allowed demonstration of marked microcirculatory changes occurring during vasospasm which cannot be detected by macroscopic techniques.

References

Transient Ischemic Attacks and External Carotid Artery
A Retrospective Study of 23 Patients with an Occlusion of the Internal Carotid Artery

JULIEN BOGOUSSLAVSKY, M.D., FRANCO REGLI, M.D., JEAN-PIERRE HUNGERBÖHLER, M.D., AND RICHARD CHRZANOWSKI, M.D.

SUMMARY Twenty-three patients with occlusion of an internal carotid artery have been followed 5 to 60 months after angiography. None had a later permanent stroke. Eight had delayed TIAs in the occluded internal carotid area, never in another area. In these TIAs the role of the homolateral external carotid is emphasized, because in the 8 cases this artery was the main collateral to the occluded internal carotid, and angiography had shown atherosomatous stenosis of homolateral external/common carotid arteries or an irregular stump at the site of the occlusion. Hemodynamic and embolic mechanisms are discussed, especially the latter, because of the absence of severe stenosis and evidence of embolicgenic plaques.

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Material and Methods
A retrospective study was made of 23 patients with an angiographically proved occlusion of an internal carotid artery. Five to 60 months after angiography (mean: 27 months) patients were asked about new transient ischemic attacks (TIAs). Angiograms were re-studied (archography and/or selective carotid arteriography). In 11 patients a contralateral carotid arteriogram was made and in 18 a Doppler-carotid ultrasonogram was taken. Eighteen men and 5 women aged 39 to 78 years (mean: 61.5) were studied. Fourteen had a left internal carotid occlusion and 10 a right internal carotid occlusion. In 11 patients hospitalization occurred after a minor stroke, and 2 after TIAs. All these ischemic events were in the area of the brain supplied by the occluded internal carotid. Thirteen patients had previously had TIAs in this area, and...
none in any other. In 17 patients anticoagulant or platelet antiaggregant therapy had been prescribed.

Results

None of the 23 patients studied had a permanent stroke during the follow up, but TIAs occurred in 8 (from 4 per month to 1 per 10 months, mean: 1 every 6 weeks). The TIAs never occurred following angiography and were not related to orthostatic factors. The duration of TIAs varied from 1 minute to 8–10 hours but were mostly 10 minutes. The clinical presentations were sensory disturbances of a limb in 4, limb paresis in 2, dysphasia in 1, and amaurosis fugax in 3. In 2 patients, amaurosis fugax occurred alternately with sensory disturbances of a limb. The angiographic studies showed a stenosis or atheromatous irregularities in the main trunk or at the origin of the homolateral external carotid artery in 11 patients and in 13 stenosis or atheromatous irregularities in the homolateral common carotid or brachiocephalic arteries. The most important stenoses involved 50% of the lumen. In 7 patients an irregular and ulcerated "stump" was found at the proximal extremity of the internal carotid thrombus. No intracranial vascular lesions were seen.

We divided our patients into 2 groups: those with no TIA after internal carotid occlusion (Group II) and those with TIAs occurring after internal carotid occlusion (Group I) (table 1). In Group I, 8 patients had an atheromatous stenosis in the homolateral external/common carotid artery or an ulcerated "stump" in the occluded internal carotid artery. All these showed good collateral supply of the area from the occluded internal carotid artery through the anastomotic channels via the external carotid artery, and reversal of ophthalmic artery flow on ultrasonography. The appearance of the narrowed external or common carotid arteries is shown in figures 1, 2, 3.

