
Arteriographic Comparison of Amaurosis Fugax and Hemispheric Transient Ischaemic Attacks

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SUMMARY The results of carotid angiography were compared in 53 patients with amaurosis fugax and 92 with hemispheric transient ischaemic attacks (TIAs). Evidence of extracranial disease was found in 71% of those with retinal ischaemia and these patients rarely had evidence of intracranial disease (11%) or normal angiograms (15%). By contrast patients with hemispheric symptoms less frequently showed disease of the cervical carotid artery (36%) and more often had intracranial disease (27%) or normal angiograms (35%). It is suggested that amaurosis fugax is usually due to emboli originating in the internal carotid artery but that the pathogenesis of cerebral TIAs in the carotid territory is more heterogeneous. These conclusions have implications for the design and interpretation of both medical and surgical trials.

Stroke Vol 16, No 5, 1985

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Received September 28, 1984; accepted January 22, 1985.

Amaurosis Fugax and transient ischaemic attacks affecting the cerebral hemisphere are both believed to be due to embolism from an atheromatous carotid artery in the majority of cases, and angiography is carried out in the search for operable carotid stenosis. Flow disturbances, cardiac embolism, migraine, and platelet disorders are thought to be occasional alternative causes. Controversy over whether these two common manifestations of carotid vessel disease differ in their pathogenesis prompted this retrospective study.1,2

Material and Methods

The results of carotid angiography were reviewed for patients referred to one of us (JM) for the investigation of amaurosis fugax or transient cerebral ischaemic attacks affecting the cerebral hemisphere. Amaurosis fugax was defined as an episode of monocular blindness of brief duration (all had lasted less than 30 minutes in fact) with no evidence of ocular disease, and with a painless abrupt onset suggestive of retinal ischaemia. Transient hemispheric ischaemic attacks were defined as episodes of neurological deficit attributable to a unilateral hemisphere disturbance lasting less than 24 hours with no residuum. Note was taken whether the symptoms conclusively implicated hemispheric disturbance (e.g. dysphasia) or were conceivably attributable to the vertebro-basilar territory (simple hemisphenomena). Patients with isolated hemianopic attacks were not studied. Those with both amaurosis fugax and hemispheric symptoms were included, but dealt with as a potentially different subgroup for the purposes of analysis. Patients' age, sex and blood pressure were noted as were the results of auscultation of the neck vessels.

The angiographic findings were classified according to whether occlusion, stenosis or simple wall irregularity was demonstrated in either the extracranial, or intracranial part of the carotid tree. Only changes in the carotid artery on the symptomatic side were noted. When abnormalities were detected both intracranially and extracranially the result was classified according to the more severe lesion. For example a patient with minor wall irregularity at the carotid bifurcation together with stenosis in the siphon was listed as having an intracranial lesion.

Results

There were 92 patients with TIAs attributed on clinical grounds to the carotid territory. Nineteen had dysphasia. The remainder (73) had hemisphenomena either motor or sensory, or both. In only one was the face spared.
Thirty-seven patients had one or more attacks of amaurosis fugax but denied any symptoms suggesting cerebral involvement. Eighteen had both amaurosis fugax and cerebral TIAs on the same side. None had simultaneous symptoms of retinal and hemispheric ischaemia.

TIA patients less frequently had cervical bruits (8/92 cf 27/55 Chi square 30.6, p < .001) but no other striking differences emerged (table 1).

The relative extent of extracranial and intracranial abnormality in the different patient groups is shown in table 2. As there was no difference between the group with amaurosis fugax and with amaurosis fugax plus TIAs these were grouped together as indicative of the findings associated with attacks of retinal ischaemia.

