“Stump” of Internal Carotid Artery — A Source for Further Cerebral Embolic Ischemia

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SUMMARY A series of 9 patients have experienced hemisphere and retinal ischemia at an interval after occlusion of appropriate internal carotid arteries. All had radiological evidence of a persisting proximal stump to the occluded artery and, in most, pathological evidence of thrombotic material attached to atheromatous lesions within the stump. Thromboembolism from the stump via the anastomotic supply through ipsilateral common and external carotid arteries is thought to be responsible for the ischemic events to the brain or retina despite absence of flow through the internal carotid artery. Seven of the 9 were treated by surgical excision or obliteration of the stump and, when indicated, common and external carotid endarterectomy. Turbulence in the stump contributed to progressive atherosclerotic changes and probably aggravated thrombogenesis in this location with subsequent embolization into the anastomotic arteries.

THE RELATIONSHIP between recurrent cerebral or retinal ischemic events and stenotic and ulcerative atheromatous lesions of the carotid artery is widely accepted. A statement has been made frequently that ischemic events could be expected to cease once a stenosed artery becomes occluded.1-6 Recent publications7-10 have drawn attention to ischemic events within the territory of an occluded artery. The majority of these later recurrent events appear to be of embolic origin related to stenotic and ulcerative lesions in the collateral circulation, particularly in the common and external carotid arteries. Continuing study of this type of case has drawn the authors’ attention to the occurrence, in some of them, of lesions within the stump of the internal carotid artery which have been shown by angiography and pathological examination of surgical specimens to be capable of causing or contributing to the post-occlusion thromboembolic attacks.

Patients and Methods

This report is based on a prospective study of 9 patients presenting with retinal or cerebral ischemic event(s), all with evidence of occlusion of the internal carotid artery in the neck, and all with radiological evidence of a non-obliterated proximal remnant (a “stump”) of the internal carotid artery. The clinical features have been analyzed and the radiological evidence assessed to determine if it is a reasonable assumption that the symptoms and signs could have been related to thromboembolism from the region of the stump. The availability of anastomotic channels to carry the emboli to the target organs of brain and eye was studied. In 7 of the 9 cases, pathological material was available, subsequent to surgery, and this was correlated with the radiological and clinical findings. The outcome was assessed in the 7 treated by surgery and in the 2 treated conservatively.

A separate review was conducted of all radiographs demonstrating internal carotid artery occlusion admitted to this unit over a 3-year period to determine in 74 consecutive cases the incidence and the length of internal carotid “stumps” encountered.

Case 1

A 69-year-old man presented with sudden headache and visual disturbance. Examination disclosed a right homonymous hemianopia and carotid angiography displayed an occlusion of his left internal carotid artery about 1 cm above its origin from the common carotid artery (fig. 1a). There was no significant disease visible in the external carotid or common carotid arteries on this side. The right internal carotid artery was stenosed with a 50% reduction in the lumen in the region of the carotid sinus. The posterior circulation, visualized with bilateral vertebral angiography, was normal. The left hemisphere was receiving collateral circulation through the left ophthalmic artery (fig. 1b) and through the anterior communicating artery. The left posterior cerebral artery was supplied through the anterior circulation.

The hemianopia cleared in a few weeks and the patient was symptom-free until 20 months later when he had a right hemiparesis with maximum involvement of the hand, some weakness of the leg and some diminished sensation in the hand. This recovered in 1 hour and neurological investigation 1 month later revealed no residual neurological signs. A carotid angiogram now indicated that the stump (fig. 2a) of the internal carotid artery was shorter, and that the common and external carotid arteries were not significantly abnormal. In a later picture in the series, there appeared to be a collection of thrombus or an atheromatous plaque intruding into the stump (fig. 2b). An endarterectomy of the common and external carotid arteries was carried out, with ligation and excision of the stump of the internal carotid artery. Attached to the de-endothelialized wall of the base of the stump of the internal carotid artery was a mass of thrombus (fig. 3) and beneath it was an atheromatous...
ulcer with necrotic material, foamy histiocytes and multi-nucleated foreign body giant cells characterizing the base of the ulcer (fig. 4). In the apex of the stump there was a smooth reddish ulcer and beneath this there was a large area of atheromatous material including cholesterol crystals, clefts, red cells, platelets and calcified debris. The external and common carotid arteries were patent, with intact endothelium, and no more than minor intimal hyaline change.

