Original Contributions

The Pathogenesis of Strokes From Internal Carotid Artery Occlusion. Diagnostic and Therapeutical Implications

ERICH BERND RINGELSTEIN, M.D.,* HERMANN ZEUMER, M.D.,† AND DIMITRIOS ANGELOU, M.D.‡

SUMMARY In order to relate clinical, angiologic and brain-morphological findings, 107 patients with internal carotid artery occlusion were examined clinically and by Doppler sonography, computerized tomography and cerebral angiography. During computerized tomography, haemodynamically induced infarctions could be differentiated from those, caused by periocclusive embolism towards the major brain arteries. This differentiation was based on an integration of angiologic as well as brain-morphological data and implicates some important diagnostic, therapeutic and prognostic consequences. The first group may benefit from immediate surgical restoration of carotid blood flow and have a relatively good prognosis. By contrast, subjects presenting territorial infarctions mostly experience severe and permanent neurological deficits. They should not undergo revascularization since endarterectomy increases the risk of cerebral hemorrhage but cannot cause clinical improvement. In five patients, following special angiographic techniques, the lesions of the internal carotid artery turned out to be pseudoocclusions. This condition threatens the patient considerably but is well accessible to surgical reconstruction. Thus, pseudoocclusion seems to be one of the most convincing indication for emergency endarterectomy of the internal carotid artery.

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IT SEEMS TO BE WIDELY ACCEPTED that a stroke, resulting from an occlusion of the ICA is a clinical and pathogenetic entity, which can only be differentiated according to the degree and course of the concomitant neurological deficit. Nowadays, however, the clinician’s objective should be a more subtle diagnostic analysis of patients suffering from ICA occlusion.† With the help of continuous wave (CW) Doppler sonography, cerebral angiography and computerized tomography (CT) an integration of angiological and morphological information has become possible. This new approach permits clearer insight into the different pathogenetic mechanisms underlying the ischemic brain damage from ICA occlusions. This leads to more sophisticated diagnostic and therapeutic consequences.

The diagnostic tools available before the advent of CT were limited by several shortcomings: Cerebral angiography — besides its invasiveness — could do no more than elucidate the angiologic aspects of post-occlusive brain infarctions, whereas the nature and extent of brain-damage could not be defined directly and simultaneously. By contrast, postmortems revealed the ischemic brain lesions, but often failed to shed light on the specific vascular pathology that had led to the stroke.

From postmortems it is known, that occlusion of the ICA remains asymptomatic in a considerable percentage of cases. In the majority of patients, however, either haemodynamically induced "extraterritorial" infarctions occur or ICA occlusions lead to embolic lesions of the intracranial arteries with ischemic brain damage within the territory of these vessels. There are only few authors who have emphasized this latter mechanism of cerebral insults in patients with ICA occlusions.

In this paper we present an analysis of clinical, angiologic and CT-morphological findings in 107 patients with ICA occlusion. Five subjects, which at first glance seemed to have complete occlusions, turned out to be pseudoocclusions of the ICA and were included in the study.

Patients and Methods

In 107 patients, an ICA occlusion was diagnosed by either direct CW Doppler sonography, or transfemoral selective cerebral angiography or both. Patients with traumatic (1 case), inflammatory (2 cases) or proven embolic (9 cases) ICA occlusion were excluded from the study.

Doppler sonography was applied to the periorbital branches of the ophthalmic artery as well as directly to the whole extracranial segment of the carotid artery. The technique is described in detail elsewhere with special reference to the detection of pseudoocclusion of the ICA.

In order to detect pseudoocclusion of the internal carotid artery, special techniques of transfemoral angiography were applied in 21 subjects: (1) a long serial imaging run, lasting 8 sec., was performed on the side...
of the carotid lesion. Subtraction techniques were routinely used. The flow rate of the contrast medium was reduced to 8 ml/sec with an injection bolus of 10 ml.

(2) After ipsilateral angiography was done contralateral intracranial series of the selectively catheterized non-occluded carotid artery was performed if one of the following conditions was present: CT examination had revealed brain infarction within the territory of one or more single pial arteries or their major branches, suggesting an embolic pathogenesis. In such cases where CT was not helpful within the first 24 hours in defining the type of brain infarction, a complete angiographic examination was necessary. The rationale was to visualize embolization distal to the occlusion. (3) If under these circumstances collateralization through the anterior communicating artery did not permit the passage of contrast medium to contralateral supraocclusive vascular bed, verteobasilar angiography was performed.

