15. Wolman M, Nishimoto K, Klatzo I: The binding of sodium fluorescein to plasma proteins and its implications in the study of the blood-brain barrier (in preparation)
16. Little JR: Personal observations

External Carotid Artery in Internal Carotid Artery Occlusion. Angiographic, Therapeutic, and Prognostic Considerations

ROGER W. COUNTEE, M.D. AND THURAIRASAH VIJAYANATHAN, M.D.

SUMMARY Twenty-three instances of internal carotid artery occlusion occurring with minimal neurological deficit in 22 patients are described. Although each of these patients was referred to the neurosurgical service for evaluation for an extracranial-intracranial microvascular bypass procedure, complete arteriographic evaluations of their cerebrovasculature suggested that alternative methods should be the treatment of choice. For each patient reported the ipsilateral external carotid artery was demonstrated by angiography to be an important source of collateral blood supply to the cerebral hemispheres or retinae distal to the occluded internal carotid arteries. Ten patients with no significant ath erosclerotic narrowing or ulceration of the external carotid artery have remained free of symptoms of cerebral ischemia for 6 to 40 months. In twelve patients who developed delayed recurrent cerebral or retinal ischemia ipsilateral to their internal carotid artery occlusion, there were found obstructive and/or ulcerative plaques involving the common and/or external carotid arteries. Thromboendarterectomy in 11 of these patients gave complete relief of ischemic symptoms during the 4 to 36 months of postoperative follow up. One of these 12 patients refused operation and went on to develop a major cerebral infarction. Angiographic identification of a functionally important external carotid artery ipsilateral to an internal carotid artery occlusion carries considerable prognostic and therapeutic significance.

EMBRYOLOGICAL STUDIES,¹ postmortem dissections,²-³ and angiographic investigations,⁴-¹⁸ have amply demonstrated the collateral connections between the extracranial carotid artery and the intracranial and orbital circulations. In occlusion of an internal carotid artery in the neck, this contribution from the external carotid artery may be vital in preventing cerebral and/or ocular ischemia. This report describes our experiences with 23 such instances of internal carotid artery occlusion which occurred with minimal or no neurological residual. In each patient the homolateral external carotid artery was the major, if not the sole, source of alternative blood supply to the hemisphere or eye distal to the occluded vessel. In these patients the angiographic identification of a functionally significant external carotid artery proved to be of considerable value in determining prognosis and appropriate surgical management.

Patients and Methods

Twenty-two patients with 23 internal carotid artery occlusions were referred to the neurosurgical service for an extracranial-intracranial microvascular bypass...
procedure. Because of specific intracranial arterial flow patterns identified angiographically, alternative procedures were thought to be the treatment of choice. All 22 patients were male and their ages ranged from 39 to 67. All the patients had complete angiographic evaluations of their cerebrovasculature including the aortic arch, the neck vessels, and the intracranial circulation. This was usually done by retrograde femoral or right axillary catheterization with selective catheterization of both common carotid arteries and at least one vertebral artery. Photographic subtraction techniques were routinely used.

**Asymptomatic Internal Carotid Artery Occlusion**

In 10 patients the internal carotid artery occlusion was asymptomatic and remained so throughout a follow up of 6 to 40 months. In each of these patients the common carotid and external carotid arteries, when visualized angiographically, were minimally, if at all, affected by atherosclerotic narrowing or ulceration. In each of these patients the external carotid artery on the side of the internal carotid artery occlusion was dilated. In 8 of these 10 patients the enlarged external carotid artery fed dilated arterial channels in the orbit and a markedly enlarged ophthalmic artery filled in retrograde fashion. Blood flow through the ophthalmic artery, in turn, more than adequately filled the intracranial internal carotid artery and its branches. (See patient 1). In 2 of these 10 asymptomatic patients, the external carotid collateral flow was to the ipsilateral vertebral artery through an enlarged occipital artery branch. Blood flow from the vertebral artery in these patients filled the posterior circulation as well as the ipsilateral anterior cerebral circulation via large posterior communicating arteries (See patient 2).

