**CONTRIBUTION OF THE CIRCLE OF WILLIS TO THE SUBCLAVIAN STEAL SYNDROME**

By Reginald S. A. Lord, M.D., F.R.C.S., F.R.A.C.S., Rafael Adar, M.D., and Robert L. Stein, M.D.

**SUMMARY**

The arteriograms and other findings in 42 patients with the subclavian steal syndrome were statistically evaluated to determine which factors predisposed to vertebrobasilar ischemia. Evidence of disconnection of the circle of Willis between the territories of the carotid and vertebral arteries was found in a significantly higher proportion of the patients with vertebrobasilar insufficiency. This finding was interpreted to mean that in these patients one of the major routes normally available for compensating for the basilar ischemia produced by the steal was not present.

It is suggested that this or similar mechanisms may be necessary before the subclavian steal produces symptoms in individual patients.

**Additional Indexing Words:**
Vertebrobasilar ischemia  Subclavian lesions  Carotid lesions  Collateral circulation  Brachial blood pressure

The subclavian steal syndrome is commonly believed to be a cause of vertebrobasilar ischemia. However, many patients with subclavian artery stenosis or occlusion and reversed vertebral flow are asymptomatic, while others present a clinical spectrum so variable that it is difficult to determine which symptoms, if any, are characteristic of the pure syndrome. The frequent coexistence of atherosclerotic lesions of the other extracranial and intracranial vessels in symptomatic patients only increases this difficulty. Precise elucidation of the mechanisms responsible for the neurologic symptoms is important since the subclavian steal syndrome is the prototype and the best established of the several vascular steal syndromes. General conclusions derived from the subclavian steal syndrome may be applicable to the understanding and treatment of these other syndromes.

In this study the hypothesis is proposed that vertebrobasilar insufficiency in the subclavian steal syndrome represents a failure of compensatory mechanisms to maintain normal flow in the posterior intracranial circulation. Anatomically these compensatory mechanisms are mediated through the contralateral subclavian and vertebral arteries, the carotid and innominate arteries, the circle of Willis and its branches, and secondary extracranial collateral pathways. Each of these will be examined in this paper.

The results of our study indicate that interruption of the components of the circle of Willis between the carotid and basilar arteries is a significant factor in causing vertebrobasilar insufficiency in the subclavian steal syndrome.

**Methods**

From January 1962 to August 1968, arteriograms were obtained because of suspected cerebrovascular insufficiency in 680 patients. In
154 cases the proximal subclavian artery was occluded or compromised by stenosis of at least 30% of its luminal diameter, and 53 of these patients demonstrated reverse flow in the ipsilateral vertebral artery. Eleven of the 53 cases were excluded because of inadequate arteriographic data or because the reversal of vertebral artery flow was caused by occlusion of the innominate artery. There were thus available for analysis 42 patients with subclavian artery occlusion or stenosis and reversed vertebral flow.

The neurologic symptoms of each patient were evaluated for evidence of vertebrobasilar insufficiency, and the patients were divided into two groups: those with vertebrobasilar symptoms and those without vertebrobasilar symptoms (table 1). Symptoms considered typical of vertebrobasilar insufficiency were one or more of the following: diplopia, vertigo, ataxia, and heterogeneous motor and sensory symptoms. Dizziness and binocular blurring of vision alone were regarded as not specifically indicating vertebrobasilar insufficiency and were not included in this category.

Several arteriographic technics were used, including retrograde catheterization from the femoral and axillary arteries and percutaneous carotid, subclavian, and left vertebral punctures. The degree of stenosis of each extracranial and intracranial artery was estimated as the maximal reduction in the luminal diameter in any radiographic projection. The degree of stenosis was graded in percentage of the luminal diameter occluded as follows: 0, 1-29, 30-49, 50-89, 90-99, and 100 (occlusion). The extracranial arteries were examined from the aortic arch to their termination, and the extent of collateral development was assessed. The maximal diameter of each vertebral artery was measured in the anteroposterior projection at the arbitrarily selected level of the fourth cervical vertebra. To exclude observer bias each arteriogram was interpreted without knowledge of the patient's symptoms.