As far as could be determined, the time interval between onset of neurological symptoms and angiography ranged from several hours to 38 days.

All vascular brain lesions which appeared to be related to the occlusion of the carotid artery were classified according to Zülch 1961,15,16 Gastaut et al 19711 and Wodarz 198017 as well as according to the description and topographical analysis of the territories of single pial arteries and their branches by von Keyserlingk.30

Additionally, special emphasis was placed on differentiating lacunes from other brain infarctions31 in order to exclude patients with atherosclerosis of small penetrating arteries from this study.7,9,18

The diagnostic scheme for classifying ischemic brain lesions in CT1 from a pathogenetic point of view was the following (fig. 1).

Results

There were 107 patients (66 males, 41 females) with ICA occlusions. The mean age was 58 years ranging from 34 to 79 years. As expected, the maximum frequency of ICA occlusions (73 cases) occurred during the fifth and sixth decade of life. Bilateral ICA occlusions were found in four patients. In half of the patients additional significant lesions of other arteries (contralateral carotid artery, posterior circulation) were present.

Clinical data concerning premonitory symptoms, previous strokes and acuteness of ischemic brain symptoms are tabulated (table 1). The most striking feature is the high percentage of sudden onset strokes with or without premonitory attack in patients suffering from pial artery territory infarctions. By contrast, hemodynamically induced infarctions most often lead to repetitive minor strokes and fluctuating symptoms.

The frequency of the different localization patterns of ischemic brain lesions resulting from ICA occlusion is listed in Table 2. Three distinct patterns of brain lesion predominated: 1) infarctions suggesting embolic pial artery occlusions, i.e. pial artery territory infarctions (fig. 2); 2) hemodynamically induced subcortical terminal supply area infarctions (fig. 3) (“terminal zone infarctions,”19); and 3) typical cortical-subcortical watershed infarctions (fig. 4) (“borderline infarctions of the convexity,”17) which could be observed in only 8 patients of the whole group.

Five patients with watershed infarctions were more than 70 years old. Two of them had contralateral high-grade stenosis of the ICA. One 77-year-old patient with a slowly progressive hemiparesis was severely dehydrated on admission. However, none of these patients had a recent history of myocardial infarction or hypotensive crisis.

“Extended infarction of the lentiform nucleus” was only found once in this series. Angiography in this patient revealed secondary embolism into the middle cerebral artery following ICA occlusion (fig. 5). Forty-one percent of the lesions were classified as hemodynamically induced and forty percent as a consequence of periocclusive embolism (table 2).

When we attempted to relate the type of brain lesion with the type and severity of the neurological deficit, a close relationship was established between subcortical terminal supply area infarctions as well as pial artery territory infarctions on one hand and marked hemiparesis or hemiplegia on the other. In contrast, watershed infarctions of the convexity tended to cause only mild neurological sequelae and the prognosis for the restitution of function was good (table 3).

Angiological screening of the contralateral brain arteries and verteobasilar system included visualization of the collateral circulation as well as the supraocclusive vascular bed. This revealed ipsilateral pial artery occlusions (i.e. “occlusio supra occlusionem” (2) or “secondary emboli”19) in 15 of the 21 cases thoroughly examined. All of them had the typical pattern pial artery territory infarction by CT (fig. 1 and 2).

Within the first day after the onset of symptoms twelve of the patients showing CT patterns of terminal supply area infarction had angiography. None revealed secondary emboli to the intracranial arteries.

In five patients, pseudoocclusion of the ICA could be demonstrated (fig. 6). By means of CW Doppler

All CTs were made with the SIRETOM 2000, (SIEMENS GMBH, Nuernberg, FRG) with overlapping slides, each of them 10 mm thick.

FIGURE 1. Brain infarction due to internal carotid artery occlusion. The pathomechanism, the terminology proposed here and used in the literature, the site and the topographical delineation of the lesions in computerized tomography resulting from internal carotid artery occlusion are presented.