**Delayed Hemispheric Ischemia Ipsilateral to Internal Carotid Occlusion**

In 11 instances in 10 patients, symptoms of recurrent transient cerebral ischemia occurred several weeks to months after the internal carotid arteries had become occluded. In each the external carotid collateral flow was through dilated orbital anastomotic channels to an enlarged ophthalmic artery which by retrograde flow filled the ipsilateral intracranial internal carotid and its branches. In each instance angiograms revealed obstructive and/or ulcerative lesions in the common carotid arteries and/or the ostia of enlarged external carotid arteries.

**Amaurosis Fugax Distal to an Internal Carotid Occlusion**

In 2 of our 22 patients recurrent monocular blindness occurred several months after a documented occlusion of the homolateral internal carotid artery. In each of these patients the ipsilateral external carotid artery was found on angiography to fill an enlarged ophthalmic artery in retrograde fashion. Obstructive and/or ulcerative plaques were demonstrated in the distal common carotid arteries in question. In neither of these 2 patients did collateral flow to the intracranial circulation depend on the affected external carotid arteries.

**Surgical Management and Results**

In the 10 patients who had asymptomatic internal carotid occlusions and uncompromised flow through external carotid collateral pathways, it was believed that no surgical intervention was warranted. This decision is supported by the fact that these patients have remained asymptomatic throughout the 6 to 40 month follow up (patients 1 and 2). In 5 of the patients with recurrent cerebral ischemic attacks and a compromised external carotid orifice, interesting arterial flow patterns were identified angiographically. In these patients the contribution of the external carotid arteries was so extensive that they not only supplied blood to the intracranial distribution of the obstructed internal carotid arteries, but also to the more proximal portions in retrograde fashion back to the base of the skull (See patient 3). In these 5 patients a prolonged selective angiographic injection technique demonstrated a thread of antegrade flow through “almost totally occluded” internal carotid artery origins with markedly atretic cervical segments. The markedly slowed and minuscule antegrade flow in these internal carotid arteries made little, if any, contribution to ipsilateral cerebral perfusion, but it was apparently important in preserving patency of these diminutive proximal segments. Using the angiographically demonstrated dominant arterial flow patterns to the surgeon’s advantage, common carotid to external carotid artery shunting intraoperatively in these patients resulted in brisk back bleeding into the cervical segments of these internal carotid arteries and facilitated safe reconstruction of these vessels in the neck. Postoperative angiography in each of these patients has documented excellent antegrade flow through normal-sized internal carotid arteries and regression of the preoperative dilatation of the external carotids and their enlarged anastomotic channels. Each of these patients has had complete relief of ischemic symptoms and long-term patency of his vessels has been documented with serial Doppler examinations during 4 to 36 months of follow up. In another 5 patients with delayed recurrent cerebral ischemia, endarterectomy of the common and external carotid arteries was performed (See patient 3). In each patient unobstructed cerebral perfusion via these vessels was demonstrated at postoperative angiography. These patients have remained symptom free in 4 to 30 months of postoperative observation.

The eleventh patient in this group with cerebral ischemic attacks refused operation and went on to develop a major cerebral infarction 8 months later (See patient 4). In the 2 patients with amaurosis fugax, external carotid endarterectomy ended all symptoms. Postoperative angiograms documented improved ophthalmic artery flow. However, no changes in the
intracranial arterial flow patterns were demonstrated (See patient 5). These patients have been followed 12 to 18 months.

In all of the operated patients a common carotid to external carotid shunt was used intraoperatively. There was no surgical mortality or worsened neurological deficit in any of the 12 patients who had surgery. One patient had to be reexplored within hours after external carotid endarterectomy because of an expanding hematoma in the neck. An imperfect suture line at the arteriotomy site was found and repaired and the patient made an uneventful recovery. One patient died from a myocardial infarction 4 months postoperatively.

Patient Reports

Patient 1 (E.C.)

A 47-year-old, white male was admitted to the hospital in July, 1976, with a 2-month history of transient episodes of numbness and weakness in his left hand. Twelve months previously he had had a femoral-popliteal bypass procedure for peripheral arterial insufficiency of the left leg. Carotid angiography at that time demonstrated an asymptomatic left internal carotid artery occlusion. On examination, he was generally healthy and normotensive with a mild right central facial paresis and bilateral carotid bruits. Three-vessel cerebral angiography via a right axillary catheterization revealed an ulcerated plaque at the bifurcation of the right common carotid artery. The completely occluded origin of the left internal carotid artery was again demonstrated. The left middle cerebral artery was supplied entirely by the left external carotid artery via retrograde flow through the ophthalmic artery (See fig. 1). The left anterior cerebral artery was supplied by the right carotid. The right vertebral artery was occluded and the posterior circulation filled by a normal left vertebral artery. There was no angiographically demonstrable contribution to the left anterior circulation by the vertebrobasilar system.