No new neurological symptoms occurred and the patient died from unrelated causes 4 months later.

Case 2
This 53-year-old male became aware of right hand tingling, persisting for 6 weeks. Over the next 3 months he was aware of 10–12 episodes each lasting 5 minutes of recurrent tingling in the same area. He had a 10 minute dysphasic episode in September, 1975, and 6–7 episodes of about 5 minutes duration, of numbness in the right arm, leg, and trunk in October, 1975. These episodes led to investigation which disclosed no neurological abnormality. Cerebral angiography on November 11, 1975, demonstrated an occluded left internal carotid artery with a 1½ cm tapering stump of the internal carotid artery and no abnormalities in the common or external carotid arteries on this side (fig. 5a). Collateral flow was demonstrated, retrograde through the ophthalmic artery into the distal portion of the carotid siphon (fig. 5b). The right carotid artery was the site of marked atheromatous change in the carotid siphon and a moderate stenosis over the first 1½ cm of the internal carotid artery in the neck. Cross circulation through the anterior communicating artery supplied anterior and middle cerebral artery territory on the left side. The right vertebral artery was normal. The left vertebral artery appeared to be occluded at its origin with its distal portion receiving generous supply from the cervical anastomotic network. The patient was...
entered into a platelet-inhibiting drug trial and was taking sulfinpyrazone.

The symptoms persisted such that a total of 75 episodes of right-sided numbness and/or weakness without transient dysphasia occurred until the next year. At that time the patient had an episode of persistent dysphasia which cleared up over several weeks. Because of this further episode, a repeat angiogram was carried out. The findings in all vessels were the same except for the stump of the internal carotid artery which looked shorter and narrower (fig. 5c).

It was concluded that this might be a source for embolic ischemic events. As a therapeutic trial he was given heparin for 2 weeks and had no further episodes. Accordingly, the left carotid bifurcation was surgically explored and an arteriotomy was done centered on the bifurcation of the internal carotid artery and carried 1 cm down along the common carotid artery. The internal carotid artery was occluded in a smooth, tapering manner. The external carotid artery was free of significant disease. A plaque was identified in the posterior wall of the common carotid artery which had not been seen in the arteriogram nor could it be identified with hindsight. A common, internal and external endarterectomy was performed. A large metallic clip was placed across the origin of the internal carotid artery to obliterate the blind sac and to prevent further emboli from arising within this stump.

On pathological examination a large organizing thrombus virtually occluded the internal carotid stump and was firmly attached to the atherosclerotic wall. This red thrombus clearly occupied most of the lumen prior to fixation contraction (fig. 6). The common carotid artery contained an elongated ulcer (fig. 7) 5 mm long. The ulcer was almost filled by a layer of old thrombus showing signs of organization with deposits of hemosiderin pigment, cholesterol clefts and multi-nucleated giant cells. This thrombus probably explained why the ulcer crater was not visible in the angiograms taken 1 month previously.

During the next 2 months, the patient’s attacks were reduced from 10 a month to 2 a month and were very transient. After 2 months they stopped occurring, and he has had 4 minor sensory attacks in the subsequent 9 months. These later episodes may have been ischemic or they may have been minor sensory seizures. In an attempt to clarify their nature, ophthalmodynamometry was carried out in the recumbent as well as the erect position. The pressures in the right eye were 70/34 and in the left eye 60/12 but they did not alter between the recumbent and the erect positions on either side.
FIGURE 5. Patient 2, Nov., 1975, demonstrating tapering 1½ cm stump with narrow "waist" indicative of a plaque (arrow) a). The later films b), indicated a hold-up of contrast in the stump and a filling of the carotid siphon (arrow) from the ophthalmic artery. In Aug., 1976, c), stump was shorter and narrower.