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Clinical findings

<table>
<thead>
<tr>
<th>Type of brain infarctions</th>
<th>No deficit recognized</th>
<th>One or more TIs or minor strokes</th>
<th>Progressive stroke</th>
<th>Completed stroke with premonitory attacks</th>
<th>Completed stroke without premonitory attacks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pial artery territory infarction (n = 43)</td>
<td>3 (7%)</td>
<td>10 (23%)</td>
<td>—</td>
<td>5 (12%)</td>
<td>25 (58%)</td>
</tr>
<tr>
<td>Extraterritorial watershed infarctions (= borderline infarctions of the convexity) (n = 8)</td>
<td>—</td>
<td>3</td>
<td>2</td>
<td>—</td>
<td>3</td>
</tr>
<tr>
<td>Terminal supply area infarctions (n = 36)</td>
<td>2 (6%)</td>
<td>18 (50%)</td>
<td>4 (11%)</td>
<td>5 (14%)</td>
<td>7 (19%)</td>
</tr>
<tr>
<td>Σ 87</td>
<td>5 (6%)</td>
<td>31 (36%)</td>
<td>6 (7%)</td>
<td>10 (11%)</td>
<td>35 (40%)</td>
</tr>
</tbody>
</table>

Sonography this could be done prior to angiography in four cases, four times with the angiographical techniques described above and three times by combination of both techniques. In one case only, pseudoocclusion was misdiagnosed by Doppler sonography as an occlusion of the ICA. Thus, in most cases, pseudoocclusion of the ICA was predicted correctly by CW Doppler sonography. They presented the otherwise typical sonographic features of ICA occlusion (i.e. retrograde blood flow of the ophthalmic artery, altered blood velocity profile of the common carotid artery and missing signal from the ICA). Additionally, in examining the region of the carotid bulb a faint, sharp and continuously hissing sound appeared. Its faintness prevented documentation on both the scope as well as the recording chart. However, the tone of this non-pulsatile hissing was so characteristic, that its recognition definitely permitted the diagnosis of pseudoocclusion of the ICA. Subsequent short serial carotid angiograms suggested a complete occlusion, and only extended angiographic series with subtraction techniques made the correct diagnosis of pseudoocclusion possible. One striking feature of pseudoocclusion of the ICA should be emphasized: In all of the cases observed, a subcortical terminal supply area infarction occurred (fig. 6). By contrast, lesions suggestive of embolic pial artery occlusion were not found in this group.

Three of the patients with pseudoocclusion were immediately operated on successfully. (One refused operation, one died from subarachnoid hemorrhage due to a berry aneurysm of the basilar artery).

Out of the group with hemodynamically induced infarctions an additional nine patients underwent vas-
cular surgery. Emergency endarterectomy was performed in three patients having had progressive strokes. Another two out of four patients with stable neurological deficits successfully underwent revascularization of the ICA. Up to now they have had no further attacks. Extracranial-intracranial bypass procedures were done in only two cases with stabilization of symptoms. None of the patients in the secondary embolism group had reconstructive surgery.

Discussion
The pathogenetic mechanisms underlying brain infarctions from ICA occlusion are obviously manifold. The spectrum of lesions ranges from asymptomatic carotid occlusion to hemodynamically induced insults and to embolic occlusions of the main intracranial arteries, especially of the middle cerebral artery (MCA) and its branches. Therefore, a thorough diagnostic work-up of patients with internal carotid artery occlusions and pseudoocclusions is mandatory. This should include angiological (CW Doppler sonography, and bilateral carotid arteriography, as well as CT. Both hemodynamically induced and embolic infarctions following ICA occlusion are equally frequent. However, pial artery occlusions resulting from embolism are of far greater clinical importance because of their worse prognosis and their more severe neurological deficits.