A right carotid endarterectomy was performed in July, 1976. The patient has remained without symptoms referable to either carotid system for 22 months.

Comment

Though we have followed this patient for over 22 months, his left internal carotid occlusion has been documented to be asymptomatic for over 3 years. Since the origin of the left external carotid artery was minimally diseased in this patient, no operation to improve perfusion to the left hemisphere was believed necessary. The functional adequacy of this alternative route via the ophthalmic artery to the intracranial circulation is shown by the length of time that this patient has remained asymptomatic.

An additional 7 patients with a similar clinical and angiographic picture have been followed 12 to 36 months. Their courses have been equally benign.

Patient 2 (W.B.)

A 54-year-old, right-handed, normotensive, white male was admitted to the hospital, July, 1977, for recurrent episodes of monocular blindness in the right eye beginning 4 weeks prior to admission. Selective angiography revealed a highly obstructive lesion at the bifurcation of the right common carotid artery. The left internal carotid artery was completely occluded at its origin. The left external carotid artery was enlarged and filled the left vertebral artery through an anastomosis with its occipital branch (fig. 2). The left

FIGURE 1. Left common carotid angiogram in patient 1. The origin of the internal carotid is completely occluded. The origin of the external carotid (A) is mildly deformed but unobstructed and fills via retrograde flow the dilated ophthalmic artery (B) and the intracranial carotid (C) and its branches. The left anterior cerebral artery was filled by the right internal carotid artery.
vertebral artery, as well as the right, completely supplied the posterior and anterior intracranial circulations on both sides. The left anterior cerebral artery also received blood flow from the right carotid artery. Both vertebral arteries were normal.

A right carotid endarterectomy was carried out. Postoperative angiography showed no change in the arterial flow patterns to the left hemisphere. No other surgical procedure was believed to be warranted. The patient has remained asymptomatic for 12 months since operation.

Comment

This patient illustrates another route by which the external carotid artery may adequately supply blood flow to the anterior circulation as well as to the vertebrobasilar system, or to both. We have encountered this situation in another patient who has been followed for 20 months and has remained asymptomatic. Our favorable experience with these 2 patients encourages us to agree with Corkill et al.\textsuperscript{10} that surgical anastomoses of external carotid to vertebral arteries may substantially augment intracranial blood flow in vertebral artery occlusion. It is conceivable that this type of anastomosis may be beneficial for some patients with internal carotid artery occlusion as well.

Patient 3 (H. L.)

A 57-year-old, right-handed, hypertensive white male was admitted to the hospital in January, 1976, with a 4-month history of several transient episodes of weakness of the right arm and leg and, less often, similar periods of weakness of the left arm. His blood pressure and physical examination were normal. In September, 1975, an aortic arch arteriogram had demonstrated occlusion of both internal carotid arteries and the right vertebral artery. Because of continued symptoms, complete cerebral angiography was repeated. The anterior circulation of the brain was supplied wholly by collateral flow through dilated external carotid arteries on both sides, stenotic at their origins. The intracranial internal carotid arteries and their branches were filled in a retrograde fashion through dilated ophthalmic arteries (figs. 3, 5). There
was no crossover flow or demonstrable contribution of blood from the left vertebral artery to either internal carotid artery circulations.

There was retrograde flow in the right internal carotid artery ample enough to fill it intracranially and its more proximal segments at the base of the skull. With a prolonged selective right common carotid artery injection at angiography a thread of antegrade flow came through a severely stenotic internal carotid artery origin and a markedly hypoplastic cervical segment was visualized. At operation a common carotid to external carotid artery internal shunt was established and resulted in brisk back bleeding from the diminutive, though patent, cervical internal carotid artery. An endarterectomy of the carotid bifurcation was performed and excellent antegrade flow in the internal carotid artery was reestablished. Postoperative angiography 3 weeks later demonstrated that the right internal carotid artery was widely patent with antegrade filling of its intracranial branches (fig. 4). The origin of the external carotid artery was completely open. The ophthalmic artery, as well as the external carotid artery, were both much diminished in size when compared to their preoperative state. There was no crossover filling to the anterior circulation on the left.

