Clinical Phenomena and Their Correlation to Angiographic Findings in Cerebrovascular Disease

DAVID J. THURMAN, M.D. AND CLARK H. MILLIKAN, M.D.

SUMMARY Seventy-one patients who had cerebral angiography because of clinical evidence of focal ischemic cerebrovascular disease were studied. Seventy-seven per cent of these patients had an arterial lesion in the distribution appropriate to the symptoms. However, the degree of dissemination of the atherosclerotic lesions, lesion morphology and severity of stenosis could not be predicted from the clinical manifestations. The presence of atherosclerotic cerebrovascular disease can usually be predicted by analysis of the symptoms and signs, but the nature and extent of the lesions can only be determined using cerebral angiography.

Stroke, Vol 12, No 1, 1981

FACED with a number of options for treatment, the clinician has important problems in the evaluation of each patient with acute cerebral ischemic disease. Three questions repeatedly arise: 1) Is there a relationship between the signs of focal ischemia and the sites of arterial lesions demonstrated angiographically? 2) Is there a relationship between the clinical severity of the ischemia and the character or morphology of the arterial lesions? (Is the severity of the patient’s symptoms and signs of use in predicting which of the arterial lesions are ulcerated, stenotic or occluded?) 3) Is angiography necessary to determine the nature of the vascular disease process and the site or sites of arterial pathology? Treatment may vary depending on answers to the last question.

Methods

The records of all patients having angiography because of clinical evidence of cerebrovascular disease during 30 months between 1977 and 1979 were reviewed. All patients had been admitted to the neurology or neurosurgery service. Patients were excluded who had a cardiac source for emboli, classic migraine or disorders causing hypercoagulability such as polycythemia. Criteria for the diagnosis of transient ischemic attacks (TIAs), reversible ischemic neurological deficit (RIND), completed stroke and neurological deficit (RIND), completed stroke and tent neurological deficit was divided into 4 functional status categories as defined in “A Classification and Outline of Cerebrovascular Disease II.” Category I refers to no significant impairment; Categories II, III and IV refer to mild, moderate, and severe impairments respectively.

Results

Of the 71 patients, 44 were male and 27 female. The mean age was 60.2 with a range of 33 to 78 years. Thirty patients had had TIAs or RINDS; 41 had had a cerebral infarction, with or without associated TIAs. The patients with cerebral infarction had had a persistent neurological deficit, a positive CT head scan or both. Angiograms showed that 82% of these patients had some evidence of cervical or cerebral arterial disease; the remaining 18% had no visible arterial lesions or minimal or equivocal lesions. Table 1 demonstrates the correlation between focal symptoms and the number of lesions which were identified at sites appropriate to these symptoms (e.g., in patients with hemiparesis the number where contralateral arterial lesions were identified). Seventy-seven percent of the patients had a definite lesion in the arterial system appropriate to their symptoms. In addition, an attempt was made to find a relationship between the categories of clinical presentation and the distribution of the lesions seen on angiography, that is, the sites of lesions and the degree of dissemination of the lesions. Tables 2 a and b show there is a wide scatter in the distribution of lesions seen with most clinical categories. No definite correlation can be made between the number and severity of symptoms and the dissemination of the vascular lesions identified. The numbers are too small for statistical analysis.

The relationship between categories of clinical presentation and the morphology of the angiographic
lesions were studied (table 3). The results were widely scattered and no significant correlation could be found between the morphology of the lesions and the clinical presentations.

The relationship between the severity of the neurological deficit in patients with cerebral infarction and the distribution and morphology of the angiographic arterial lesions was studied. Patients with stable neurological deficits of less than a week's duration were not included, nor were patients with exclusively retinal phenomena. Patients with severe neurological deficits are under-represented in this study, as they are usually not considered candidates for angiography or surgery. There was no relationship between the severity of the stroke and the distribution or morphology of the arterial lesions seen on angiograms (tables 4 and 5).

### Table 1. Number of Lesions at Site Appropriate to Symptoms

<table>
<thead>
<tr>
<th>TIAs (1)</th>
<th>TIA (2)</th>
<th>TIAs (≥ 3)</th>
<th>TIA and RIND</th>
<th>Cerebral Infarct without TIAs</th>
<th>Cerebral Infarct with TIAs</th>
<th>Multiple Cerebral Infarcts</th>
</tr>
</thead>
<tbody>
<tr>
<td>No lesion at appropriate site</td>
<td>Equivocal lesion at appropriate site</td>
<td>Definite lesion at appropriate site</td>
<td>No lesion at appropriate site</td>
<td>Equivocal lesion at appropriate site</td>
<td>Definite lesion at appropriate site</td>
<td>No lesion at appropriate site</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>0</td>
<td>1</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td>10</td>
<td>3</td>
<td>2</td>
<td>16</td>
<td>6</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>6</td>
<td>1</td>
<td>0</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>TOTALS</td>
<td>6 (8.4%)</td>
<td>10 (14.1%)</td>
<td>55 (77.5%)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 2a. Transient Ischemic Attacks, RINDs and the Distribution of Lesions Seen on Angiography

