


**Contractile Effects of TxA2/ Ellis et al.**

**Relationship of Transient Ischemic Attacks and Angiographically Demonstrable Lesions of Carotid Artery**

RONALD L. EISENBERG, M.D., WILLIAM R. NEMZEK, M.D., WESLEY S. MOORE, M.D., AND RICHARD L. MANI, M.D.

**Summary** Eighty-eight percent of arteries in patients with amaurosis fugax or hemispheric transient ischemic attacks had angiographically demonstrable lesions at the carotid bifurcation. Eighty-one percent had stenoses or occlusions at the carotid bifurcation; 7 percent had ulcerative lesions without stenoses at this site. Forty-nine percent of arteries in these patients demonstrated ulcerative lesions at the carotid bifurcation.

**Hemispheric Transient** ischemic attacks and amaurosis fugax have been related to ulcerative and occlusive lesions of both the extracranial and intracranial carotid arterial systems. Recent publications suggested that there is a relatively high occurrence of normal findings on arteriography in patients with well-documented transient ischemic attacks. Lemak and Fields, reviewing the data from the cooperative study on extracranial arterial occlusive disease, demonstrated angiographically normal carotid arteries in 22% of patients with amaurosis fugax (with or without hemispheric transient ischemic attacks) and in 43% of patients with hemispheric transient ischemic attacks alone. Since these data were originally collected when the importance of the non-stenotic but ulcerative lesion of the carotid artery had not yet been recognized, we felt that the reported incidence of normal angiographic findings in these patients might be erroneously high.

The purpose of this communication is to review our series of patients with hemispheric transient ischemic attacks and amaurosis fugax with regard to (a) the incidence and types of arteriographically-identifiable lesions of the appropriate carotid artery and, as a corollary, (b) the incidence of lesions potentially correctable by surgical intervention.

**Methods**

From August 1974 to August 1976, 123 patients underwent carotid angiography at the San Francisco Veterans Administration Hospital for evaluation of hemispheric transient ischemic attacks (93) or amaurosis fugax (with or without hemispheric transient ischemic attacks) (30). Fifteen of 123 had bilateral symptoms, bringing the total number of individual carotid arteries for study to 138.
Indications for angiography were one or more episodes of amaurosis fugax or hemispheric transient ischemic attacks within six months. Approximately 25% of patients had single attacks; the balance had multiple attacks prior to angiography. None had experienced cerebral infarction, and therefore there were no fixed neurologic deficits at the time of the evaluation for transient ischemic attacks.

All arteriograms included at least two views (lateral and
Table 1 Angiographically-demonstrable Carotid Lesions

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Normal</th>
<th>Occlusion</th>
<th>Stenosis at Ulceration</th>
<th>Ulcer Alone</th>
<th>Isolated Siphon Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amaurosis fugax</td>
<td>0</td>
<td>9 (26%)</td>
<td>19 (54%)</td>
<td>3 (9%)</td>
<td>4 (11%)</td>
</tr>
<tr>
<td>Hemispheric TIAs</td>
<td>9 (9%)</td>
<td>15 (15%)</td>
<td>70 (67%)</td>
<td>6 (6%)</td>
<td>3 (3%)</td>
</tr>
<tr>
<td>Total</td>
<td>9 (7%)</td>
<td>24 (17%)</td>
<td>89 (64%)</td>
<td>9 (7%)</td>
<td>7 (5%)</td>
</tr>
</tbody>
</table>

Table 2 Ulcerative Lesions at Carotid Bifurcation

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Ulcer with Stenosis</th>
<th>Ulcer without Stenosis</th>
<th>No Ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amaurosis fugax</td>
<td>14 (40%)</td>
<td>3 (9%)</td>
<td>18 (51%)</td>
</tr>
<tr>
<td>Hemispheric TIAs</td>
<td>44 (43%)</td>
<td>6 (6%)</td>
<td>53 (51%)</td>
</tr>
<tr>
<td>Total</td>
<td>58 (42%)</td>
<td>9 (7%)</td>
<td>71 (51%)</td>
</tr>
</tbody>
</table>

Table 3 Grade of Ulceration at Carotid Bifurcation

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amaurosis fugax</td>
<td>18 (51%)</td>
<td>6 (17%)</td>
<td>8 (23%)</td>
</tr>
<tr>
<td>Hemispheric TIAs</td>
<td>53 (51%)</td>
<td>14 (14%)</td>
<td>26 (24%)</td>
</tr>
<tr>
<td>Total</td>
<td>71 (51%)</td>
<td>20 (15%)</td>
<td>33 (24%)</td>
</tr>
</tbody>
</table>

Two major theories have been used to explain the pathogenesis of hemispheric transient ischemic attacks in patients with atherosclerotic lesions of the carotid circulation. One theory, based upon hemodynamics, states that a high-grade stenosis of the carotid artery produces symptoms by an intermittent pressure drop with resulting reduction in critical perfusion of the hemisphere. The second theory states that emboli originating from the surface of an atherosclerotic ulcerative plaque and consisting of platelet aggregates, small bits of thrombi, or degenerative atheromatous material, break off from these lesions to embolize to the brain.

In our series, 89% of arteries in patients with amaurosis fugax and 88% of arteries in patients with hemispheric transient ischemic attacks had angiographically-demonstrable lesions (ulceration, stenosis, occlusion) of the carotid bifurcation disease had a marked ulceration in the internal carotid artery. Nine patients (9%) had normal carotid arteriograms. In these patients, no proximal brachiocephalic lesions were identified.