In Group II, 7/15 patients (Group II A) had stenosis of the external/common carotid arteries or a "stump." The external carotid artery14, 15, 20 was not an embolic channel in 4 of the 7 patients because intracranial supply through the external carotid artery was absent angiographically, with no reversal of ophthalmic flow shown on ultrasonography. Among the 7 patients in Group II there were 3 in whom collateral supply via the external carotid artery existed but in whom TIAs did not occur. In Group II B, no stenosis in the external/common carotid artery was demonstrated on angiography and these patients did not have further TIAs. There were no important differences in age (I: 64, II: 60) sex (I: 7M/1F; II: 11M/3F) or follow up (I: 21 months, II: 27 months) between Groups I and II. There was no significant difference in medical treatment between Groups I and II. During the period of follow up treatments had been frequently intermittently followed. In Group I, 2 patients had been on anticoagulant therapy and 2 on platelet antiaggregant therapy. In Group II, anticoagulant medication had been prescribed for 2 and platelet antiaggregant therapy for 9.

### Table 1: Classification of the Results of Angiography and Ultrasonography According to the Presence (Group I) or Absence (Group II) of TIAs

<table>
<thead>
<tr>
<th>Cases</th>
<th>Atheromatous stenosis of homolateral external carotid</th>
<th>Atheromatous stenosis of homolateral common carotid</th>
<th>Irregular &quot;stump&quot; on the occluded internal carotid</th>
<th>Intracranial collateral circulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>++</td>
<td>+</td>
<td>—</td>
<td>e</td>
</tr>
<tr>
<td>1</td>
<td>++</td>
<td>—</td>
<td>—</td>
<td>e</td>
</tr>
<tr>
<td>2</td>
<td>—</td>
<td>—</td>
<td>s</td>
<td>e</td>
</tr>
<tr>
<td>3</td>
<td>++</td>
<td>—</td>
<td>s</td>
<td>e</td>
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<tr>
<td>4</td>
<td>+</td>
<td>—</td>
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<td>e</td>
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<tr>
<td>5</td>
<td>++</td>
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<td>e</td>
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<td>6</td>
<td>+</td>
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<td>e/i</td>
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<td>7</td>
<td>+</td>
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<tr>
<td>8</td>
<td>—</td>
<td>—</td>
<td>s</td>
<td>e</td>
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<tr>
<td>Group II</td>
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<td>A B</td>
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<td>9</td>
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<tr>
<td>10</td>
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<td>e</td>
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<tr>
<td>11</td>
<td>+</td>
<td>+</td>
<td>—</td>
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</tr>
<tr>
<td>12</td>
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<td>—</td>
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<td>14</td>
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<td>s</td>
<td>i</td>
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<td>15</td>
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<td>—</td>
<td>—</td>
<td>i</td>
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<td>16</td>
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<td>17</td>
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<td>20</td>
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<td>s</td>
<td>i</td>
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<td>21</td>
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<td>s</td>
<td>i</td>
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<tr>
<td>22</td>
<td>+</td>
<td>+</td>
<td>—</td>
<td>e</td>
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<tr>
<td>23</td>
<td>+</td>
<td>—</td>
<td>s</td>
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</tbody>
</table>

+ e/i equality  
++ important (50% and more)  
+ absent  
+ irregular stump  
e external carotid channel predominates  
i contralateral internal carotid channel predominates

Discussion

These results suggest that the presence of atheromatous stenosis of the external common carotid homolateral to an occluded internal carotid artery, or the presence of an irregular "stump" at this occlusion, is an important factor in the further occurrence of TIAs, when there is a good intracranial supply through the branches of the external carotid artery. Atheromatous emboli could be carried to the cerebroretinal level through these collaterals. These observations support the conclusions that TIAs do not always stop after the corresponding artery is occluded. It is of interest that in 3 patients (Nos. 1, 4, 6) TIAs occurred only after the corresponding internal carotid artery had become occluded.

In table 2, 20 of 23 patients studied support the role
played by the external carotid artery in the occurrence of TIA.