<table>
<thead>
<tr>
<th></th>
<th>Normal angiogram</th>
<th>Equivocal lesion</th>
<th>Single cervical carotid lesion</th>
<th>Single intraocular carotid system lesion</th>
<th>Vertebrobasilar system lesion only</th>
<th>Multiple or bilateral carotid system lesions</th>
<th>Multiple carotid and V-B system lesions</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single TIA</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Two TIAs</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Three or more TIAs</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>6</td>
<td>6</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>RINDs with or without TIAs</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>TOTALS</td>
<td>1</td>
<td>6</td>
<td>2</td>
<td>0</td>
<td>11</td>
<td>6</td>
<td>30</td>
<td>30</td>
</tr>
</tbody>
</table>

### Table 2b. Cerebral Infarction and the Distribution of Lesions Seen on Angiography

<table>
<thead>
<tr>
<th></th>
<th>Normal angiogram</th>
<th>Equivocal lesion</th>
<th>Single cervical carotid lesion</th>
<th>Single intraocular carotid system lesion</th>
<th>Vertebrobasilar system lesion only</th>
<th>Multiple or bilateral carotid system lesions</th>
<th>Multiple carotid and V-B system lesions</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single CI without prior TIA</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>7</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>Single CI with prior TIA</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>6</td>
<td>9</td>
<td>21</td>
</tr>
<tr>
<td>Multiple CI</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>TOTALS</td>
<td>2</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>14</td>
<td>13</td>
<td>41</td>
</tr>
</tbody>
</table>

### Table 3. Cerebral Infarction, Transient Ischemic Attacks and the Morphology of Lesions Seen on Angiography

<table>
<thead>
<tr>
<th></th>
<th>No Lesion</th>
<th>Equivocal Lesion</th>
<th>Mild Stenosis</th>
<th>Moderate Stenosis</th>
<th>Marked Stenosis</th>
<th>Mild Stenosis</th>
<th>Moderate Stenosis</th>
<th>Marked Stenosis</th>
<th>Occlusion</th>
<th>FMD, &quot;kinking&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single TIA</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Two TIAs</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Three + TIAs</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>RINDS with or without TIA</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>(totals)</td>
<td>(1)</td>
<td>(6)</td>
<td>(2)</td>
<td>(1)</td>
<td>(1)</td>
<td>(3)</td>
<td>(1)</td>
<td>(5)</td>
<td>(0)</td>
<td>(1)</td>
</tr>
<tr>
<td>Single CI without prior TIA</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Single CI with prior TIA</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Multiple CIs</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>(totals)</td>
<td>(2)</td>
<td>(4)</td>
<td>(3)</td>
<td>(5)</td>
<td>(1)</td>
<td>(1)</td>
<td>(1)</td>
<td>(3)</td>
<td>(11)</td>
<td>(4)</td>
</tr>
</tbody>
</table>
TABLE 4. Severity of Residual Neurologic Deficit Following Infarction and Distribution of Lesions on Angiography

<table>
<thead>
<tr>
<th>Status</th>
<th>Normal angiogram</th>
<th>Equivalent lesion</th>
<th>Single cervical carotid lesion</th>
<th>Single intracranial carotid system lesion</th>
<th>Vertebrobasilar system lesion only</th>
<th>Multiple or bilateral carotid system lesions</th>
<th>Multiple carotid and V-B system lesions</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>(8)</td>
</tr>
<tr>
<td>II</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td>8</td>
<td>(16)</td>
</tr>
<tr>
<td>III</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>(6)</td>
</tr>
<tr>
<td>IV</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>(1)</td>
</tr>
</tbody>
</table>

Twenty-five patients had retinal and/or vertebrobasilar ischemic symptoms or mixed vertebrobasilar and carotid symptoms. There was no relationship between the nature of the symptoms and distribution or morphology of the arterial lesions (See tables 6 and 7).

Discussion

Is there a relationship between the anatomic sites of origin of the signs (or symptoms) of focal ischemia and the sites of the arterial lesions seen angiographically? An association of occlusive disease of the cervical and intracranial portions of the cerebral circulation with the occurrence of cerebral infarction has been known for more than a century; more recently the association with transient ischemic attacks has been defined.24 Eighty-two percent of the population in this study showed definite evidence of arterial disease at angiography and 77.5% of these lesions were in the appropriate arterial system. However, there was no clear relationship between the symptoms and the degree of dissemination of the arterial disease. This observation has been noted by others making an effort to find some correlation between the symptoms of focal cerebral ischemia and the distribution and severity of the lesions seen angiographically.7-11

Our second question was whether a relationship could be found between the clinical severity of ischemic symptoms and the character or morphology of arterial lesions demonstrated.

