TWO PATIENTS with symptomatic tandem stenotic lesions involving the carotid sinus and siphon have recently been referred to our neurosurgical department for further evaluation. Both patients had cervical carotid endarterectomy, and their postoperative arteriogram showed resolution of the intracranial lesion. The clinical and angiographic course of these patients may define a subgroup of “inaccessible” stenotic siphon lesions that should not receive microvascular bypass surgery as initial treatment.

**Patient Reports**

**Patient #1**

A 68-year-old man developed left carotid distribution TIAs manifested by left amaurosis fugax and right arm paresthesiae. Three months after the onset of symptoms, an arteriogram revealed tandem stenotic lesions of the left carotid sinus and siphon (fig. 1 a and b). A left carotid endarterectomy was performed 3 months after the arteriogram, during which the patient suffered a mild left cerebral hemisphere infarction. TIAs ceased, however, until 3 months postop, when the patient experienced a questionable episode of transient global amnesia. He was subsequently referred to the University of Florida Hospitals for consideration for a microvascular bypass procedure.

On admission neurologic exam he was found to have a mild residual right arm clumsiness and weakness. Arteriography (performed 6 months after his endarterectomy) revealed left external carotid artery occlusion and mild bilateral atheromatous irregularity of the cavernous carotid arteries. His previous high grade left carotid siphon stenosis had resolved, and no distal vascular occlusions were evident (fig. 1 c). The patient has remained asymptomatic on aspirin therapy.

**Patient #2**

A 51-year-old man experienced a mild left cerebral hemisphere infarction after a 2-year history of episodic left amaurosis fugax and right arm paresis and paresthesiae. An arteriogram (performed one week after the infarction) revealed left carotid sinus and siphon stenosis, with poor left middle cerebral artery filling (fig. 2 a and b). Anticoagulant treatment was initiated, but discontinued when a history of pancreatitis was discovered. The patient was transferred to the University of Florida Hospitals for further evaluation.

On arrival, the patient had slight word-searching difficulties and a mild right central facial weakness. Review of the arteriogram suggested that the apparent siphon stenosis might actually represent embolic disease. Five weeks after the infarction, a left carotid endarterectomy was performed. The procedure was uncomplicated and the patient was discharged on aspirin one week after surgery.

Repeat arteriography, performed 6 weeks after the endarterectomy, showed marked resolution of the carotid siphon defect (fig. 2c), and the patient’s neurologic deficit had improved. No further TIAs have been reported during a 6-month follow up.

**Discussion**

Carotid endarterectomy is a well established and very effective treatment for TIAs caused by carotid sinus ulceration and/or stenosis. The decision to perform a cervical carotid endarterectomy to relieve these lesions, however, is considerably influenced by the angiographic appearance of the carotid circulation distal to the cervical obstruction, as nearly one-third of patients will have concomitant distal “inaccessible” lesions which may increase the risks of this procedure or reduce its effectiveness.1,2

The most common site for these “inaccessible” lesions is the region of the carotid siphon, which is that portion of the internal carotid artery (ICA) that lies between the exit of the artery from the petrous bone and its terminal division into anterior and middle cerebral branches. Atheromatous disease of this segment occurs most often in the cavernous sinus region, proximal to the ophthalmic artery origin. Siphon atherosclerosis has a marked tendency to calcify and frequently can be seen on plain radiographs.3,4 Plaques in this region are usually flat, rather than the more typical bulging atheromatous nodules, in-
FIGURE 1a. AP projection, left common carotid angiogram. Pre-endarterectomy angiogram demonstrates severe atherosclerotic stenosis of the bifurcation and the proximal portion of the internal carotid artery.

FIGURE 1b. Lateral projection, left common carotid angiogram. Pre-endarterectomy angiogram of the intracranial carotid circulation demonstrates severe stenosis of the supraclinoid portion of the left internal carotid artery (open arrow) just distal to the infundibulum of the posterior communicating artery (PCA). There is poor filling of either middle or anterior cerebral arterial branches. Diffuse intimal irregularity of the cavernous carotid is present.

FIGURE 1c. Lateral projection, left common carotid angiogram. Post-endarterectomy carotid angiography demonstrates significantly improved left cerebral flow with ready opacification of intracerebral arterial branches of both anterior (ACA) and middle (MCA) cerebral artery circuits. The focal supraclinoid carotid artery stenosis is no longer evident, while the diffuse atherosclerotic intimal irregularities remain unchanged from the prior study.

dicating that the calcification may impede growth in thickness of lesions at this site. Many siphon lesions, therefore, do not greatly reduce cerebral blood flow, but may provide a substrate for thrombus formation and distal embolization.