FIGURE 6. Same patient (2) as in fig. 5 with constrictive atherosclerosis reducing stump lumen and with organizing thrombus attached to plaque.
Case 9

A 52-year-old female developed episodic hemianopia in November, 1976, experiencing an attack every few days, and on occasions as many as 2 or 3 attacks daily, each lasting approximately 15 minutes. Most involved the left side but occasionally a transient right hemianopia occurred. In early April, 1977, she became aware, after an episode, of a persistent right visual defect. Two weeks later, the left arm and leg were the site of several minutes of numbness. On May 13 the left leg became persistently weak and the right visual field loss increased and persisted.

Upon examination, she had a right upper temporal quadrantanopia, steep and congruous, and involving the point of fixation. This was appropriate to the finding of a cholesterol plaque within the inferior nasal retinal arteriole (fig. 8). Minimal weakness of the left leg dorsiflexors and pathological briskness of the left lower limb reflexes were apparent.

Carotid angiography indicated an occlusion of the right internal carotid artery with an irregular stump 6 mm long. The right external carotid artery was the site of irregular stenosis. The common carotid artery appeared normal. The cavernous sinus portion of the carotid artery was filled through the ophthalmic artery.
and the posterior cerebral, as well as middle and anterior cerebral arteries, were supplied from this reconstituted artery. On the left side, there was complete occlusion of the external carotid artery with minor irregularities in the left internal carotid artery and cross-filling intracranially through the anterior communicating artery to the opposite anterior and middle cerebral arteries.

On May 25, 1977, an arteriotomy was carried out with endarterectomy involving the stump of the internal carotid artery and the external carotid artery. A single, large metallic clip was placed across the origin of the internal carotid artery to obliterate the stump and direct the blood smoothly into the external carotid artery.

The base of the stump was the site of very significant atheroma with 2 sizable ulcerative lesions (fig. 9) showing some evidence of fresh and old thrombus attached to these de-endothelialized areas. There was no ulcer or thrombus in the smoothly stenosed external carotid artery.

Post-operatively, she had no further episodes.

Results of Analysis

The 9 cases were included because all had a demonstrable stump and ipsilateral cerebral or retinal ischemic events appropriate to the territory of the occluded internal carotid artery. In the majority (referred to in the tables as cases 1 through 6) there were proven ischemic events after the artery appropriate to the symptoms was known to be occluded. In the remaining 3 cases (referred to in the tables as cases 7 through 9), the identification of an irregular or ulcerated lesion in the stump was followed at once by definitive therapy, twice surgical and once anti-coagulation. Thus, no further ischemic events occurred after identification of the presumptive cause, leaving the relationship less certain.

The ischemic events occurred in 4 women and 5 men averaging 57 years-of-age and were transient (TIA), persistent, partial, non-progressing stroke (PNS), or both, as depicted in table 1. None was dependent on physical activity nor on change of position, and none was associated with recognized cardiac arrhythmias. All patients were tested for orthostatic hypotension without any evidence of its presence. All ischemic events were focal rather than diffuse. In 4 of the patients, recurrent amaurosis fugax characterized the symptomatology, once as the only type of event, 3 times in association with hemisphere episodes and in 2 of the latter (cases 5 and 9), bright ("Hollenhorst") plaques were identified (fig. 8). The others had hemisphere symptoms, mainly transient paresis, sensory dysfunction and speech disorder.