Table 2 summarizes the incidence of arteriographically-identifiable ulcerative lesions at the carotid bifurcation. Sixty-seven (49%) of all the carotid arteries examined had ulcerative lesions; 58 (42%) had combined ulcerative and stenotic lesions; and 9 (7%) had ulceration without stenosis. Seventy-one arteries (51%) showed no arteriographic evidence of ulceration at the carotid bifurcation.

Table 3 summarizes the extent of ulceration seen at the carotid bifurcation, according to our classification. Overall, 20 (15%) arteries demonstrated Grade A lesions, 33 (24%) had Grade B lesions, and 14 (10%) had Grade C lesions. There was no significant difference in the incidence and types of ulcerations at the carotid bifurcation between patients with amaurosis fugax and those with hemispheric transient ischemic attacks.

Discussion

Two major theories have been used to explain the pathogenesis of hemispheric transient ischemic attacks in patients with atherosclerotic lesions of the carotid circulation. One theory, based upon hemodynamics, states that a high-grade stenosis of the carotid artery produces symptoms by an intermittent pressure drop with resulting reduction in critical perfusion of the hemisphere. The second theory states that emboli originating from the surface of an atherosclerotic ulcerative plaque and consisting of platelet aggregates, small bits of thrombi, or degenerative atheromatous material, break off from these lesions to embolize to the brain.

In our series, 89% of arteries in patients with amaurosis fugax and 88% of arteries in patients with hemispheric transient ischemic attacks had angiographically-demonstrable lesions (ulceration, stenosis, occlusion) of the carotid bifurcation.
cation. In arteries of patients with amaurosis fugax, 80% had occlusive lesions and 9% had non-stenotic, ulcerative lesions at the carotid bifurcation. In arteries of patients with hemispheric transient ischemic attacks, 82% had occlusive lesions and 6% had non-stenotic, ulcerative lesions at this site.

It is of interest that 49% of our patients had arteriographically identifiable ulcerative lesions, either stenotic or non-stenotic. This relatively high figure represents an increased awareness not only of the importance of these lesions related to the patients’ symptoms (transient ischemic attacks), but also reflects past experience in which surgical findings have confirmed small ulcers previously thought to have been unimportant.

The importance of ulcerative lesions, even without stenosis, in the genesis of hemispheric transient ischemic attacks, is supported by the work of Moore and Hall7 who describe a series of 49 patients with hemispheric transient attacks and arteriographically-identifiable, non-stenotic, ulcerative lesions of the carotid bifurcation. These patients promptly became asymptomatic following surgical removal of these lesions.

Toole and colleagues8 demonstrated an incidence of 71% abnormality of the extracranial carotid artery in patients having hemispheric transient ischemic attacks. This would correspond to the figures presented in the current study. However, Woolsey and Henbrook9 demonstrated that only 50% of their patients had positive angiographic findings at the carotid bifurcation that they could associate with symptoms of hemispheric transient cerebral ischemia.

Lemak and Fields1 reported that 43% of their patients with hemispheric transient ischemic attacks and 22% of patients with amaurosis fugax had normal angiography as documented in the large cooperative study on extracranial arterial occlusive disease. Ramirez-Lassepas and colleagues10 indicated that 27% of their patients with hemispheric transient ischemic attacks and 4% of patients with amaurosis fugax had normal arteriograms. Our results (9% of patients with hemispheric transient ischemic attacks and 0% of patients with amaurosis fugax having normal arteriograms) are in marked contrast to these two groups. In a personal communication, Dr. Fields has pointed out that the data reported in his study were gathered at a time when the quality of angiography, as well as the expertise and interpretation, was still in the developing stage. His personal observation is that, presently, approximately 90% of patients with hemispheric transient ischemic attacks will have identifiable lesions in either the extracranial or intracranial carotid distribution and that 100% of patients with amaurosis fugax should have an identifiable lesion, which would correspond closely to the figures in our present study.

Based upon our current data, patients with a clear-cut diagnosis of hemispheric transient ischemic attacks or amaurosis fugax should have a high associated incidence of appropriate lesions of the carotid distribution, which should be demonstrable with current high-quality angiographic techniques.

The management of these lesions, once identified, will vary depending upon the orientation and preference of the individual physician or institution. Data show that lesions of the extracranial carotid bifurcation can be removed surgically with a good result. Removal of carotid bifurcation plaques will result in an abrupt cessation of hemispheric attacks or amaurosis fugax and will prevent, in the majority of cases, a subsequent stroke in that distribution. In that context, 71% of the arteries examined had lesions appropriate to surgical repair located at the carotid bifurcation. The remaining 29% had lesions that were not amenable to carotid bifurcation endarterectomy and included occlusion (17%), disease localized to the carotid siphon (5%), and a 7% incidence of no identifiable lesion. If one wishes to consider the patients with occlusion of the internal carotid artery who are having symptoms to be candidates for extracranial to intracranial revascularization, then 88% of the arteries examined in this series would be amenable to surgical reconstruction, making the angiographic documentation of their disease most worthwhile.

References

Relationship of transient ischemic attacks and angiographically demonstrable lesions of carotid artery.
R L Eisenberg, W R Nemzek, W S Moore and R L Mani

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