### Results

#### Symptoms

Fifteen patients had symptoms of vertebrobasilar insufficiency and 27 did not (table 1). Three patients in the vertebrobasilar group had additional symptoms suggesting carotid insufficiency. Ten of the patients without symptoms of vertebrobasilar insufficiency had typical carotid insufficiency, 14 had nonspecific neurologic symptoms not indicative of vertebrobasilar insufficiency, and three had no neurologic symptoms.

Eleven of the 42 patients gave a history of arm pain or premature fatigability on exercise. In only two patients, neither of whom had symptoms of vertebrobasilar insufficiency, did arm exercise actually precipitate neurologic symptoms, namely episodic dizzy spells.

#### Age and Sex

The mean age of the 42 patients was 58 years (range, 42 to 73 years). Males slightly outnumbered females (23 to 19). Age and sex were not significantly related to the presence or absence of symptoms ($P > 0.05$, table 1).

#### Brachial Blood Pressure Inequality

The brachial systolic blood pressure differed by at least 20 mm Hg in every case. The mean pressure was 52 mm Hg (table 2), which is in agreement with the mean of 55 mm Hg obtained from a survey of the world literature to 1966. There was no significant difference between the two groups. The minimal systolic pressure differences of 20 mm Hg corroborated with the suggestion of Sammartino and Toole that a gradient of at least 20 mm Hg is necessary to reverse vertebral artery flow.
Table 2

Brachial Systolic Blood Pressure Difference

<table>
<thead>
<tr>
<th>Patients</th>
<th>BP difference (mm Hg)</th>
<th>Mean</th>
<th>SD</th>
<th>Min.</th>
<th>Max.</th>
<th>t value</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>With vertebro-basilar symptoms</td>
<td>48.0</td>
<td>20.8</td>
<td>20</td>
<td>100</td>
<td>0.80</td>
<td>&gt; 0.05</td>
<td></td>
</tr>
<tr>
<td>Without vertebro-basilar symptoms</td>
<td>54.1</td>
<td>24.8</td>
<td>20</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>51.9</td>
<td>23.4</td>
<td>20</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3

Primary Subclavian Lesion and Vertebral Artery Size

<table>
<thead>
<tr>
<th>Patients</th>
<th>Affected subclavian artery Laterality</th>
<th>Lesion</th>
<th>Mean vertebral artery size (mm)</th>
<th>Laterality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
<td>Occlusion</td>
<td>Stenosis</td>
</tr>
<tr>
<td>With vertebro-basilar symptoms</td>
<td>11</td>
<td>4</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Without vertebro-basilar symptoms</td>
<td>23</td>
<td>4</td>
<td>27</td>
<td>—</td>
</tr>
<tr>
<td>All patients</td>
<td>34</td>
<td>8</td>
<td>37</td>
<td>5</td>
</tr>
</tbody>
</table>

Principal Subclavian Lesion

The subclavian lesion responsible for the steal was an occlusion in 88% of the patients and was left-sided in 81% of the patients (table 3). There were five high-grade stenotic lesions (90-99%), virtually equivalent to complete occlusions.

Vertebral and Contralateral Subclavian Arteries

In all but five patients the vertebral artery on the side opposite the subclavian occlusion was of larger caliber than the ipsilateral vertebral artery (table 3). Since in normal subjects the left vertebral artery is usually dominant and most patients had occlusion of the left subclavian artery, this difference presumably resulted from compensatory arterial dilatation in response to chronically increased demand. There was no significant difference between the two groups in this respect.

Lesions of the contralateral proximal subclavian artery were equally infrequent in both groups; only one hemodynamically significant lesion (60% stenosis) was found in the entire group of 42 patients.

Stenotic lesions of the vertebral arteries usually involved the origin or the intraosseous part (table 4). Stenosis was more frequent in the contralateral than in the ipsilateral vertebral arteries. There was no differing incidence of both contralateral and ipsilateral vertebral artery lesions between the two groups (P > 0.05, table 4).

Innominate and Carotid Arteries

Obstructive lesions of the carotid arteries were common, and the carotid arteries were involved with almost equal frequency in each group (table 4). Innominate lesions were less common. Lesions of the innominate and the carotid arteries did not discriminate between the two groups.

