Clinical Indicators of Extracranial Carotid Artery Disease in Patients With Transient Symptoms

BY MANUEL RAMIREZ-LASSEPAS, M.D.,* 
BURTON A. SANDOK, M.D., 
AND ROBERT C. BURTON, M.D.

Abstract: Clinical Indicators of Extracranial Carotid Artery Disease in Patients With Transient Symptoms

The clinical findings in 64 patients with transient symptoms of unilateral carotid system ischemic disease (amaurosis fugax or transient focal cerebral ischemic attacks or both) were reviewed in an effort to determine the value of the neurovascular examination in predicting the presence and extent of roentgenographically demonstrated ipsilateral extracranial internal carotid artery disease. Amaurosis fugax seems to be a highly specific indicator of disease, being associated with 4% of normal vessels as compared to 27% for patients with transient focal cerebral ischemic attacks alone. In patients with transient symptoms, the incidence of a normal, ipsilateral carotid artery was 36% in the absence of any positive neurovascular findings, 15% in the presence of an ipsilateral carotid bruit, 6% in the presence of ipsilateral retinal embolic events, 5% in the presence of ipsilateral reduction in superficial temporal or carotid (or both) pulse, 4% in the presence of an ipsilateral reduction in retinal artery pressure, and 3% when more than one of these findings were noted. The neurovascular examination appears to be a useful adjunct in detecting the presence or absence of ipsilateral carotid disease. However, no combination of symptoms or signs would, in all cases, allow one to accurately determine whether a vessel was abnormal, stenotic, or occluded.

Additional Key Words
neurovascular examination
amaurosis fugax
carotid bruit
retinal artery pressure
retinal emboli

In recent years, increasing emphasis has been placed on the diagnosis and treatment of extracranial carotid atheromatous disease. Although angiography is the most definitive diagnostic procedure, it is not without significant risk,¹ and not all patients with suspected cerebrovascular disease should be subjected to this study. Therefore, as clinicians, we are frequently asked to determine the presence and nature of a potentially responsible lesion in the extracranial vessels on the basis of various historic and clinical findings. This study was conducted to review our observations in the clinical evaluation of extracranial carotid disease in patients with transient symptoms.

Methods
The clinical records of all patients who underwent cerebral angiography in the 25-month period between January, 1970, and February, 1972, were studied (1,080 patients). From this group, we selected patients

From the Department of Neurology and the Clinical Cerebrovascular Research Center, Mayo Clinic and Mayo Foundation, Rochester, Minnesota 55901.

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*Former Research Assistant, Clinical Cerebrovascular Research Center. Current appointment: Department of Neurology, University of Minnesota, Minneapolis, and St. Paul-Ramsey Hospital, St. Paul, Minnesota.

Reprint requests: Clinical Cerebrovascular Research Center, Mayo Clinic, Rochester, Minnesota 55901.
who were more than 40 years old in whom angiography was performed for suspected cerebrovascular disease. We excluded all patients in whom the final diagnosis was not carotid system ischemic disease. We included only patients who had transient symptoms of unilateral carotid system disease (that is, transient focal cerebral ischemic attacks or amaurosis fugax or both). Excluded were patients in whom the precise nature of the transient symptoms or the precise source (carotid or the vertebrobasilar system) was doubtful. In addition, all patients who had histories of a focal neurological deficit that persisted more than 24 hours or who had abnormalities in the general neurological examination were eliminated from the study. The study group consisted of 64 patients who had transient symptoms of unilateral carotid system disease.

The angiograms and protocols of these patients were reviewed to determine the relationships among the clinical history, the neurovascular examination, and the nature of the ipsilateral extracranial disease demonstrated by angiography.

An attempt was made to grade the roentgenographical evidence of the ipsilateral carotid artery disease in accordance with the following classification: (1) normal, (2) stenosis of less than 50% without demonstrable ulcer, (3) stenosis of less than 50% with ulcer, (4) stenosis of greater than 50% without ulcer, (5) stenosis of greater than 50% with ulcer, and (6) occlusion. Uniform agreement could be reached only for categories 1 (normal) and 6 (occlusion). In many instances, definite ulceration was evident at endarterectomy but was not demonstrable on roentgenogram. Therefore we used a simpler classification (normal, stenotic, and occluded) and concerned ourselves primarily with vessels that were normal or occluded.