Patient 6 is of interest because isolated amaurosis fugax occurred without episodes of hemispheric ischemia. The angiograms showed flow from the good ophthalmic artery through the external carotid artery but little hemispheric supply through this artery. Hemispheric flow came mainly from the anterior communicating artery. Emboli coming from atheromatous plaques in the stump or external carotid artery did not go beyond the ophthalmic artery because of the predominance of the contralateral blood flow to the cerebral hemisphere. This phenomenon has been mentioned in the study of Countee et al.9

The differences in the occurrence of TIAs between our 2 groups suggest the importance of the external carotid artery in the genesis of further ischemic dysfunction when this artery is a major collateral. Two mechanisms are likely: 1) emboli originating from the stump or ulcerated plaques located in the external carotid artery or 2) more proximal hemodynamic modifications due to severe stenosis of the external carotid. The latter was not observed in these patients and the embolic hypothesis is most likely. Hemodynamic and embolic mechanisms may be combined, because in a badly perfused area emboli are more likely to be symptomatic. Cerebral blood flow changes with carotid artery stenosis are not frequently noted.4-7

In the patients reported by Furlan et al.,8 with isolated occlusion of an internal carotid artery, there was no amaurosis fugax on exposure to light. The authors suggest the cause was an intermittent deficit in the ophthalmic supply through external carotid col-

FIGURE 1. Some aspects of irregular narrowing of the external or common carotid arteries associated with an occlusion of the internal carotid artery (1: common carotid artery. 2: occluded internal carotid artery. 3: external carotid artery).

FIGURE 2. Left carotid angiogram of Patient 5 showing occlusion of internal carotid artery and stenosis of the trunk of external carotid artery.

FIGURE 3. Left carotid angiogram of Patient 6 showing occlusion of internal carotid artery with an irregular stump and stenosis of the trunk of external carotid artery.
lateralis, when the patients’ metabolic needs were increased because of sudden bright light.

Emboli from the tail of the stump have been reported in isolated episodes.4,6,12 Emboli from the contralateral internal carotid artery through the anterior communicating artery is improbable. In our patients who had hemispheric TIsAs the collateral flow to the ischemic area via contralateral channels was less important than through the homolateral external carotid.

Early reports14,16 mentioning the role of the external carotid in the pathogenesis of some TIsAs are rare. Atherosclerosis of the external carotid probably develops simultaneously with that of the internal carotid.1,14,16 The role of the external carotid in retinal emboli has been suggested after the discovery that emboli occur in patients with an occlusion of the internal carotid.3,4,17 Amaurosis fugax has occurred following injection of anesthetics near the alveolar branches of the external carotid artery,18,20 further evidence of a collateral role of the external carotid artery.

In summary, after the occlusion of an internal carotid artery the occurrence of TIsAs in the same vascular area must point to the possibility of pathology of the external carotid artery. This is of special importance during the investigation preceding an extra-intracranial bypass, in order to avoid facilitating intracranial embolic phenomena or to perform an unnecessary operation in patients where an external carotid artery endarterectomy would be sufficient to increase CBF.

References


Table 2: Cases in Which Evolution is Explained by the Role of External Carotid as an Intracranial Collateral

<table>
<thead>
<tr>
<th>Angiography</th>
<th>TIA's</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atheromatous stenosis of external/common carotid with or without ulcerated stump, with or without good intracranial supply through external carotid</td>
<td>present</td>
<td>1,2,4,5,6,7</td>
</tr>
<tr>
<td>Isolated ulcerated stump with good intracranial supply through external carotid</td>
<td>present</td>
<td>3,8</td>
</tr>
<tr>
<td>Atheromatous stenosis of external/common carotid, with or without ulcerated stump, without intracranial supply through external carotid</td>
<td>absent</td>
<td>14,18,21</td>
</tr>
<tr>
<td>Isolated ulcerated stump without intracranial supply through external carotid</td>
<td>absent</td>
<td>20</td>
</tr>
<tr>
<td>Absence of visible atheromatous lesions of external/common carotid, without ulcerated stump, with or without good intracranial supply through external carotid</td>
<td>absent</td>
<td>9,10,11,12,13</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15,16,19,23</td>
</tr>
<tr>
<td></td>
<td>Total = 20</td>
<td></td>
</tr>
</tbody>
</table>

Total = 20
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J Bogousslavsky, F Regli, J P Hungerbühler and R Chrzanowski

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