients with symptomatic lacunar infarcts. We studied the frequency of extracranial internal carotid artery (ICA) lesions and the relation between the presence of ICA stenosis and hypertension. Cardiac hypertrophy, retinal vasculopathy, and impaired renal function were assessed as attendant features of hypertension; the latter two measures reflect small vessel disease in general.

Subjects and Methods

From 1982 to 1986, we prospectively studied the results of selective carotid angiography in 45 patients (mean age 59, range 41–69 years) who had suffered transient hemiparesis or a minor stroke with features of a lacunar syndrome and who furthermore proved on computed tomography (CT scanning) to have at least one corresponding lacunar infarct in the internal capsule or the corona radiata. For the definition of the four main lacunar syndromes, we used the criteria of Bamford et al.22 Visual field defect, evidence of higher cerebral dysfunction, or

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clinical features of brainstem deficit excluded the diagnosis of a lacunar syndrome. Pure motor hemiparesis included at least two of three areas (face, arm, leg). If sensation was also shown to be affected on clinical testing, we diagnosed sensorimotor stroke. Ataxic hemiparesis, including the dysarthria-clumsy hand syndrome, consisted of a combination of ipsilateral pyramidal and cerebellar signs. We diagnosed pure sensory stroke in cases of an exclusively sensory deficit involving at least two of three areas (face, arm, leg) and including the abnormal perception of touch, pain, and vibration.

All patients were selected as fit for surgery. The decision to undertake angiography was not guided by the presence of a cervical bruit or by ultrasound assessment. Patients with a possible cardiac source of embolism were excluded. The angiograms were reviewed without knowledge of the clinical features.

Blood pressures were the mean of the first three measurements after admission. A systolic pressure of >160 mm Hg and/or a diastolic pressure of >95 mm Hg were considered to indicate hypertension.

The ocular fundus was photographed to record the retinal vasculature. The photographs were reviewed separately by two ophthalmologists without information about the carotid angiogram and the blood pressure. The presence of hypertensive vascular changes in the retina was assessed according to the Scheie classification23 (H1 and higher and/or S2 or higher were considered to indicate hypertensive vascular changes).

Hypertrophy of the wall of the left ventricle as a measure of chronic hypertension was assessed by means of echocardiography. We determined the creatinine clearance per 1.73 m² body surface, corrected for age and sex, as a measure of renal damage, particularly from small vessel disease.

### Results

Most of our patients (28) suffered from pure motor hemiparesis; the clinical features in the other patients were ataxic hemiparesis (in eight), sensorimotor stroke (in five), and pure sensory stroke (in four). The results of carotid angiography are summarized in Table 1. Stenosis of the ICA was found in 14 patients (31%, 95% confidence limits 18-47%); the degree of stenosis was 0-25% in three patients, 25-50% in five, 50-75% in four, and >75% in two. One patient had an occlusion of the M1 segment and no stenosis of the carotid bifurcation on the symptomatic side. The other 30 patients had normal angiograms or only slight irregularities of the ICA. Half of the 14 patients with ICA stenosis also had a stenosis on the contralateral (asymptomatic) side. Carotid stenosis was not associated with any particular lacunar syndrome (Table 1).

We found arterial hypertension on repeated measurement in 28 patients (62%). There was no correlation with the results of angiography. Fundus photographs were not available in four patients for practical reasons; none of these four had extracranial carotid stenosis, but one had an occlusion of the M1 segment. Both ophthalmologists found retinal abnormalities consistent with hypertension in 32 of the remaining 41 patients (78%). In three patients both ophthalmologists found a normal retinal vasculature while in six patients they disagreed (Table 2). The proportion of patients with hypertensive retinopathy did not differ significantly between the groups with and without carotid stenosis.

Echocardiography was performed in 39 patients, seven of whom (18%) showed hypertrophy of the left ventricular wall. In 18 of 37 patients (49%), creatinine clearance was abnormal. No significant relation existed between ventricular hypertrophy or creatinine clearance and the results of angiography.

### Discussion

We found carotid stenosis in a minority of patients with lacunar infarcts. Moreover, even if such lesions are present, the question is whether they are relevant and whether they should be surgically treated. Those who find angiography indicated in cases of

### Table 1. Results of Carotid Angiography According to Type of Lacunar Stroke in 45 Patients

<table>
<thead>
<tr>
<th>Type of lacunar stroke</th>
<th>Normal</th>
<th>Minor irregularities</th>
<th>Stenosis</th>
<th>Intracranial occlusion</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure motor hemiparesis</td>
<td>13</td>
<td>6</td>
<td>9</td>
<td>0</td>
<td>28</td>
</tr>
<tr>
<td>Ataxic hemiparesis</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Sensorimotor stroke</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Pure sensory stroke</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>12</td>
<td>14</td>
<td>1</td>
<td>45</td>
</tr>
</tbody>
</table>

