Surgical Management of Extracranial Carotid Artery Disease

Christopher M. LOFTUS, M.D., F.A.C.S.

Summary: In the last six years the indications for performance of carotid endarterectomy have become standardized by the availability of level one evidence from cooperative trial data. Four randomized cooperative trials for asymptomatic carotid disease and three randomized cooperative trials of symptomatic carotid disease have been completed and published. Asymptomatic carotid disease with 60% or greater linear stenosis on angiography has been shown to be better treated with surgery than with medical therapy alone. For symptomatic patients, linear stenoses of 70% or greater in all patients have been shown to have a significant benefit with surgical treatment. Symptomatic moderate stenosis of <50% is best treated medically, but NASCET now shows that surgery is best for >50% symptomatic stenosis in healthy patients. All surgical recommendations are based on a morbidity/mortality rate of 3% or less for the individual surgeon. Areas remain which were not addressed directly by randomized trials, and for which only lesser levels of evidence are available. These include Hollenhorst plaque, complete occlusion, silent cerebral infarcts, emergency surgery for stroke, and "stump" syndromes. This review discusses the evidence for carotid surgery in these categories, as well as my personal technique for successful carotid reconstruction.

Key words:
- carotid artery diseases
- carotid surgery
- ACAS
- NASCET

Introduction

Since the first description of a carotid endarterectomy for the prevention of stroke,7) the operation has been widely debated and often criticized, yet the numbers of endarterectomy procedures performed annually have steadily increased. Early studies suggested that medical management was superior to surgical intervention.10) This is clearly no longer the case. Gratifying and unimpeachable results from recent multi-center trials have validated the superiority of surgical therapy over medical management in specific cases8)28)30) of both asymptomatic and symptomatic carotid stenosis. The NASCET data, recently completed, indicates that carotid endarterectomy has benefit in all asymptomatic patients with lesions of ≥70% linear stenosis and for specific subgroups of symptomatic patients with ≥50% stenosis. The ACAS trial indicates that asymptomatic patients with ≥60% stenosis have a better outcome with carotid endarterectomy than with medical management.

In this chapter I describe my standard technique for carotid endarterectomy, as well as discussing the various surgical options and different variations of the procedure. Although there are numerous ways to perform carotid endarterectomy, one must adhere to several basic principles of carotid reconstruction. The surgeon must have complete preoperative knowledge of the patient’s vascular anatomy, must maintain complete vascular control at all times, must have sufficient working anatomical knowledge to prevent harm to adjacent structures, and must assure the patient a widely patent repair free of technical errors.

Indications

My policy regarding indications for surgery is to follow the recommendations of the large randomized cooperative trials. I propose surgery for all asymptomatic patients with linear stenosis of ≥60%, since this was group was shown to benefit from surgery in the ACAS trials.8)
The benefit is not as great for women, and the benefit depends on the ability to perform surgery with a combined morbidity/mortality of less than 3%. Patients need to have an expected survival of five years following surgery to benefit from CEA if they are asymptomatic; for this reason patients with malignancies or other life-threatening conditions should not be offered surgery.

Patients with symptomatic carotid stenosis, whether evident as TIA or stroke, benefit from surgery if their angiogram shows ≥50% linear stenosis as measured by the NASCET method. This benefit is greatest in men who are non-diabetic (diabetes clearly increases the surgical risk). According to several carotid carotid trials (ECST, NASCET, and VASST) the benefit is realized immediately, so there should be no delay in operating on such patients.

The benefit of surgery is also greatest in patients who have hemispheric symptoms instead of amaurosis fugax, and patients with plaque ulceration are at higher risk than those with smooth plaques. I offer surgery to all patients with symptoms and ≥50% linear stenosis.