**Results**

Angiographical study in these 64 patients revealed that 17% (11 patients) had a normal ipsilateral carotid artery, 69% (44 patients) had stenosis, and 14% (nine patients) an occluded artery. The condition in the symptomatic side bore no detectable relationship to the condition in the contralateral or "nonsymptomatic" vessel; in the contralateral vessels, 34% were normal, 59% were stenotic, and 7% were occluded.

**CLINICAL HISTORY**

Although all patients had transient symptoms of unilateral carotid system disease, patients with amaurosis fugax as part of their symptom complex differed significantly from patients who had transient focal cerebral ischemic attacks alone. The incidence of a normal ipsilateral carotid artery was 4% (1 of 27) for patients with amaurosis fugax (with or without associated transient focal cerebral ischemic attacks), but was 27% (10 of 37) in patients with transient focal cerebral ischemic attacks alone (table 1). Furthermore, the incidence of roentgenographically severe stenosis was higher with amaurosis fugax than with transient ischemic attacks alone, and the incidence of occlusion was 22% (6 of 27) for the patients with amaurosis fugax compared to 8% (3 of 37) for patients with transient ischemic attacks alone.

**NEUROVASCULAR EXAMINATION**

The following aspects of the neurovascular examination were specifically studied: (1) presence of an ipsilateral carotid bruit, (2) ipsilateral reduction in retinal artery pressure, (3) presence of embolic events (that is, cholesterol, platelet, fibrin emboli, retinal infarction, or retinal arteriole occlusion) in the ipsilateral retina, and (4) ipsilateral reduction in palpable carotid or superficial temporal pulsations.

Results of the neurovascular examination were normal for 14 patients (22% of the study group) (table 2). Although, in the absence of findings, the incidence of a normal ipsilateral vessel was 36%, nine patients with normal findings on neurovascular

**TABLE 1**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Ipsilateral carotid artery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Amaurosis fugax</td>
<td>1</td>
</tr>
<tr>
<td>(with or without TIA)</td>
<td></td>
</tr>
<tr>
<td>Transient focal cerebral ischemic attacks alone</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
</tr>
</tbody>
</table>

**TABLE 2**

<table>
<thead>
<tr>
<th>Findings</th>
<th>Patients No.</th>
<th>%</th>
<th>Roentgenographical evidence (ipsilateral carotid artery)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. %</td>
<td></td>
<td>Normal No. % Stenotic Occluded</td>
</tr>
<tr>
<td>None</td>
<td>14</td>
<td>22</td>
<td>5</td>
</tr>
<tr>
<td>Positive</td>
<td>50</td>
<td>78</td>
<td></td>
</tr>
<tr>
<td>Bruit</td>
<td>9</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Alone</td>
<td>41</td>
<td>64</td>
<td>6</td>
</tr>
<tr>
<td>Entire group</td>
<td>28</td>
<td>43</td>
<td>1</td>
</tr>
<tr>
<td>Pulse decreased</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Alone</td>
<td>19</td>
<td>30</td>
<td>1</td>
</tr>
<tr>
<td>Entire group</td>
<td>39</td>
<td>60</td>
<td>1</td>
</tr>
<tr>
<td>Retinal emboli</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Alone</td>
<td>17</td>
<td>26</td>
<td>1</td>
</tr>
<tr>
<td>Entire group</td>
<td>39</td>
<td>60</td>
<td>1</td>
</tr>
<tr>
<td>Combinations</td>
<td>64</td>
<td>100</td>
<td>11</td>
</tr>
</tbody>
</table>

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examination had abnormalities visualized angiographically, and two of these had occluded arteries. Fifty patients (78%) had one or more of these abnormalities noted on neurovascular examination.

Forty-one of the 64 patients (64%) had an ipsilateral carotid bruit (table 2), and nine of these had a bruit as their only abnormality; five of the nine patients had normal vessels on the angiogram. The significance of a carotid bruit seems much greater when found in combination with other neurovascular abnormalities. Four patients with occluded internal carotid arteries had ipsilateral carotid bruits noted on examination. In all four patients, the external carotid artery was angiographically patent, and we assume that to be the source of the bruit in these four.

Twenty-eight patients had ipsilateral reduction in retinal artery pressure of greater than 10% when compared to the contralateral eye. Only one patient in this group had an angiographically normal ipsilateral carotid artery, and the other 27 patients had severe stenosis or occlusion or both.