In the angiograms, the carotid stumps varied in length, 2 measuring 3 mm, 3 were 5 to 10 mm, and the others 15 to 25 mm, with the average length 10 mm. The irregularity in the stump suggested ulcerative atheroma or thrombus in 6 of the 9 cases. In 3 of the patients, serial radiographic examinations were available, with 9 months to 2 years between the examinations and the progressive nature of the lesion was evident in each series. The stump tended to become shorter and narrower and in 2 of the 3 was progressively irregular in shape (figs. 1, 2, 5).

It would be misleading to state that the carotid stump was the sole potential source for emboli to the retina or hemisphere in these cases. It was, indeed, the only visible source in 2 cases, but was accompanied by the addition of significant irregular stenosis of the ex-

![Table 1 Ischemic Events](http://stroke.ahajournals.org/)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Number &amp; nature of attacks after occlusion known</th>
<th>Amaurosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>69</td>
<td>1 TIA</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>53</td>
<td>75 TIA, 1 PNS†</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>40</td>
<td>2 TIA, 2 PNS</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>51</td>
<td>2 TIA</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>53</td>
<td>2 TIA</td>
<td>Yes*</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>59</td>
<td>100 (+) TIA</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>75</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>64</td>
<td>—</td>
<td>Yes</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>52</td>
<td>—</td>
<td>Yes*</td>
</tr>
</tbody>
</table>

*with retinal "bright plaque."
†PNS = Partial Non-progressing Stroke.
ternal carotid artery in 4, by minimal common carotid stenosis in 2, and moderate external and common carotid non-stenosing disease in 1 instance. Contrast material passing retrograde into the opthalmic artery was adequate to generous in 8 of the 9 cases where the films were sufficiently satisfactory for a judgment.

Seven of the 9 patients submitted to surgery. This was done within 2 to 43 days after the last ischemic event. A description of the pathological process identified at operation is in table 2.

The lesions visualized at operation and in the pathological laboratory were consistent in 6 of the 7 patients with an active process of recent and older thrombus formation adherent to ulcerative atheromatous lesions within the stump. In only 1, (no. 3), was the lining of the stump described as smooth. In 3 (nos. 2, 5, and 6), there was additional ulcerative atheroma in the common carotid artery, leading to the stump and in the outflow tract, the external carotid artery. In 1 of these, the pre-operative angiograms failed to demonstrate the ulcerative common carotid lesion (fig. 7). In the other 4 surgical cases, the common and external carotid arteries were the site of minimal smooth and non-stenosing atherosclerotic lesions.

The operative procedure in all 7 instances was a bifurcation endarterectomy, with removal of the diseased intima and some media of the common and external carotid arteries, and either removal of the stump by excision with oversewing of the origin of internal carotid or its obliteration by a large metallic clip placed so as to direct the blood smoothly into the external carotid artery.

The results of the surgery are shown in table 2. As a rule there was cessation of attacks after the procedure, although the follow up period is reasonably short. Two patients have not been totally free of episodes since surgery. In 1, a reduction of very frequent attacks occurred (no. 2) and then, after a 2-month period, they stopped. In the other, (no. 3), they stopped for 6 months, returned with increased frequency for 3 months, and then stopped spontaneously for the last 10 months. The carotid angiograms were repeated early in this final 10-month period and the common carotid artery was occluded about 2 cm above its origin. Proximal common carotid ulcerative atheroma might have caused the return of the attacks and may have been the initiating factor in the subsequent occlusion. In these 2 patients with delayed response, measurements were made of the retinal artery pressures by ophthalmodynamometry and, although the readings on the occluded side in both instances were significantly below the contralateral pressures, no additional differences existed between the recumbent and erect positions.