It is clear that amaurosis fugax is more commonly associated with extracranial disease and less commonly with intracranial disease or a normal angiographic appearance. The possibility that TIAs characterised by hemiphenomena might be related to vertebo-basilar disease, invalidating the comparison with amaurosis fugax, was considered by looking at the albeit small (n = 19) subgroup with dysphasia. There were no differences in the angiographic findings between the TIA patients whose attacks included dysphasia, and those that did not. The finding of extracranial disease was more frequent in amaurosis fugax than in dysphasic TIAs (71% cf 37%, p < .05) and a normal angiogram was rarer (15% cf 53%, p < .01), as found when the comparison included all TIA patients.

**Discussion**

Cervical bruits and angiographic evidence of major atheromatous changes at the carotid bifurcation were found far more frequently in patients presenting with amaurosis fugax than in those with hemispheric TIAs. Over two-thirds of patients with amaurosis fugax were found to have extracranial atheromatous disease of the internal carotid artery.\(^1,4\) Patients with hemispheric TIAs often had extracranial disease.\(^3\)

These findings are in keeping with earlier suggestions that there are differences in the two groups of cases, though both have atheromatous disease of the carotid tree. Ramirez-Lassepas et al\(^6\) noted that angiograms were normal in only 4% of their cases of amaurosis fugax but in 27% of those with hemispheric TIAs. Slepyan et al\(^7\) similarly found carotid disease more common, and cardiac disease rare in cases of amaurosis fugax when compared with TIA patients but their numbers were small. By contrast, Eisenberg and Mani\(^2\) found comparable angiographic abnormalities in 40 patients with amaurosis fugax and 47 with TIAs. Their cases of amaurosis fugax had a high incidence of congestive cardiac failure and myocardial infarction (60%), and so may not be representative.

Two other studies that have reported no difference between the angiographic appearances of amaurosis fugax and TIAs\(^7,8\) have grouped patients with both retinal and cerebral symptoms with those with cerebral symptoms alone. Our data suggests that these cases are better considered with the amaurosis fugax patients. It would be of interest to rework the data of Lemak and Fields,\(^3\) and of Ross and Morrow\(^4\) in this way.

The present findings suggest that attacks of transient retinal ischaemia are likely to be due to embolism of small size from an origin in the carotid artery, whilst TIAs, often longer in duration\(^9\) may arise more often from emboli in the aorta or heart, which are of larger size, and traverse an open carotid\(^10\) to cause temporary intracranial branch occlusions. Intracranial disease is also a more frequent finding in cases of hemispheric TIAs, suggesting intracranial embolic or flow phenomena account for some such episodes.

These considerations further stress the heterogeneity of TIAs, and the need to consider subgroups when assessing the results of interventional trials whether surgical or medical.

**Acknowledgment**

We are grateful to Ms. B. Laatz for assistance.

**References**


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**Table 1** Details of Patients

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>M/F</th>
<th>Mean age (range) years</th>
<th>BP &gt; 150/90 (%)</th>
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</thead>
<tbody>
<tr>
<td>1. Amaurosis fugax</td>
<td>37</td>
<td>2.7</td>
<td>58.3 (42-71)</td>
<td>46</td>
</tr>
<tr>
<td>Amaurosis fugax + TIA</td>
<td>18</td>
<td>2.6</td>
<td>56.8 (40-64)</td>
<td>55</td>
</tr>
<tr>
<td>2. TIA (hemiphenomena)</td>
<td>73</td>
<td>3.6</td>
<td>56.3 (41-73)</td>
<td>55</td>
</tr>
<tr>
<td>TIA (dysphasia alone)</td>
<td>19</td>
<td>3.7</td>
<td>57.0 (45-64)</td>
<td>37</td>
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</tbody>
</table>