Extracranial Collaterals

The reversed flow in the ipsilateral vertebral artery was the major source of collateral circulation to the subclavian artery distal to the obstruction. Supplementary collateral routes were often present, most frequently connecting the external carotid to the distal subclavian artery (36%, table 5). This pathway was commonly mediated either through anastomoses between the occipital artery and the cervical branches of the costocervical and thyrocervical trunks or through anastomoses...
Table 4

Incidence and Severity of Lesions of Other Vessels

<table>
<thead>
<tr>
<th>Artery</th>
<th>15 patients with vertebrobasilar symptoms</th>
<th>27 patients without vertebrobasilar symptoms</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of arteries</td>
<td>Mean degree of stenosis (%)</td>
<td>No. of arteries</td>
</tr>
<tr>
<td>Innominate</td>
<td>15</td>
<td>8.3</td>
<td>27</td>
</tr>
<tr>
<td>Ipsilateral carotid†</td>
<td>15</td>
<td>30.3</td>
<td>27</td>
</tr>
<tr>
<td>Contralateral carotid†</td>
<td>15</td>
<td>31.6</td>
<td>27</td>
</tr>
<tr>
<td>Ipsilateral vertebral</td>
<td>12</td>
<td>22.9</td>
<td>25</td>
</tr>
<tr>
<td>Contralateral vertebral</td>
<td>15</td>
<td>48.0</td>
<td>27</td>
</tr>
</tbody>
</table>

* t calculated without assumption of the variance.
† Includes common and internal carotid considered as one unit.

Table 5

Extracranial Collaterals to the Obstructed Subclavian Artery

<table>
<thead>
<tr>
<th>Patients</th>
<th>No. of patients</th>
<th>External carotid to vertebral</th>
<th>External carotid to subclavian</th>
<th>Subclavian to subclavian</th>
<th>Intercostal to subclavian</th>
<th>Transcervical vertebral to vertebral</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>With vertebro-basilar symptoms</td>
<td>15</td>
<td>1</td>
<td>5</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>Without vertebro-basilar symptoms</td>
<td>27</td>
<td>2</td>
<td>10</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>All patients</td>
<td>42</td>
<td>3</td>
<td>15</td>
<td>12</td>
<td>2</td>
<td>1</td>
<td>33</td>
</tr>
</tbody>
</table>

Table 6

Number of Intracranical Branch Arteries Examined

<table>
<thead>
<tr>
<th>Intracranial branch</th>
<th>Patients</th>
<th>Anterior</th>
<th>Middle</th>
<th>Posterior</th>
<th>Cerebral</th>
<th>Basilar</th>
<th>Cerebellar</th>
</tr>
</thead>
<tbody>
<tr>
<td>With vertebro-basilar symptoms</td>
<td>11</td>
<td>11</td>
<td>10</td>
<td>6</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without vertebro-basilar symptoms</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>16</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>25</td>
<td>25</td>
<td>24</td>
<td>22</td>
<td>21</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 7

Filling of the Posterior Cerebral Arteries (P.C.A.) from the Internal Carotid Artery (I.C.A.)

<table>
<thead>
<tr>
<th>P.C.A. filled from I.C.A.</th>
<th>Patients</th>
<th>No.</th>
<th>%</th>
<th>Total examined</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>With vertebro-basilar symptoms</td>
<td>11</td>
<td>79</td>
<td>14</td>
<td>&lt; 0.002</td>
<td></td>
</tr>
<tr>
<td>Without vertebro-basilar symptoms</td>
<td>9</td>
<td>33</td>
<td>27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>20</td>
<td>49</td>
<td>41</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

between the superior and inferior thyroid arteries.

The extent of collateral development was comparable in the two groups of patients.

Intracranial Circulation

Complete radiologic demonstrations of the anterior, middle, and posterior cerebral arteries and the basilar and cerebellar arteries were obtained in only 20 of the 42 patients, although a greater number of individual arteries were available for analysis (table 6). Lesions of these vessels were few and were not responsible for the differences in the symptoms of the two groups.

In 41 of the 42 patients it was possible to determine whether one or both posterior cerebral arteries filled predominantly from the internal carotid or basilar artery (table 7). In 11 of 14 patients with vertebrobasilar symptoms at least one of the posterior cerebral arteries was observed to fill from the internal
Carotid arteriogram showing an embryonic type of posterior cerebral artery (PCA) filling with radiopaque medium directly from the internal carotid artery (ICA).