Two patients were not submitted for operation. One (no. 4) has been followed with only 1 minor episode of very transient weakness appropriate to the occluded artery. If new and significant symptoms, relative to

### Table 2: Pathological Process Identified at Surgery

<table>
<thead>
<tr>
<th>Interval from latest episode to surgery</th>
<th>Stump</th>
<th>Other arteries at bifurcation</th>
<th>Results of surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>43 days</td>
<td>Ulcerated atheroma with loosely adherent thrombus (figure 2)</td>
<td>Minor hyaline changes, common and external carotid arteries</td>
<td>Attacks ceased. Death, unrelated cause, 9 mos. later</td>
</tr>
<tr>
<td>4 days</td>
<td>Organizing thrombus obliterating stump (figure 6)</td>
<td>Unsuspected shallow common carotid ulcer (figure 7)</td>
<td>Attacks reduced and then ceased after 2 mos.</td>
</tr>
<tr>
<td>28 days</td>
<td>Smoothly-lined plaque without ulcer</td>
<td>Minimal smooth atheroma</td>
<td>Attacks ceased but returned in 6 mos., stopped spontaneously - nil for past 10 mos.</td>
</tr>
<tr>
<td>15 days</td>
<td>Shallow ulcer overlying thick friable plaque in posterior aspect of stump. &quot;Grumous material&quot; oozing from this area of wall</td>
<td>Necrotic material in atheroma in common carotid artery</td>
<td>Two weeks later died of myocardial infarction</td>
</tr>
<tr>
<td>2 days</td>
<td>Large ulcer with large amount of friable red thrombus, loosely attached to ragged crater</td>
<td>Moderately large ulcer in posterior wall of common carotid artery containing fragment of fresh thrombus</td>
<td>With 8 months follow-up, no further episodes</td>
</tr>
<tr>
<td>22 days</td>
<td>2 mm. ulcer at point of origin of stump. Similarly an ulcer in distal part of stump. Fresh and old thrombus in stump</td>
<td>Minimal atheroma common carotid</td>
<td>11 mos., no new attacks</td>
</tr>
<tr>
<td>14 days</td>
<td>At base of stump, large ulcerative lesions with fresh and old thrombus within the stump</td>
<td>Smooth stenosis external and common carotid</td>
<td>7 mos., no new attacks</td>
</tr>
</tbody>
</table>
the site of the stump and occlusion develop, surgery is planned. One other will not be explored (no. 7). As soon as the ulcerative stump lesion was recognized by angiography, the patient's recurrent, brief but alarming, hemiparetic symptoms were treated with anticoagulant therapy. Without experiencing further episodes, he died within 18 months of an acute myocardial infarction.

Discussion

Incidence and Length of Carotid Stumps

A patent proximal remnant of the internal carotid artery is a common finding following internal carotid occlusion in atherosclerotic disease. A review of a consecutive series of 74 patients with internal carotid artery occlusion in our institution revealed 26 with no stump, 28 with a stump of 5 mm or less, and 20 with a stump longer than 6 mm. It is possible that the longer stumps are of more importance since the stump was more than 5 mm long in all but 2 instances which presented with related ischemic events.

Biophysical Aspects of Flow in Stumps

In vitro flow studies using glass tubing fashioned to resemble the carotid bifurcation have indicated that the internal carotid stump would be the site of excessive turbulence (fig. 10). This would therefore be an ideal site for platelet aggregation, thrombogenesis, endothelial damage and further aggravation of pre-existing atherosclerosis.

Nature of Ischemic Events

Several mechanisms may be potentially responsible for the ischemic episodes in these cases. Firstly, they might represent hemodynamic attacks. These might be thought to result from alterations in blood flow through precarious anastomotic channels such as might occur with changes in cardiac rhythm leading to altered cardiac output or from postural changes with imperfect autoregulatory mechanisms. None of the patients had evidence of these phenomena. Furthermore, all had focal ischemic symptoms which are rare with impaired cerebral perfusion, even when there is a readily recognizable cause for this condition. None had the diffuse symptoms which characterized hemodynamic phenomena. It has been suggested that "orthostatic cerebral and retinal ischemia" may produce post-occlusion symptoms. In the patients with amaurosis fugax in this series, measurements of the retinal artery pressures were made recumbent and erect and no significant orthostatic changes were found.