TABLE 5. Severity of Neurological Deficit Following Infarction and Morphology of Lesions on Angiography

<table>
<thead>
<tr>
<th>Status</th>
<th>No lesions</th>
<th>Equivalent lesion</th>
<th>Smooth plaque</th>
<th>Irregular plaque</th>
<th>Ulcerated plaque</th>
<th>Occlusion</th>
<th>FMD, &quot;kinking&quot;</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>III</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>IV</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

TABLE 6. Retinal and Vertebrobasilar Ischemic Symptoms and Distribution of Lesions on Angiography

<table>
<thead>
<tr>
<th>Status</th>
<th>Normal angiogram</th>
<th>Equivalent lesion</th>
<th>Single cervical carotid lesion</th>
<th>Single intracranial carotid system lesion</th>
<th>Vertebrobasilar system lesion only</th>
<th>Multiple or bilateral carotid system lesions</th>
<th>Multiple carotid and V-B system lesions</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single Retinal TIA</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>(4)</td>
</tr>
<tr>
<td>Multiple Retinal TIA</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>(6)</td>
</tr>
<tr>
<td>Retinal Infarcts</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>(4)</td>
</tr>
<tr>
<td>V-B TIAs and Infarcta</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>(2)</td>
</tr>
<tr>
<td>Mixed V-B and Carotid Symptoms</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>5</td>
<td>(9)</td>
</tr>
</tbody>
</table>
It is obvious that there is considerable heterogeneity in the distribution and in the underlying pathology of this arterial disease. A recent study by Imperato and associates underscores this. In atherosclerotic plaques removed at endarterectomy they demonstrated a wide variation, not only in size and degree of stenosis, but also in degrees of fibrosis, ulceration, atheromatus debris, intramural hemorrhage, and thrombosis. The situation is even more complicated than this; atherosclerosis is only one of a number of different processes that can produce cerebral ischemia and infarction.

Our study has attempted to define more precisely the nature of lesions seen on arteriography, not only their distribution and severity, but also surface characteristics. (It must be noted here that these radiographic characteristics such as "ulceration" do not correlate perfectly with actual pathological findings in specimens removed at surgery, as noted in a recent study by Edwards and associates. However, in correlating arteriographic findings with the clinical profiles we have found only a definite relationship with the presence and site of arterial disease; the clinical profiles could not predict subtypes of disease.

This leads to our final question regarding the need for angiography. In 1964 Marshall attempted clinically to separate TIs into groups with diagnostic significance. He noted a tendency toward a worse prognosis with TIs in the carotid territories and with TIs in the vertebrobasilar distribution if these included symptoms of hemiparesis. However, he found no absolute distinction between TIs which ultimately had a benign prognosis and those which led to infarction. Duncan and associates have also noted a worse prognosis for patients with TIs manifesting hemiplegia or with a duration of longer than one hour. In a similar fashion we have attempted clinically to separate cerebral ischemic disease into 2 groups with diagnostic significance, attempting to predict the morphology as well as sites of arterial pathology. We have found that clinical profiles lack this type of diagnostic specificity. The causes and underlying pathology of occlusive cerebrovascular disease are so complex that the evaluation and treatment of each patient with symptoms of cerebral ischemia must be pursued carefully and be individualized. The nature and extent of lesions in cerebrovascular disease can at present be adequately determined only by using cerebral angiography.

Angiography is recommended only in the context of electing vascular surgery if an appropriate lesion is seen on the angiogram. Discussion of the results of treatment is outside the purview of this paper.

**References**


---

**Table 7. Retinal and Vertebrobasilar Ischemic Symptoms and Morphology of Lesions on Angiography**

<table>
<thead>
<tr>
<th>Status</th>
<th>No. lesions</th>
<th>Equivocal lesion</th>
<th>Smooth plaque</th>
<th>Irregular plaque</th>
<th>Ulcerated plaque</th>
<th>FMD, &quot;kinking&quot;</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single Retinal TIA</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Multiple Retinal TIs</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Retinal Infarcts</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>V-B TIs and Infarcts</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Mixed V-B and Carotid Symptoms</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Notes:**

- **Occlusion** indicates whether the lesion was complete or partially occluded.
- **FMD, "kinking"** refers to the presence of focal narrowing or irregularity in the arterial wall.

---

The table summarizes the morphological findings associated with retinal and vertebrobasilar ischemic symptoms, dividing them into smooth plaque, irregular plaque, and ulcerated plaque, with additional data on the presence of occlusion and focal narrowing or "kinking".
Clinical phenomena and their correlation to angiographic findings in cerebrovascular disease.
D J Thurman and C H Millikan

Stroke. 1981;12:54-57
doi: 10.1161/01.STR.12.1.54

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/12/1/54

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/