Figure 1

Carotid arteriogram showing the posterior cerebral artery (PCA) filling from internal carotid artery (ICA). The presence of an angulated junction with the posterior communicating artery indicates that the posterior cerebral artery is probably not an embryonic type.

Figure 2
carotid artery rather than from the basilar artery. In five of these 11 patients, one posterior cerebral artery showed a typical embryonic form characterized by a straight posterior course from the internal carotid without significant change in caliber to the point of major branching (fig. 1). In the remaining six patients one or both posterior communicating arteries were at least as large as the posterior cerebral arteries distal to their junction. However, the presence of an angulated junction indicated that the posterior cerebral artery was probably not of purely embryonic type (fig. 2).

These findings contrasted strongly with those in the 27 patients without vertebrobasilar symptoms, in only nine of whom were one or both posterior cerebral arteries filled from the carotid arteries. There was only one embryonic posterior cerebral artery in this group, and in only three other patients was the caliber of the posterior communicating artery equal to that of the posterior cerebral artery. These differences were statistically highly significant ($P < 0.002$).

Continuity between the internal carotid and basilar arteries in the circle of Willis is normally maintained through the posterior communicating arteries and the divisional segment of the posterior cerebral arteries, that is, the part between the basilar and posterior communicating arteries. The posterior cerebral artery is most commonly filled predominantly from the basilar artery. In the embryonic type of posterior cerebral artery, filling is predominantly from the carotid artery and the divisional segment is hypoplastic or atretic, producing functional or actual interruption of the circle of Willis. These findings were, therefore, interpreted to mean that some degree of discontinuity of the circle of Willis between the carotid and vertebrobasilar systems was significantly more frequent in patients with vertebrobasilar symptoms.

Discussion

Reversal of vertebral artery flow from ipsilateral subclavian occlusion was first reported by Contorni in 1960. The following year, Baker, Toole, and Fields each presented arteriograms illustrating this phenomenon. A subsequent description of two of these patients prompted the editors of the New England Journal of Medicine to suggest the term "subclavian steal" for the new syndrome on the basis of an erroneous assumption of the interrelationship of arm exercise, reversal of vertebral artery flow, and the production of neurologic symptoms.

Experimental studies indicate that vessels as large as the carotid and iliac arteries are unlikely to suffer from the steal phenomenon since their flow capacity can normally increase to some 10 times their resting levels with little rise in resistance. These conclusions are probably applicable to the vertebral artery although its resistance is much greater than that of the carotid artery. The presence of a hemodynamically significant stenosis in the contralateral vertebral artery undoubtedly limits its flow capacity, thus predisposing to a true steal. The finding in this study that a contralateral vertebral artery stenosis was not a statistical determinant of vertebrobasilar insufficiency further weakens the concept that reversal of vertebral artery flow alone is the cause of neurologic symptoms in the subclavian steal syndrome.

Experimental evidence has indicated that proximal subclavian occlusion causes a slight reduction in total cerebral perfusion. In man and in the rabbit the deficit is normally compensated by an increased carotid inflow. The integral role of the circle of Willis in this compensatory pathway is acknowledged.

The present results indicate that anomalous development of the circle of Willis contributes to the symptom pattern of patients with the subclavian steal syndrome. Hypoplasia and atresia of the divisional segment of the posterior cerebral artery are common anomalies, having been variously estimated as occurring in $15\%$ to $30\%$ of the population. Although on a statistical basis, lesions of the divisional segment of the posterior cerebral artery were the only signifi-
cant finding to emerge from this study, it is not suggested that this is the only mechanism of vertebrobasilar insufficiency in individual patients since a variety of other mechanisms can easily be postulated. However, as suggested on theoretical grounds, the subclavian steal is probably not a definitive syndrome unless the circle of Willis is at least partially disconnected or other secondary influences are present.

These findings have obvious therapeutic application to the subclavian steal syndrome. The presence of a disconnected circle of Willis in a particular patient makes it unlikely that vertebrobasilar insufficiency will be relieved by operations which augment carotid inflow. In these circumstances correction of the steal itself or improvement of the vertebral inflow on either side is more appropriate. Operations upon the carotid and innominate arteries should be most effective when the circle of Willis is intact.

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References

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