Because of continued symptoms of left hemisphere transient ischemia, a left external carotid endarterectomy was performed 4 weeks after the first operation. This completely relieved the symptoms of left hemisphere ischemia. Postoperative angiography demonstrated an unobstructed origin of the left external carotid artery which remained enlarged but with faster flow on serial films compared to the preoperative angiograms (fig. 6). The ophthalmic artery remained enlarged and filled the internal carotid artery despite the normal right internal carotid artery flow.

When reevaluated 4 months later the patient was completely asymptomatic. Doppler studies and oculoplethysmography indicated normal flow in the right internal carotid artery and faster flow in the left external carotid artery.

Comment

In this patient the right internal carotid artery was initially thought to be irreversibly occluded. However, a modified angiographic injection technique helped to visualize a trickle of antegrade flow through its severely stenotic origin and hypoplastic cervical segment. Although this flow contributed little, if anything, to cerebral perfusion, it was important in maintaining patency in the cervical portion of the vessel which allowed its surgical reconstruction. Cerebral perfusion was provided by the ipsilateral external carotid artery and the symptoms of ipsilateral transient cerebral ischemia were believed to be related to changes in the external carotid artery. Collateral flow through the external carotid artery helped to maintain patency of the proximal segments of the internal carotid artery at the base of the skull via retrograde flow. Intraoperative common carotid to external carotid artery shunting maintained cerebral perfusion and enhanced retrograde flow in the hypoplastic cervical portions of the almost occluded
internal carotid artery. Four additional patients with this clinical and angiographic picture have been treated successfully and long-term patency of their reconstituted vessels has been documented by postoperative angiography and serial Doppler ultrasound examinations. These patients have had complete relief of symptoms for 4 to 36 months.

Restoring normal flow in the right internal carotid
artery in this patient caused no change in the symptoms or flow patterns to the left cerebral hemisphere. In patients with recurrent ischemic attacks ipsilateral to an internal carotid occlusion, endarterectomy of a contralateral carotid stenosis has often been recommended. This procedure often fails to relieve symptoms because the symptomatic hemisphere does not receive significant collateral blood supply from the contralateral carotid artery.

Left external carotid endarterectomy in this patient resulted in relief of symptoms of left hemispheric ischemia. Postoperative arteriography demonstrated improved flow to the left hemisphere through an unobstructed left external carotid artery similar to that seen in patient 1. We have managed 4 more patients in this fashion and they have remained symptom free for 6 to 30 months after external carotid endarterectomy. We have paid specific attention to resecting or obliterating the residual "stump" of the occluded internal carotid artery when it could not be reconstructed, as Barnett et al. have advised.

**Patient 4 (L.Y.)**

A 55-year-old, right-handed, hypertensive, white male was admitted to the hospital in August, 1977, with recurrent episodes of transient speech arrest and weakness of the right face and arm for 15 months prior to admission. In November, 1975, he had had a left subclavian to common carotid artery Dacron bypass graft for stenosis of the origin of the left common carotid artery. Six months after the procedure he had recurrent ischemic attacks referable to the left hemisphere and angiography demonstrated occlusion of the graft as well as the left common carotid and internal carotid arteries. He continued to have progressively worsening left hemisphere transient ischemic attacks and repeat angiography was performed in August, 1977.

The right common carotid artery and its bifurcation were widely patent and filled the intracranial internal carotid artery on the right very well. There was modest crossover filling from the right carotid to the left intracranial internal carotid territory. There was no filling of the anterior circulation from the vertebrobasilar system. Though the right vertebral artery was normal, the left vertebral was severely diseased at its origin and throughout its course. Serial films following a left subclavian injection showed the left external carotid artery to be filled by collateral flow from the left vertebral artery and an ascending branch of the costocervical trunk via the occipital artery. Flow through the left external carotid artery, in turn, filled an enlarged ophthalmic artery which then filled the intracranial internal carotid artery on the left in a retrograde manner (figs. 7, 8).