With the advancement of microsurgical techniques, isolated carotid siphon and other heretofore "inaccessible" stenotic lesions have become amenable to microvascular bypass procedures. The choice of operative management becomes more complex when the symptomatic carotid artery contains tandem lesions of the sinus and siphon. Under such circumstances, appropriate operation selection might be based on the degree of siphon stenosis. Without significant reduction of distal blood flow, an endarterectomy might be the initial procedure of choice, even though the surgical risks of this group of patients are higher. Patients with high grade siphon stenosis might be considered bypass candidates, as an endarterectomy could not be expected to eliminate the upstream obstruction.

This paper, however, reports 2 post-endarterectomy patients whose "severe" siphon stenosis resolved on postoperative arteriography. Both patients had extended periods of TIAs in the ipsilateral carotid distribution, and both had significant carotid sinus lesions. Both patients had arteriograms within one week of an ischemic episode. The intracranial obstruc-
Pre-endarterectomy cerebral angiogram demonstrates a very irregular lumen and marked stenosis of the supraclinoid segment of the internal carotid artery (open arrow). There is poor filling of the intracerebral carotid branches while external carotid branches are opacified throughout their course indicative of delayed intracranial blood flow. Labelled structures include superficial temporal artery (STA), middle meningeal artery (MMA), middle cerebral artery (MCA).

In both patients affected the siphon distal to the ophthalmic artery origin, and severely impeded blood flow to the ipsilateral anterior and middle cerebral arteries. Each patient had a cerebral infarction, one of which occurred during endarterectomy. Both patients have remained free of further ischemic events following surgery. Although the interval between surgery and arteriography was different in each patient, the final arteriogram showed siphon stenosis resolution and minimal residual arteriosclerotic irregularity at that site in both patients.

Post-endarterectomy carotid angiogram now demonstrates spontaneous resolution of the supraclinoid luminal narrowing and intimal irregularity (open arrow). There is also return to normal arterial filling sequence with dense opacification of middle cerebral vessels (MCA) prior to filling of distal external carotid arteries.
The angiographic identification of major cerebral vessel occlusions and their frequent resolution has been well documented in the literature, and appears to be related to the time interval between the onset of symptoms and the subsequent arteriogram. Earlier angiographic investigation yields a higher demonstrable occlusion frequency.

The proximal middle cerebral artery (MCA) is the most commonly affected site of these transient intracranial obstructions. Fieschi and Bozzal reported that 10 of 17 proximal intracranial occlusions resolved on serial arteriography or at autopsy, and 8 of these transient lesions involved the proximal middle cerebral trunk.8 Alcock et al. also pointed out that many patients with MCA occlusion or stenosis demonstrated significant improvement on repeat arteriography.8

Siphon occlusions appear to resolve with a similar frequency, but are considerably less common, than those occlusions affecting the MCA.8 Since the siphon is so often affected by intracranial atherosclerosis, any obstruction at this site is usually attributed to a large occlusive atheroma with distal thrombosis or vessel collapse rather than embolism. As Alcock et al. suggested, however, the differentiation between atheroma, reactive arterial spasm, embolism, or a combination of factors may be extremely difficult to make from an isolated arteriogram.

This report presents 2 patients with transient siphon stenosis that were referred for consideration of a microvascular bypass procedure. The presence of significant sinus lesions, however, indicated that the siphon stenosis might represent embolic disease. Since lysis of embolic lesions occurs in a substantial number of patients, another arteriogram was performed prior to extracranial-intracranial (EC-IC) bypass in Patient #1. The proven resolution of the lesion, therefore, avoided a second unnecessary procedure. Using experience gained from this patient, the second patient had endarterectomy rather than an EC-IC anastomosis as his primary surgical procedure, and subsequent resolution of the intracranial lesion was demonstrated on postoperative arteriography.

Intracranial occlusion of major cerebral vessels is more frequent in cerebral embolism than in cerebral thrombosis, and the phenomenon of spontaneous clot lysis is almost always associated with embolism.7 The presence of a more proximal extracranial lesion, or the identification of a more distal siphon stenosis or occlusion, therefore, should alert the surgeon to the possibility that an embolus may be responsible for the intracranial obstruction. Repeat angiography, as suggested by Fox,9 may show resolution of some of these lesions, even when they involve the carotid siphon. As preservation of the internal carotid circulation is preferable in most cases, demonstration of a resolving embolic occlusion may avoid a potentially unnecessary bypass procedure and allow subsequent endarterectomy, when indicated, to be performed more safely.

References
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