Nineteen patients had ipsilateral reduction in palpable carotid or superficial temporal pulsations. Although caution must be exercised in performing this aspect of the neurovascular examination, this proved to be a finding which, when present, was associated with a significant incidence of carotid disease. Only one patient with this finding had an angiographically normal vessel.

Seventeen patients had embolic events in the ipsilateral retina. One patient had a normal vessel, and 16 patients had carotid arterial disease. In spite of the limitations in judging the presence of an ulcerated plaque, it was our impression that retinal emboli were associated with a high incidence of angiographically demonstrated ulcerated lesions.

Thirty-nine of the patients (60%) had one or more of these abnormalities on neurovascular examination; only one patient had an angiographically normal vessel.

In summary, in our patients with transient symptoms, the incidence of an angiographically normal ipsilateral carotid artery was 36% (5 of 14) in the absence of positive findings on neurovascular examination, 15% (6 of 41) when an ipsilateral carotid bruit was present, 6% (1 of 17) in the presence of ipsilateral retinal embolic events, 5% (1 of 19) in the presence of an ipsilateral reduction in superficial temporal or carotid artery pulsation, 4% (1 of 28) in the presence of an ipsilateral reduction in retinal artery pressure, and 3% (1 of 39) when more than one of the above findings were noted (table 2). In these patients, no single symptom, sign, or combination enabled us to predict with accuracy the nature of the defect seen on the angiogram.

Discussion

The patient with unilateral transient ischemic attacks of the carotid system and an ipsilateral extracranial carotid stenosis appears to be a suitable candidate for endarterectomy. It was evident that, as reported in other studies, the neurovascular signs utilized were helpful in making an inference about the presence of disease in the ipsilateral vessel, but we were unable to predict in any given situation whether the responsible vessel was normal, stenotic, or occluded, since each possibility was noted to be present for any given set of parameters. One patient with unilateral transient ischemic attacks of the carotid system had a carotid bifurcation bruit, cholesterol emboli in the ipsilateral retina, and a decreased ipsilateral retinal artery pressure, yet had bilaterally normal carotid and intracranial vessels. Therefore, any decision made and any treatment planned on the basis of the clinical evaluation alone are likely to be associated with significant error.

Arteriography remains the definitive diagnostic procedure. If the purpose of performing the study is to confirm the presence of ipsilateral carotid disease regardless of its nature, then the clinical findings alone may be sufficient to implicate associated disease. If, however, one wishes to determine the presence or absence of a potentially surgically correctable lesion, this cannot be accomplished without arteriography.

The diagnosis of transient ischemic attacks is made solely on the basis of a careful historic inquiry, and there is no sign evident on neurological examination that can substantiate such a diagnosis. Therefore, we believe that all patients with historically documented transient unilateral ischemic disease of the carotid system, regardless of the signs present on the neurovascular examination, should be subjected to the risk of arteriography, providing (1) their general medical and neurological condition indicates they are suitable candidates for carotid endarterectomy, and (2) they are willing to proceed with endarterectomy should a correctable lesion be detected.

Conclusions

From our study of a highly selected group of patients, we have made certain observations.

1. Amaurosis fugax is a highly specific indicator of extracranial carotid artery disease, being associated with a normal ipsilateral vessel in only 4% of our patients.
2. The neurovascular examination is a useful aid in predicting the presence of ipsilateral carotid disease, although even in the absence of findings on examination, our patients were noted to have a 64% incidence of diseased carotid arteries.
3. A carotid bruit is a common finding in patients with transient ischemic episodes. A bruit, however, is not an invariable sign of ipsilateral carotid artery disease and may be noted in instances of both normal and occluded internal carotid arteries. We believe that the significance of a carotid bruit in identifying underlying disease is greatly enhanced when it is found with other neurovascular abnormalities.

4. Reduction in ipsilateral carotid or superficial temporal pulse, reduced ipsilateral retinal artery pressure, and the presence of ipsilateral retinal embolic events, although encountered less frequently, are associated with a low incidence of normal vessels and may indicate ipsilateral carotid artery disease.

5. No single abnormality or combination of abnormalities enabled one to accurately predict the angiographical findings. Although the presence of isolated or combined abnormalities was associated with a higher incidence of diseased vessels, we could not, by any combination of symptoms or signs, distinguish between a severely stenotic vessel and one that was occluded.

References


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