**Table 2** Angiographic Comparison of Patients with Amaurosis Fugax (+ TIA) and Patients with Cerebral TIAs

<table>
<thead>
<tr>
<th>Group</th>
<th>Extra-cranial disease</th>
<th>Intra-cranial disease</th>
<th>Normal</th>
<th>Kink or loop</th>
<th>Total</th>
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<tbody>
<tr>
<td>TIA</td>
<td>33</td>
<td>25</td>
<td>32</td>
<td>2</td>
<td>92</td>
</tr>
<tr>
<td>Amaurosis fugax</td>
<td>39</td>
<td>6</td>
<td>8</td>
<td>2</td>
<td>55</td>
</tr>
<tr>
<td>Total</td>
<td>72</td>
<td>31</td>
<td>40</td>
<td>4</td>
<td>147</td>
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<tr>
<td>Chi square</td>
<td>16.9</td>
<td>5.47</td>
<td>6.5</td>
<td>—</td>
<td>NS</td>
</tr>
<tr>
<td>Significance (p)</td>
<td>&lt;.001</td>
<td>&lt;.02</td>
<td>&lt;.02</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>

Risk of Stroke During Coronary Artery Bypass Graft Surgery in Patients With Internal Carotid Artery Disease Documented by Angiography

ANTHONY J. FURLAN, M.D., AND A. ROMEU CRACIUN, M.D.

SUMMARY: We retrospectively identified 144 patients who underwent coronary artery bypass graft (CABG) surgery in the presence of angiographically documented ≥ 50% internal carotid stenosis or occlusion. Of these, 115 patients had bilateral carotid lesions and received combined operations involving carotid endarterectomy on only one side. The remaining 29 patients, including 11 with bilateral carotid lesions, underwent coronary bypass alone. Nine cerebral infarcts occurred (6%), but only three strokes (2%) were appropriate to the cerebral hemisphere ipsilateral to unoperated carotid stenosis. There was one stroke (3%) among the 29 patients who did not undergo combined procedures. In the group of 115 patients with bilateral carotid disease who received unilateral combined carotid endarterectomy there were 8 perioperative strokes (7%), of which 6 were ipsilateral to the endarterectomy. Asymptomatic unilateral < 90% ICA stenosis or ICA occlusion does not increase stroke risk during CABG surgery.

THE RELATIONSHIP between cerebrovascular disease and stroke complicating coronary artery bypass graft (CABG) surgery is controversial. Neurologists are often consulted regarding the risk of stroke in patients requiring elective myocardial revascularization in the presence of internal carotid artery (ICA) stenosis or occlusion. In an attempt to reduce the risk of perioperative stroke, some centers screen neurologically asymptomatic patients with carotid non-invasive tests to identify candidates for angiography and staged or combined carotid and coronary operations. Presently there is insufficient data to determine the stroke risk posed by ICA stenosis or occlusion in patients undergoing cardiac or other surgical procedures because no study employing cerebral angiography has addressed this issue. In this study, we retrospectively examined the perioperative stroke rate in a group of patients with angiographically proven ICA stenosis or occlusion who subsequently underwent CABG surgery.

Methods

We identified 144 patients who underwent CABG surgery at the Cleveland Clinic from 1973 through 1983 with known, unoperated ≥ 50% stenosis or occlusion of at least one ICA as documented by intra-arterial angiography. Of these 144 patients, 115 were identified by cross-referencing our angiographic and surgical records. Most of these patients had intracranial ICA stenosis or ICA occlusion. In this group 18 patients had unilateral ICA lesions and 11 had bilateral ICA lesions.

Charts were reviewed for evidence of perioperative focal brain infarction. Stroke laterality and potential factors related to brain infarction, such as hypotension and cardiac sources of emboli, were noted.

Results

Cerebral angiography was performed in 73 patients because of an asymptomatic carotid bruit, and in 71 patients because of a history of transient ischemic attacks or stroke. The mean age was 59 years.

The distribution of ICA lesions and their correlation with perioperative infarcts involving the ipsilateral cerebral hemisphere are given in table 1. A total of 9 cerebral infarcts occurred (6%), including 5 minor strokes and 4 moderate to severe strokes. Only 3 strokes (2%) involved a cerebral hemisphere ipsilateral
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Stroke. 1985;16:795-797
doi: 10.1161/01.STR.16.5.795

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