The predominantly embolic nature of strokes following ICA occlusion had already been described in earlier papers (see references in Hultqvist 1942 as well as in Landolt and Millikan 1970. Unfortunately, this view did not receive adequate attention. In 1942, Hultqvist analysed the postmortems of 54 cases with ICA occlusion. He found the prognosis especially poor if a thrombus was located distal to the atherosclerotic occlusion. This important finding was later reemphasized by other authors. Furthermore, emboli from the tail of the spongy clot could be sheared by the collateral blood stream within the circle of Willis and consequently be shed into the major pial arteries. Einsiedel-Lechtape has recently placed new emphasis on the poor prognosis of strokes caused by these "secondary emboli" due to ICA occlusion. The prognosis of ICA occlusions clearly depends on the occur-

![Figure 3 A-C. Occlusion of the ICA causing subcortical terminal supply area infarction.](http://stroke.ahajournals.org/Downloaded from http://stroke.ahajournals.org/)

(A) Cerebral angiography, by cross-collateral flow, reveals slow filling of the supraocclusive cerebral arteries. Additionally, perfusion pressure in the left middle cerebral artery is decreased by stenosis of the anterior cerebral artery on the left (small arrows). Retrograde filling of the distal stump of the left internal carotid artery is visible (large arrow).

(B) Subtraction of the images from a later period of the imaging run clearly indicates delayed but complete filling of the lenticulo striate vessels (left arrow). On the right side, contrast medium has still passed the lenticulo striate arteries (right arrow).

(C) As a consequence of the severe drop of perfusion pressure within the left-sided cerebral arteries, a terminal supply area infarction has occurred extending from beyond the basal ganglia up to the peri- and supraventricular white matter of the corona radiata and the centrum semiovale. The lentiform nucleus remained completely intact (not shown here). Compare with figure 5, where the lentiform nucleus was predominantly involved.
rence of this latter mechanism of "delayed stroke" or "occlusio supra occlusionem" whereas "the extracranial carotid occlusion is only indirectly responsible" for the clinical state of the patient. This view is supported by the findings of Landolt and Millikan in patients after carotid artery ligation. In these subjects, early ischemic complications, mostly transient, were predominantly attributed to either hemodynamic disturbances or to early fragmentation of fresh clot with rapid lysis of "smaller and more brittle" emboli (p. 60). Later brain damage, by contrast, mostly led to persistent neurological deficits due to occlusion from older and more organized embolic material.

Recently, other insult mechanisms following ICA occlusion have gained some interest. The ICA "stump" as well as stenosing lesions of the external carotid artery (ICA) stump are often the sites of additional emboli that can cause cerebral infarction.

**Figure 4 A & B.** Watershed infarction due to ICA occlusion. (A) Contralateral cerebral angiography, by cross filling, reveals intact blood flow into the supraocclusive major intracranial arteries. (B) Nevertheless, watershed infarction occurred in the borderline area between the anterior and middle cerebral artery. The watershed infarction indicated a critical loss of perfusion pressure in the extraterritorial pial network of arterioles between the territories of the anterior and middle cerebral arteries (CT images inverted for better demonstration).

**Figure 5 A & B.** "Extended infarction of the lentiform nucleus" due to ICA occlusion. (A) Contralateral cerebral angiography, by cross filling, reveals distal stump of the occluded ICA (large arrow) and recanalized secondary embolus in the middle cerebral artery (small arrows) lodging exactly at the origin of the lenticulostriate arteries and blocking their orifices. The ICA occlusion was additionally proved by ipsilateral arteriography. (B) Blockage of the lenticulostriate arteries causes cerebral infarction restricted to the lentiform nucleus, visible in the two upper pictures of CT. Additionally, infarction occurred in the centrum semiovale, i.e., in the whole territory of the lenticulostriate arteries (CT images inverted for better demonstration).
carotid artery are occasional sources of emboli. The embolic material may pass through extracranial anastomotic channels to lodge in the retinal and cerebral arteries. The evaluation of this special pathogene-

TABLE 2 Lesions in CT from 111 Internal Carotid Occlusions in 107 Patients

<table>
<thead>
<tr>
<th>Type of lesion</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Thrombo-)embolic:</td>
<td></td>
</tr>
<tr>
<td>pial artery territory infarction</td>
<td>43</td>
</tr>
<tr>
<td>extended lentiform nucleus infarction</td>
<td>1</td>
</tr>
<tr>
<td>Hemodynamically induced:</td>
<td></td>
</tr>
<tr>
<td>terminal supply area infarction</td>
<td>36</td>
</tr>
<tr>
<td>watershed infarction (convexity)</td>
<td>8</td>
</tr>
<tr>
<td>No lesions in CT visible or lesion not attributable to ICA occlusion (predominantly lacunes)</td>
<td>24*</td>
</tr>
</tbody>
</table>

*8 lesions clinically asymptomatic.