It is still unclear what treatment is best for ulcerated plaques of less than 50%, and for recurrent stenosis, whether symptomatic or asymptomatic. I sometimes offer surgery for deep ulcers of less than 50% stenosis, especially if they have hemispheric symptoms. I offer surgery for recurrent stenosis when symptoms are present, and also for patients who rapidly progress to high-grade stenosis while being followed with serial non-invasive studies. The risk of surgery is of course higher in cases of recurrent stenosis, and the patient must be informed of this.

Surgical Technique

I perform the operation with 3.5× loupe-magnified technique. Microscopic repair of the internal carotid artery, which I have also tried, allows a primary repair that is unquestionably finer and superior to loupe-magnified technique, but which in my experience did not alter the overall patient outcome or incidence of restenosis or acute occlusion. In the ongoing effort to reduce morbidity I have adopted instead universal patch grafting with collagen impregnated Dacron (Hemashield graft), which has essentially eliminated the problem of acute postoperative thrombosis or rapid restenosis. In my opinion the graft procedure is more easily and expeditiously accomplished with 3.5× magnification rather than the microscope. There is no doubt that the suture lines are not as fine with this method, but the added lumen diameter with patch angioplasty ren-
I routinely use general anesthesia with full-channel EEG monitoring. A recent review between my technique and an institutional vascular surgeon using local anesthesia showed decreased incidence of EEG changes and intraoperative shunting with local. However, there was no difference in stroke rate, complication, length of stay, or overall outcome. I prefer general anesthesia for a number of reasons, not the least of which is the controlled environment. Additionally, all commonly used inhalational anesthetic agents and intravenous barbiturates significantly reduce the cerebral metabolic rate of oxygen, giving a theoretical advantage to cerebral ischemia. I keep my patients normocapneic. While there has been much interest in arterial levels of carbon dioxide, in short, non-physiological hypercapnia and hypocapnia provide no cerebral protection. Blood pressure is maintained at normotensive levels with a tolerance of up to 20% increase in systolic pressure. While some surgeons prefer to induce hypertension at cross-clamping if there are EEG changes and then shunting if no improvement is seen in the EEG recordings, I shunt immediately if EEG changes are evident.

I perform monitoring-dependent shunting based on EEG criteria. I use a custom commercial shunt of my own design (Loftus Shunt, Heyer-Schulte Neurocare, Pleasant Prairie, Wisconsin, USA) (Fig. 6). In my experience, I shunt about 15% of carotid endarterectomies. This increases to about 25% if the contralateral carotid is occluded. After the shunt is placed, the monitoring should return to baseline. If this does not occur, the shunt must be inspected for possible kinking, thrombosis, or misplacement. I always auscultate the shunt with a Doppler probe that confirms patency and shunt flow.

Of note, there are also some surgeons who normally practice selective shunting but who advocate shunting all patients who have had recent strokes or reversible neurologic events (due to their belief that intraoperative monitoring is unreliable in the face of recent ischemic events). Whereas I understand their concerns, this has not been my practice.

Almost all carotid surgeons perform patch angioplasty for recurrent carotid stenosis. Many will also use selective patching in cases where the internal carotid is small, where plaque and arteriotomy has extended far up the ICA, or in any similar case where compromise of the lumen and
a high risk for thrombosis is anticipated. I have taken this policy one step further, and for four years have used a Hemashield patch graft primarily in all of my patients.

I administer a single dose of 5000 units of IV heparin to the patient when the carotid is first visualized. Some individuals reverse the heparinization with protamine after the operation. I have not found any benefit in this. For those patients that come to the operating on a continuous heparin drip, I continue the infusion until the arteriotomy closure is finished. It is important to check coagulation studies (ACT) in the operating room for patients who come to surgery fully heparinized. In my experience many of these patients have not received enough heparin and a supplemental bolus does is required.