It was believed that surgical augmentation of flow through the external carotid artery might increase blood flow through the enlarged ophthalmic artery.
FIGURE 7. Serial lateral films of the left subclavian injections in patient 4. The external carotid (A) and the left internal carotid arteries are occluded at their origins. The external carotid is filled by collateral flow through an ascending branch of the costocervical trunk (F) and the left vertebral artery (G) through a descending branch of the occipital artery (E). The ophthalmic artery (B) is dilated and filled by the external carotid artery (A) with retrograde flow into the intracranial left internal carotid artery (C) and its branches.

Comment

In this patient the extent of the left external carotid contribution to cerebral blood flow was demonstrated angiographically only with selective catheterization of the left subclavian artery. A subclavian to external carotid artery bypass graft procedure has been reported to relieve symptoms of transient cerebral ischemia in similar situations. 

Patient 5 (J.C.)

A 61-year-old, hypertensive, white male was admitted to the hospital in May, 1977, with a 6-month history of episodic left monocular blindness. Twelve months previously he had had a right carotid endarterectomy and a left internal carotid occlusion had been documented angiographically at that time. His neurological examination was normal. On fundoscopic examination a cholesterol plaque was seen to occlude a superior temporal branch of the left central retinal artery. This had not been noted on the previous admission. Repeat angiography on this admission showed the right carotid system to be widely patent and filling the anterior intracranial circulation bilaterally. The distal left common carotid artery was stenosed at its bifurcation which was worse, when compared to angiograms of a year earlier (fig. 9). There was a ragged stump of the occluded left internal carotid origin. There was visualization of a dilated left ophthalmic artery which filled from the left external carotid artery. There was no demonstrable contribution to the intracranial circulation by the external carotid artery on the left.

A left external carotid endarterectomy, with excision of the “stump” and angioplasty, was performed in May, 1977. Postoperative angiograms showed improved flow through the left external carotid and good visualization of the left ophthalmic artery. The left intracranial carotid artery was faintly visualized. However, no impressive contribution to the intracranial circulation could be demonstrated angiographically (fig. 10). The patient has been symptom free for 18 months since operation.
FIGURE 9. Left carotid angiogram in 1976 (A), and 12 months later (B) in patient 5. The internal carotid artery is occluded close to its origin and only a ragged stump remains (D). The distal common carotid has become progressively more stenosed near the origin of the external carotid (A) which fills the ophthalmic artery (B).

Comment

In this patient, the external carotid artery was shown to supply the ipsilateral ophthalmic artery. No contribution from the external carotid artery to the intracranial circulation could be demonstrated pre- or postoperatively. External carotid endarterectomy resulted in improved orbital blood flow on postoperative angiograms, although the intracranial circulation was unchanged. Operation in this instance was performed to remove a presumed source of emboli to the retina and relieve obstruction to ipsilateral ocular blood flow.

FIGURE 10. Postoperative left carotid lateral (A) and A-P (B) angiogram in patient 5. The origin of the left external carotid artery (A) is widely patent and retrograde filling of the ophthalmic artery (B) is seen. The intracranial internal carotid artery (C) is only faintly visualized and no filling of its branches is seen.

Discussion

In patients with minimal or no neurological deficit after an internal carotid artery occlusion, recurrent ischemic attacks in the ipsilateral eye or hemisphere may subsequently occur. In these situations, surgical procedures that presumably augment collateral blood flow to the brain supplied by the occluded carotid artery have been advocated. When symptomatic internal carotid occlusions coexist with a contralateral carotid stenosis, endarterectomy of the confederate stenotic vessel has been recommended. In patients in whom there is no significant compromise of the contralateral internal carotid artery, external carotid endarterectomy, as well as extracranial to intracranial microvascular bypass procedures, have been advised. Though technical success with these various surgical approaches have been reported to yield beneficial results in many of these patients, the outcome of either of these procedures has too often been variable and disappointing.

Conscription of dormant collateral pathways is probably a safe assumption in patients who survive an internal carotid artery occlusion without a major neurological deficit. Angiographic visualization of these collateral connections is imperative in order to quantify their functional and therapeutic significance. When an extracranial vessel is functionally a significant source of alternative blood supply to the brain formerly supplied by an occluded internal carotid artery, this contribution can invariably be demonstrated angiographically with proper arteriographic techniques. When an extracranial cerebral vessel cannot be demonstrated to contribute substantial collateral blood flow to the intracranial or ocular circulation as seen on preoperative arteriograms, in our experience there has been no significant enhancement of collateral flow after removal of obstructive lesions in the particular extracranial vessel in question (See patients 2, 3, 5).

