Cerebral Revascularization in Skull Base Tumors

**Stacey Quintero Wolfe, M.D.,¹ Ramachandra P. Tummala, M.D.,¹
and Jacques J. Morcos, M.D., F.R.C.S., F.R.C.S. (Ed)¹**

ABSTRACT

Skull base tumors involving the carotid artery pose a difficult surgical challenge. The potential for bypass grafting for cerebral revascularization carries inherent risks but may aid in tumor resection and control in those who warrant carotid sacrifice but have inappropriate natural cerebrovascular reserve. We include a review of the literature discussing the indications for carotid resection as part of skull base tumor surgery, indications for cerebral revascularization, balloon test occlusion, graft types and operative technique, complications, and results.

KEYWORDS: Skull base tumor, carotid occlusion, revascularization, saphenous vein bypass graft, radial artery bypass graft

Resection of skull base tumors proves to be a significant challenge when the vasculature becomes involved. Carotid sacrifice and subsequent cerebral revascularization may be entertained when there is neoplastic encasement of the carotid artery. The decision hinges on a comparative balance of the following factors: feasibility of complete tumor resection, natural risk of stroke without bypass, and surgical risks of bypass. After reviewing the literature, we discuss the potential for carotid sacrifice in the setting of skull base tumors, the indications for cerebral revascularization, the screening process and associated risks, the operative nuances of bypass grafting, complications of revascularization, and overall outcomes.

HISTORY

Cerebral revascularization dates back to 1953 when Conley¹ performed the first reported saphenous vein graft (SVG) bypass of the cervical internal carotid artery (ICA) for a tumor of the neck. In 1971, Loughheed and colleagues² performed the first long-segment SVG bypass from the common carotid to the intracranial ICA. Fisch and associates³ and Glasscock and coworkers⁴ pioneered direct vein graft reconstruction of the petrous ICA. Sekhar and colleagues⁵ and Spetzler and associates⁶ both described SVG bypass of the petrous to supraclinoid carotid artery.

Although first described in the setting of cervical neoplasms, cerebral revascularization was

Skull Base, volume 15, number 1, 2005. Address for correspondence and reprint requests: Jacques J. Morcos, M.D., F.R.C.S., F.R.C.S. (Ed), Department of Neurosurgery, University of Miami, 1095 NW 14th Terrace, Miami, FL 33136. E-mail: jmorcos@med.miami.edu.

¹Department of Neurosurgery, University of Miami, Miami, Florida. Copyright © 2005 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel: +1(212) 584-4662. 1531-5010,p;2005,15,01,071,082,ftx,en;sbs00432x.

primarily developed for the prevention of stroke in chronic cerebral ischemia due to carotid stenosis. This practice flourished through the superficial temporal artery-middle cerebral artery (STA-MCA) bypass until it peaked in 1985, when the EC/IC Bypass Study Group⁷ failed to show a reduction in the stroke rate in the surgical group. Improved testing for tolerance to carotid occlusion (balloon test occlusion, [BTO]) has also contributed to reducing the role of cerebral revascularization. In selected cases of skull base tumors or complex aneurysms, however, bypass grafting can be an invaluable technique—if not the only means—to achieve a cure.

INDICATIONS FOR CAROTID SACRIFICE

When there is carotid involvement by a skull base tumor, the first decision is whether there is an indication for carotid resection. Three tumor-related considerations factor into this decision: tumor type, extent of infiltration, and tumor location.

Tumor type is a significant factor in deciding whether carotid sacrifice is warranted. Clinical behavior is more relevant than pure histological grade. A meningioma of the skull base, in spite of histological “benignity,” may impart drastic disability and easily lead to neurological dependency and death in spite of all measures. Furthermore, Muhm and coworkers⁸ state that there is no curative therapeutic option for head and neck neoplasms with the exception of complete tumor removal, supporting the role of carotid artery resection when it is found to be intimately invaded by tumor. However, in patients with benign tumors that are slow growing or already preclude complete resection, tumor control with or without adjuvant radiation or radiosurgery may well be the best therapy, given the patient’s age and extent of disease. We would not consider carotid sacrifice or revascularization in patients with tumors that are

either too slow growing or impossible to eradicate, such as a meningioma with bilateral cavernous sinus invasion. We agree with the opinion of authors who would not perform arterial resections in malignancies where there is no prospect for long-term tumor control.⁹ While en bloc resection, including the carotid artery, may not prolong survival, it does improve regional tumor control which may prevent carotid rupture and other local tumor problems.^{10,11}