Tacking sutures to secure the distal intima in the internal carotid artery after plaque removal are considered a great advance by some and deemed unnecessary by others. The concern with tacking sutures is that they may narrow the lumen, but to us this risk seems small compared to the concern of intimal dissection from an unsecured intimal flap. Many surgeons feel that if the arteriotomy is carried far enough to see normal intima distal to the plaque, that the tacking sutures are unnecessary. I strongly agree with an arteriotomy that extends past the plaque, but I am not always satisfied with how the intimal plaque tapers. In recent years, because of negative experiences with plaques that do not feather cleanly when they are pulled down from the distal ICA, I have adopted the use of fine scissors to “trim” the plaque cleanly in the ICA as it is removed. When
this is done tacking sutures are rarely necessary. I estimate that we selectively place tacking sutures in the distal ICA in about 10% of cases.

Space constraints prohibit a full discussion of surgical technique here. The illustrations provide a basic outline of my technique (Figs. 1–12). Several complete references (two in Japanese journals)\(^{14)-26}\) may be consulted for a complete description of surgical technique.

Complications and Special Situations

Complete occlusion

Surgery is generally indicated for cases of acute carotid occlusion if the patient is not so debilitated as to make recovery untenable. Surgery is essentially always indicated for known acute postoperative occlusion, reflecting technical error in most cases. Surgery is sometimes indicated for cases of subacute carotid occlusion, and in cases of chronic occlusion if the possibility of a “string sign” minimally patent vessel (which can usually be reopened) exists, justifying exploration. My surgical technique for complete CCA/ICA occlusion involves opening (or reopening) of the CCA and ICA once the vessels have been controlled. The thrombus is usually seen at the carotid bulb and extending into the distal ICA; in our experience, the ECA is usually patent. Removal of thrombus and associated ICA plaque may establish backbleeding; if not, the ICA can be explored with No. 8 feeding tube cut to a 15 cm length and attached to a 10 cc syringe. The tube is advanced into the ICA, and the syringe is drawn back to establish suction, which often will pull down the distal thrombus at the tubing is withdrawn. If this fails, Fogarty catheters are passed into the ICA, but the risk of establishing a carotid-cavernous fistula with these must be considered. If backbleeding cannot be established, I cleanly ligate the distal and proximal ICA stumps and perform a CCA/ECA endarterectomy and repair. If six hours or more have passed since occlusion, the likelihood of successful neurologic salvage is diminished, and the risk of intracerebral hemorrhage appears to increase.

Stump syndrome

The term “stump syndrome” describes the continuation of ipsilateral ischemic symptoms after internal carotid occlusion due to emboli from the internal carotid intraluminal thrombus that enters the intracranial circulation via the external carotid artery and its collateral blood flow. After strict criteria are met,\(^{13}\) surgical correction is undertaken via a standard common to external carotid endarterectomy. After removal of the thrombus from the ICA stump, I attempt to reopen the ICA and establish backbleeding. If this is not possible (it usually is not), the stump is obliterated with inside out sutures or with external application of large Weck clips. I stress that the internal carotid lumen must be obliterated. A standard external carotid endarterectomy is then performed (we place a common to external patch graft) and the arteriotomy is closed in the usual fashion.

Bilateral CEA

Bilateral carotid endarterectomy runs the risk of extreme swings in blood pressure from concurrent denervation of both carotid sinuses and from risk of bilateral cranial nerve injury. For those patients that require bilateral endarterectomy, I recommend a staged procedure,
with at least a six week window between the procedures. We customarily have the patients examined by an otolaryngologist to rule out an occult cranial nerve injury before the second procedure is undertaken. Unilateral nerve dysfunction in the cervical region is troublesome, but a bilateral one can be disabling. I have on occasion deferred the second surgery due to an occult vocal cord paralysis. When this happens, the patient is maintained on medical management until such time as cord function returns (as it usually does), after which the second side CEA is performed.

Intraluminal thrombi

The problem of surgical timing in patients with angiographically demonstrated propagating intraluminal thrombus remains an open question among cerebrovascular experts. In patients who present with TIA’s (which, in my experience, have always resolved with anticoagulation) and an intraluminal thrombus, I have opted for delayed surgery (at six weeks after repeat angiography) in every case, and have never seen a negative outcome from intercurrent embolization once heparin is instituted.