In each of the 23 patients reported here cerebral angiography identified the external carotid artery ipsilateral to the internal carotid occlusion as a vital
source of collateral blood supply. In 21 patients this was a functionally important alternative source of blood flow to the cerebral hemisphere distal to the internal carotid occlusion, and in 2 it was an important collateral to the ipsilateral ocular circulation. The functional adequacy of this collateral source for cerebral and ocular perfusion has been demonstrated by the length of time that many of our patients have remained asymptomatic after their internal carotid occlusions (See patients 1, 2). When arteriography has shown that the functionally important external carotid artery has unobstructed flow and no source of arterial-to-arterial emboli, subsequent symptoms of cerebral or ocular ischemia distal to the internal carotid occlusions have not occurred in our patients. Though retrograde flow through ipsilateral ophthalmic arteries appears to be one of the more frequent collateral routes, external carotid to verteobasilar connections have also proved to provide ample alternative pathways for cerebral perfusion (See patient 2). Consequently, the angiographic features we have described in patients 1 and 2 have served as valuable prognostic indicators.

It is evident that with some internal carotid artery occlusions, obstruction to flow in the ipsilateral external carotid artery may carry considerable consequences. When associated with occlusion of other extracranial vessels in the cerebrovascular circuit, external carotid obstructions have been reported to cause facial pain, amaurosis fugax, painful ocular ischemia, and transient ischemic attacks in the carotid as well as the verteobasilar systems. Intermittent hypoperfusion through the external carotid artery has often been thought to cause these situations because recurrent ischemic symptoms have been related to head and neck movements. A convincing argument for an embolic phenomena through the external carotid artery has also been made by the observation of retinal emboli distal to documented "old" internal carotid artery occlusions. Ulcerative lesions in the orifice of the external carotid, the common carotid, and/or thrombus in the "stump" of the occluded internal carotid artery are thought to be the source of these embolic fragments.

It is probable that both obstruction to flow as well as emboli through the external carotid artery with internal carotid occlusion play varying roles in causing symptoms. More importantly, relief of recurrent ischemic symptoms in hemispheres, and eyes, distal to an internal carotid occlusion, has been reported after ipsilateral external carotid endarterectomy and suggests that external carotid endarterectomy is indicated in all patients with symptomatic internal carotid occlusion accompanied by obstructive lesions in the homolateral external carotid artery, or the residual "stump" of the occluded internal carotid artery. Our experience prompts us to qualify more rigidly these recommendations. We believe that external carotid endarterectomy can be expected to relieve symptoms of transient cerebral ischemia only when preoperative arteriograms demonstrate that a diseased external carotid artery is a major source of blood supply to the symptomatic hemisphere distal to the occluded internal carotid artery. Similarly, preoperative arteriograms should at least demonstrate opacification of the ophthalmic artery by the external carotid artery, before external carotid endarterectomy can be expected to relieve symptoms of recurrent retinal ischemia distal to an occluded internal carotid artery.

Although in several of the patients described by others the angiograms have shown this contribution from the external carotid to the symptomatic eye or cerebral hemisphere, the importance of this angiographic feature has not been sufficiently emphasized. Deitrich et al. have stated that angiographic visualization of these collateral connections is not a prerequisite for external carotid artery surgery. We believe that detailed arteriographic assessment of the entire cerebrovascular circuit in these patients is important to discriminate objectively among candidates for this, as well as other, cerebrovascular operative procedures.

In internal carotid occlusion which is sustained with minimal or no neurological deficits, conscription of collateral blood flow is a safe assumption. These alternative pathways, however, are variable and their functional significance can be quantified only by cerebral angiography. When the ipsilateral external carotid artery is identified as a functionally significant source of collateral blood supply to the symptomatic hemisphere or retina distal to an occluded internal carotid artery, surgical procedures to remove sources of emboli and/or obstruction to flow in these external carotids may offer considerable benefit.

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R W Countee and T Vijayanathan

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