Second, the extent of carotid involvement must be considered. “Encasement” of the carotid creates a lesser technical challenge than true “invasion” of its adventitia. Invasive pituitary adenomas tend to encase, while some aggressive fibrous meningiomas tend to invade. Complete tumor resection without carotid sacrifice is therefore still possible with acceptably low risk of recurrence.¹⁰ Meredith¹² found that 81% (33 of 41 patients with benign skull base tumors involving the carotid) were amenable to carotid preservation with simple dissection of the tumor off the artery in the adventitial plane. If dissection proves impossible, “shaving” the tumor off of the artery with a small amount of residual tumor may be an option in a benign, slow-growing lesion or in an advanced malignancy, with postoperative adjuvant radiosurgery or fractionated external beam radiation. Preoperative evaluation of invasion of the carotid is unreliable by computed tomography (CT), magnetic resonance imaging, or conventional angiogram, with the best definitive evaluation at the time of surgery. Carotid sacrifice, at least theoretically, is indicated in all patients with tumor invasion of the carotid wall for whom there is solid evidence that complete surgical resection is superior to subtotal or radical subtotal resection coupled with adjuvant treatment.

Finally, the location of carotid involvement by the tumor plays a part in deciding the role of carotid sacrifice. For example, exposure of the carotid artery to the nasopharynx at the end of tumor removal carries an unacceptably high risk of vascular wall infection and carotid rupture, making this an important indication for carotid resection.^{11,13} Tumors involving the cervical carotid artery are more amenable to sacrifice in that both

proximal and distal control are easily secured. As one proceeds rostrally, however, the complexity of the problem magnifies, as one must deal with branches and perforators, the vascular territories of which are either impossible or impractical to "revascularize." Therefore, the role of revascularization lessens the more rostral the arterial involvement.

INDICATIONS FOR CEREBRAL REVASCULARIZATION

Once the decision has been made to sacrifice the carotid artery as part of the resection of a skull base tumor, the possibility of cerebral revascularization with a bypass graft must be entertained. The indications for cerebral revascularization remain controversial, with options ranging from revascularizing all patients who have undergone carotid resection (universal approach), to revascularizing only those patients predicted (by preoperative testing) to harbor inadequate collaterals (selective approach).¹⁰ The question that must be addressed is whether the risk of carotid sacrifice outweighs the combined risk of preoperative testing and bypass grafting.

Due to the unacceptably high risk of stroke after unselected carotid sacrifice (15 to 40% morbidity and mortality), many techniques have been developed to evaluate cerebrovascular reserve and stroke risk. Carotid stump pressures, ocular plethysmography, somatosensory-evoked potentials, electroencephalography, and, most recently, temporary BTO with or without single photon emission CT (SPECT) nuclear scan or xenon-enhanced CT have all been used with varying success. BTO has become widely used for preoperative evaluation of stroke risk after acute carotid sacrifice (Fig. 1). This usually encompasses a 20- to 30-minute period of carotid balloon occlusion, including 10 minutes of induced hypotension, during which clinical and/or electroencephalogram (EEG) evaluation is conducted and collateral flow is angiographically visualized. The addition of SPECT nuclear scan to BTO improves sensitivity for detecting ischemia but suffers in

Figure 1 Balloon test occlusion: Right carotid angiogram with cross compression of left carotid. Apparent adequacy of collaterals through anterior communicating artery.

specificity (Fig. 2). Xenon-enhanced CT can be added to evaluate cerebral blood flow (CBF), which is useful in stroke risk stratification.

Similar to the historical measurement of carotid stump pressures (> 70 mm Hg indicates adequate collateral flow, while < 55 mm Hg carries unacceptable stroke risk¹¹), Tarr and colleagues¹⁴ have classified stroke risk in patients undergoing carotid occlusion. Patients in Group I have CBF > 35 mL/100 g/min with low risk for infarction after carotid sacrifice. Group II consists of patients with CBF between 20 and 35 mL/100 g/min with moderate stroke risk, and Group III patients have CBF < 20 mL/100 g/min, putting them at high risk for stroke. Sekhar et al⁵ describe a similar classification with the low-risk group (I) being those who tolerated BTO with only mild, symmetric decrease in hemispheric CBF and CBF > 30 mL/100 g/min, the moderate-risk group (II) tolerating balloon occlusion but with marked hemispheric decrease in CBF (< 30 mL/100 g/min), and the high-risk group (III) those who did not tolerate BTO.