Likewise there is a small subset of patients with postoperative neurological events (most often a TIA) after carotid endarterectomy who are found to have a fresh thrombus adherent to the suture line (by angiography), partially occluding the artery, and which is presumably the source of embolic phenomena. If there is no other angiographic evidence of technical inadequacy, I have chosen to manage these patients conservatively as well, with full anticoagulation and a six week follow-up angiography. In every case the thrombus has resolved, and there have been no negative neurological outcomes in my series with this plan of management.

Tandem lesions of the carotid siphon

In the NASCET trial symptomatic patients were excluded if the degree of siphon stenosis exceeded that at the carotid bifurcation. The presence of stenotic disease at the carotid siphon has been proposed as a contraindication to carotid endarterectomy because of both the inability to pinpoint the symptomatic source and the reputed increased possibilities of postoperative occlusion from decreased carotid flow velocity. This has not been my experience, and I do not hesitate to operate on patients with tandem lesions if I am convinced that an active plaque at the carotid bifurcation is the source of their embolic phenomena.

Concurrent carotid disease and intracranial aneurysm

There is always a concern that cervical carotid revascularization (for either symptomatic or asymptomatic carotid stenosis, especially high grade) will lead to rupture of a known intracranial aneurysm when both lesions are present. While this is no doubt a small risk, several articles have shown that it is safe to proceed with carotid endarterectomy with a silent intracranial aneurysm discovered on angiography. Obviously, the symptomatic lesion should be treated first. I do not hesitate to operate in the light of an asymptomatic intracranial aneurysm, but I do customarily recommend subsequent craniotomy and aneurysm clipping as well.

Recurrent stenosis

There is a small but finite incidence of recurrent carotid stenosis after primary carotid endarterectomy. I have seen a decrease in my restenosis rate after adopting patching in all of our cases. Piepgras et al. show a symptomatic restenosis rate of 1% with an asymptomatic restenosis rate of 4-5% at a two year follow-up using a patch graft. Aside from technical inadequacies, it has been difficult to identify risk factors associated with recurrent carotid stenosis, although continuation of smoking habits after endarterectomy has proved to be a significant risk factor in several studies, whereas hypertension, diabetes mellitus, family history, lipid studies, aspirin use, and coronary disease may not be as important.

Reoperation for carotid stenosis is a technically difficult procedure. It is associated with significantly higher risks than primary endarterectomy. In our institution, the possibility of reoperation for carotid stenosis is entertained in patients who present with angiographically proven disease and classical neurological symptoms referable to the appropriate artery or with documented progression to severe stenosis while being followed with annual serial duplex examinations.

Conclusions

Cooperative study data is available to support the clear superiority of surgery in the management of both asymptomatic > 60% and symptomatic carotid stenosis > 50%. Carotid artery reconstruction will undergo continued technical refinements. The surgical methods presented here have been successful in producing acceptable postoperative results in a broad spectrum of carotid patients. Minor technical details which may vary among surgeons are probably of little significance. On the other hand, subtleties of technique which may add operative time to the "routine" carotid assume greater importance when difficult
lesions or high exposures are encountered, or when the patient is unstable. The importance of a good outcome under these more difficult circumstances leads me to approach all carotid surgery, no matter how simple it may seem, with the same technical approach. Perhaps the most important factor in assuring technically acceptable carotid surgery is the availability of a skilled cerebrovascular surgeon with a demonstrable morbidity and mortality below 3% and a proper understanding of both vascular principles and cerebral physiology.

**Bibliography**

28. MRC European Carotid Surgery Trial: Interim results for symptomatic patients with severe (70–99%) or with mild (0–29%) carotid stenosis. Lancet 337: 1235–1243, 1991

Surgery for Cerebral Stroke 27 : 1999 13