TABLE 3 Type of Brain Lesion due to Internal Carotid Artery Occlusion Correlated with the Severity of the Neurological Deficit (111 ICA Occlusions in 107 Subjects)

<table>
<thead>
<tr>
<th>Type of lesion</th>
<th>Mono-or hemiplegia</th>
<th>Slight paresis</th>
<th>Minimal or no deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Watershed infarct</td>
<td>8</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>Frontal</td>
<td>5</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Parieto-occip.</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Terminal supply area infarction</td>
<td>36</td>
<td>10</td>
<td>24</td>
</tr>
<tr>
<td>Pial artery territory infarction</td>
<td>43</td>
<td>19</td>
<td>21</td>
</tr>
<tr>
<td>No lesion in CT or lesion not attributable to ICA occlusion</td>
<td>24</td>
<td>3*</td>
<td>9</td>
</tr>
</tbody>
</table>

*One subject with cerebral contusion due to trauma, two subjects with multiple lacunes involving the internal capsule.

s of stroke may lead to surgical removal of the embolizing source.

However, an exact analysis of the temporal profile of periocclusive strokes considering the sequence of the underlying pathophysiological events, only seems to be occasionally possible. At least in some patients a massive embolus is fired into the MCA immediately before the definite occlusion of the extremely stenotic carotid artery occurs. In one of the patients we could, by chance, observe this sequence. First, after occurrence of a severe hemiplegia, high-grade ICA stenosis was evaluated by Doppler sonography. Some hours later, however, angiography revealed complete ICA occlusion and a secondary embolus lodging in the MCA. Recently, another patient with a three-week history of repeated transient monocular blindness and ischemic attacks was found to have a subtotal ICA stenosis during Doppler sonography. Only a few minutes before angiography was performed, he experienced a complete left-sided hemiplegia. Angiography then showed both subtotal ICA stenosis with a large poststenotic thrombus formation (fig. 7A) and an embolus located in the proximal segment of the MCA (fig. 7B). In order to attempt lysis of the MCA embolus and to prevent complete ICA occlusion, local intraarterial fibrinolysis was successfully administered with nearly complete recovery of the neurological deficit (fig. 7C and D). Endarterectomy of the carotid plaque could be performed the next day.

The mechanism leading to the final occlusion of the embolizing ICA lesion within hours or days remains unclear. Nevertheless, with respect to pseudoclosures, the following thesis, portraying one of the mechanisms of periocclusive events, is presented (fig. 8): (A) Highgrade ICA stenosis, at the most, causes minimal poststenotic reduction of arterial diameter and poststenotic pressure drop. (B) The collapse of the poststenotic vessel reaches a maximum during the stage of pseudoclosure when the pressure gradient is nearly maximal. Small emboli may be released causing minor strokes, or hemodynamically induced in-

FIGURE 6 A & B. Pseudoclosure of the ICA causing subcortical terminal supply area infarction.

(A) Cerebral angiography, in a long serial imaging run and using subtraction techniques, reveals pseudoclosure of the internal carotid artery, leaving a triangular stump of the nearly occluded ICA at its origin (black arrow). Only on the late images minimal enhanced opacification occurs in the collapsed ICA (pairs of small white arrows).

(B) Ischemic infarction of the white matter is present in the terminal supply area of “small penetrating arteries” (7), i.e. of the lenticulostriate arteries. Only the area of the “last field” (15), involving the corona radiata and extending cephalad into the centrum semiovale, is necrotic, suggesting hemodynamical causation.
farctions may occur during this stage. (C) A large embolus is shed from the "nearly occluded ICA" into its main draining pial artery, the MCA, causing a severe stroke. Immediately, the flow in the cervical ICA is further reduced, causing spontaneous thrombosis of blood in the tiny remaining channel of the pseudoocclusion. Local flow reduction is known to multiply the importance of blood viscosity disturbances for the pathogenesis of thrombosis. Subsequent angiography will then reveal complete ICA occlusion, eventually together with CT-findings suggesting brain embolism. These findings, however, are indistinguishable from "secondary embolism" into the pial arteries which may occur after ICA occlusion.