### Table 2. Abnormalities of Retinal Vessels Related to Angiography of Internal Carotid Artery in 41 Patients With Lacunar Infarction

<table>
<thead>
<tr>
<th>Ocular fundus photography</th>
<th>Angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stenosis</td>
</tr>
<tr>
<td>Hypertensive changes of retinal vessels</td>
<td>11</td>
</tr>
<tr>
<td>Uncertain</td>
<td>3</td>
</tr>
<tr>
<td>No hypertensive changes of retinal vessels</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
</tr>
</tbody>
</table>

Uncertain, disagreement between two ophthalmologists who separately assessed photographs of ocular fundus.
symptomatic lacunar infarction1,2,16,24 anticipate that operable carotid lesions can be found in some of these patients. What proportion of such patients actually exists remains largely unknown. In the only prospective study of angiography in lacunar stroke to date, Olsen et al1 evaluated 15 patients, one of whom proved to have an ICA stenosis of >75% and seven of whom showed a lesser degree of stenosis. In 11 retrospective studies in which the selection criteria for angiography differed widely, the overall mean proportion of operable ICA lesions in patients with lacunar infarction was 22%,2,3,15,16,25-31 We selected only patients in whom CT confirmed a lacunar infarct and who were deemed fit for surgery, as our study was motivated by the clinical dilemma of whether to undertake angiography, with a view to surgery, in similar patients. Ultrasonography or the presence of cervical bruits were not used as screening methods because a few small but operable lesions might have been missed. In our study, we found 31% (95% confidence limits 18-47%) of patients to have stenotic lesions of the ICA and no occlusions. By contrast, in clinically defined groups of patients with transient hemispheric deficit independent of the presence, size, or location of the infarct, the average proportion of stenosis or occlusion angiography is 65% .21-30 Abnormalities of intracranial vessels on angiography were reported only in cases with large lacunes.15,30 The suggestion of Bogousslavsky and Regli,20 that small deep cerebral infarcts with transient neurologic deficits may be particularly frequent in patients with ICA occlusion, could not be confirmed in the reverse sense in this series since we did not find a single occlusion of the ICA.

The examination of retinal vessels in the ocular fundus might give information about vessels of similar size elsewhere in the body, such as in the brain or in the kidneys.40,41 The occurrence of retinal vasculopathy with hypertensive changes in a majority of our patients suggests that small vessel disease caused by hypertension is a main factor in the pathogenesis of lacunar infarction. Indeed, hypertension was found in 62% of our patients, although this may have been an overestimate because the blood pressure was measured early after the stroke. Some other patients also showed hypertensive retinal changes, and the normotensive patients probably had some hypertensive periods in the past. Furthermore, hypertension is common in patients with all types of ischemic strokes and does not necessarily predict strokes of the lacunar type.3 At the same time, normal blood pressure and the absence of hypertensive retinal vasculopathy are not always associated with a normal ICA bifurcation (Table 2). Apparently some additional, as yet unknown, factor determines whether hypertension will affect small or large arteries. Our results support the view that embolism from an atherosclerotic lesion in the ICA is an uncommon cause of lacunar infarction. Unfortunately, the four measures we examined (blood pressure, retinal vasculature, echocardiography, and creatinine clearance) were not helpful in predicting the result of angiography in patients with lacunar infarcts. The relevance of the carotid lesions that we did find remains unknown for two reasons. First, half of the patients with ICA stenosis also had a stenosis of the contralateral ICA. In other words, in at least half the patients with carotid lesions in our study, the stenosis is more likely to be a general marker of atherosclerosis than a specific factor contributing to the ischemic event. Second, angiography of the ICA in people who never suffered from cerebrovascular diseases may show stenosis in 3-23%.21,32 Thus, in a given patient with transient or minor neurologic deficit from a lacunar infarct, a stenosis of the ICA may be asymptomatic.

We conclude that ICA stenosis in patients with lacunar infarcts cannot be predicted from blood pressure, from retinal examination, or from other measures and that such stenosis is rare in the total group of patients with transient hemispheric deficit. Whether our results are also true for noninvasive studies of unselected patients with lacunar infarcts (surgical candidates or not) remains to be determined. For practical purposes, however, only three of every 10 angiograms in patients with lacunar infarcts are abnormal, and the uncertain benefits of carotid endarterectomy in this small proportion must be weighed against the attendant risks of angiography in the entire group. Aspirin seems to be the most appropriate treatment in patients with lacunar infarcts33,34 although there are no controlled trials that have distinguished this type of cerebral ischemia.

Acknowledgments

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