Secondly, it is conceivable that some of the ischemic events could have resulted from the fragmentation and embolization of thrombotic material breaking off the soft distal "white tail" of the thrombus located in the internal carotid artery. Ischemia occurring both as a sequel to carotid ligation and following spontaneous thrombotic events has been convincingly portrayed. Evidence is lacking to substantiate this mechanism in this series.

Thirdly, emboli could arise in the stump and pass by the external carotid artery through anastomotic channels, particularly the ophthalmic artery, to lodge in the intracranial arteries. This hypothesis is most attractive for several reasons, one of the most important being the evidence of fresh thrombotic material as well as older organizing and re-canalizing thrombi attached to the irregular walls of the stump in all but 1 of the patients submitted to surgery. The average time to surgery after the last episode was 15 days. The first patient with no visible thrombotic material, on operation, had been free of episodes for 28 days prior to surgery. This was comparable to previous observations made in patients submitted to routine carotid endarterectomy. In 1 series of 52 TIA patients, thrombotic material was detected in the surgical specimens in a mere 21% when 30 days had elapsed from the last event to the surgery. By contrast, there was a 66% chance of encountering thrombus if the last episode had been less than 30 days prior to surgery. In another, and earlier series of 12 patients, thromboembolic material was found if the procedure was done within 6 to 7 weeks of the last TIA, but not if the interval was longer. Another argument favoring the stump as the site of origin for the emboli has been the cessation of further attacks in most of the patients.

![Figure 10. Glass tubes with bifurcation simulating the carotid arteries through which Evans blue dye was circulated. Upper tube has been corked. Fluid passes into and out of corked tube with visible turbulence. (Adapted and reproduced by permission from Dr. A. D. Malcolm.)](image-url)
after surgery. In all these a major lesion capable of producing thromboemboli was present in the stump and in 2 instances it was the only site detected as a possible source.

Finally, emboli might arise in the common or external carotid arteries as well as, or exclusive of the stump. The radiological data and surgical pathology in these patients suggests that both sources may be of etiological consequence. The persistence, or later recurrence, of events in 2 of the patients may indicate that emboli arose from more proximal sites in the common carotid artery, in the innominate artery or from the aorta itself as additional thromboembolic sources.

The finding of retinal "bright plaques" in 2 of the patients is very convincing evidence of embolization. The stump was a very possible source in each of them because of the severity of the pathological process. Amaurosis fugax was remarked upon in 14 or 27 patients with continuing attacks of retinal or hemisphere ischemia despite carotid occlusion previously reported from this center, and has been described in 3 other similar patients recently reported. The external carotid artery was the apparent source and in 1 observation has been associated with platelet-fibrin material observed in the retinal arterioles during the amaurotic event. An occluded internal carotid artery, with or without a stump, thus may be associated with ipsilateral amaurosis fugax and this may be associated with emboli of atheromatous debris or of platelet-fibrin material.

**Therapeutic Significance**

These observations on the carotid stump, as well as previous observations made in respect to symptoms occurring in the territory of occluded internal carotid arteries, indicate that serious ischemic events may occur despite this occlusion. In the identification of the pathogenesis of TIA or PNS in a particular patient, these facts will require consideration. Since the majority appear to be of thromboembolic origin, the planning of anti-thrombotic therapy or surgery should be done with this in mind. Cerebral revascularization by external carotid/internal carotid anastomosis is currently under active study with the possibility of reducing effectively the risk of stroke in patients who have had internal carotid artery occlusion. Prior to embarking on anastomosis the possibility of the stump and/or the associated common and external carotid artery lesions contributing to the patient's symptoms must be borne in mind.

**Conclusion**

The stump of the occluded internal carotid should not continue to be regarded as an unimportant and trifling remnant. Distressing or devastating ischemic events may be directly related to changes within the stump lumen with thromboemboli passing to the retina and hemisphere. At times, it may prove to be the sole source and, at others, may be operating in conjunction with sources of thromboembolism in the ipsilateral common and external carotid arteries.

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