The thesis presented above is indirectly supported by prior pathologic studies. Samuel found "pure atherosclerosis" of the ICA producing "pin-point occlusions." Other authors described ICA occlusions which "remained restricted to a short segment of the ICA for a considerable period of time leaving the distal portion of the vessel free from thrombus." The findings suggest that these carotid lesions were actually pseudoocclusions. Total occlusions from atherosclerotic lesions alone do not occur, as the final occlusion is always brought about by thrombosis. With an increase in the degree of atherosclerotic lesions, there is also an increase in the tendency towards occlusion by superimposed thrombus.

Besides such diagnostic and pathophysiological aspects our findings also have therapeutic implications. Emergency carotid endarterectomy has proven useful in only three groups of patients: Firstly, in those with fluctuating symptoms, primarily indicative of oscillating hemodynamical disturbances in the borderline zones, secondly, in patients with stuttering but still limited deficits, primarily suggestive of peripheral premonitory, preocclusive small emboli, and thirdly, in the earlier stage of a rapidly progressive stroke of hemodynamic as well as embolic origin. These patients may benefit from immediate restoration of ICA blood flow or from removal of the extracranial source of emboli. From the clinical signs and course alone, however, a reliable identification of these patients cannot be made. By contrast, complete visualization of the intracranial arteries and, in some cases, CT-morphology, can adequately solve this diagnostic problem.

**Figure 8 A-C.** Diagrammatic representation of probable vascular events immediately before ICA occlusion occurs on the basis of ICA pseudoocclusion. (A) Tight ICA stenosis is present with or without symptoms due to small emboli. During this stage, no poststenotic arterial collapse is visible. (B) The stenosis has reached the degree of "pseudoocclusion." Now significant reduction of vessel diameter is evident. The drop of poststenotic blood flow is nearly maximal, and, as a result, hemodynamically induced infarctions may occur. (C) The nearly occluded ICA has shed a large embolus into its main draining branch, i.e., into the middle cerebral artery. In this situation, additional reduction of blood flow through the extreme stenosis of the ICA definitely leads to its thrombotic occlusion. Additionally, anterograde thrombus (hatched zone) may occur.
and thus provide the "guidelines for the proper selection of patients for surgery." The same prerogative is essential for the selection of patients for extracranial-intracranial bypass procedures.19, 24-26, 43

In accordance with recent publications23-26, 29 the "nearly occluded ICA", i.e. "atheromatous pseudo-occlusion of the ICA"24 offers a new and promising field for emergency endarterectomy. All of the patients operated on presented hemodynamically induced infarctions on CT. As a rule, this type of infarction would suggest good responsiveness to restoration of carotid blood flow. Unfortunately, the cited literature (with the exception of 29) does not provide CT-data on the type of stroke which occurred in the patients with pseudoocclusions. The clinical descriptions, however, suggest that these were cases with minor embolic as well as hemodynamically caused strokes.

On the contrary, emergency carotid endarterectomy cannot be helpful in subjects with severe secondary embolism into the large intracranial pial arteries, even if the neurological deficit is incomplete or decreasing. Extracranial restoration of blood flow cannot improve the blood supply of the necrotic brain area. Furthermore, high pressure perfusion of the damaged territory is known to cause intracerebral hemorrhage with fatal clinical consequences.41, 44-45 Failure to consider these prerequisites for emergency carotid endarterectomy seems to be the cause of the unsatisfactory postoperative results reported in the literature.4, 11 The striking discrepancies in previous reports on the effect of endarterectomy in ICA occlusions may be explained by differences in the type of patients included in the studies. Relying on the data in the literature41, 44-46 and on earlier personal experience, we consequently avoided emergency endarterectomy, if major secondary emboli had already occluded one or more pial arteries. In contrast, good clinical results were seen in patients who underwent endarterectomy immediately following postoperative re-thrombosis of the ICA.26 This is understandable, since these patients had not yet had periocclusive emboli.

The length of the interval between the occurrence of carotid occlusion and endarterectomy obviously determined the number of successful restorations of ICA blood flow42 but does not allow a definition of the effectiveness of surgical therapy for the patients' recovery from ischemic brain symptoms.

Acknowledgments

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