While BTO appears to be the most reliable method to predict stroke risk, it is not without its own inherent risks. In a series of 300 patients, Tarr and coworkers¹⁴ quote a 3.7% risk of complications

Figure 2 Single photon emission computed tomography-balloon test occlusion: Same patient as in Figure 1. Definite relative hypoperfusion of left hemisphere during balloon test occlusion.

(including six asymptomatic arterial dissections) with a permanent neurological morbidity of 0.7%. Origiano and colleagues¹⁵ report a significantly higher complication rate of 7%, although all deficits were transient.

Even in those patients undergoing and “passing” BTO, the risk of stroke is still variable. The risk of immediate stroke—the false-negative rate—after carotid sacrifice in Group I patients is as low as 2%¹⁶ and as high as 22%.¹⁵ Drake¹⁶ had only 2 of 114 patients with ischemic complication after carotid ligation for anterior circulation aneurysms. Gonzalez and Moret¹⁷ had a stroke rate of 9% in 22 patients who had passed the BTO. Origiano had 4 of 18 patients who experienced ischemic deficits with a 5.5% mortality rate, highlighting the potential shortcomings of BTO for defining patient selection for carotid revascularization.¹⁵ One must keep in mind that BTO can only attempt to study the likelihood of *hemodynamic* compromise after carotid sacrifice, and cannot predict the risk of the equally—if not more—important *thromboembolic* events.

The risk of delayed ischemic complications is another contributing factor to the risk of carotid sacrifice. Roski et al¹⁸ reported a 1.4% stroke risk per year over a 12-year period after carotid ligation, although a significant number of ruptured aneurysms were included, and many of the complications were in the first year. Oldershaw and Voris followed patients over 5 years after carotid sacrifice with an annual stroke risk of 1.9%, nearly 25 times that encountered in the general population.¹⁹

De novo aneurysm formation is a potential risk of carotid sacrifice. New aneurysms have been found in 1 to 10% of those undergoing carotid ligation, clearly higher than in the general population.¹⁹ Enlargement of pre-existing aneurysms is also well reported. These changes are thought to be from altered flow dynamics in the circle of Willis.

Cerebral revascularization is certainly not without risks of its own. Risks of bypass grafts are related to increased surgical time, temporary vascular occlusion during anastomosis, thromboembolic complications, and therefore risks of anticoagulation, as well as risks of graft occlusion.

Lawton et al¹⁹ report a 10% surgical risk of which 7% is attributed to revascularization. It is important to remember that this is in the most skilled of hands. The surgeon's expertise with bypass grafting must always be factored in when considering surgical risk. The magnitude of tumor resection also plays a large role in the risk of revascularization. For instance, cervical tumors are far easier to bypass than those extending into the upper ICA as bypass grafting to the intracranial ICA is technically more demanding and imparts more risk.

It appears that revascularization is clearly needed in some but not all patients following carotid sacrifice. While temporary balloon occlusion can definitively identify those who will certainly fail carotid sacrifice without revascularization, it can only assess a single, temporal event under controlled circumstances, and consequently is fallible. Nevertheless, BTO is extraordinarily helpful to guide our decision-making and is performed for all those who are being considered for carotid sacrifice. Patients who are young (< 45 years old), in good health (able to withstand a lengthy and/or multiple procedures), and have a potential for cure or excellent long-term prognosis are considered for cerebral revascularization after carotid sacrifice as part of a skull base tumor resection.

GRAFT SELECTION

Graft selection encompasses several factors including desired blood flow, size of proximal and distal

anastomotic vessels, availability of graft material, and the extent of reconstruction needed. Sekhar and coworkers¹³ describe three types of arterial grafts: Type 1, or direct reconstruction by suture or patch; Type 2, or interposition graft; and Type 3, or extracranial-intracranial (EC-IC) bypass graft. For our purposes of bypass grafting in skull base tumors, we will be discussing only Type 3 grafts, although tumors that are more cervically based may be bypassed with a Type 2 interposition graft alone.

Flow rate of the bypass graft is critical for successful revascularization. Normal ICA flow is estimated at 370 mL/min.²⁰ Therefore, a high-flow graft is usually needed to provide adequate cerebral perfusion. The saphenous vein can provide flow rates of 70 to 140 mL/min.¹³ In patients with narrowed carotid arteries secondary to tumor compression or invasion, atherosclerotic disease with ischemia, or a small recipient vessel, a lower flow rate may be necessary to prevent hyperperfusion injury. Radial artery grafts (RAGs) supply "medium" flow rates at ~40 to 55 mL/min. STA grafts are used primarily if there is already some collateral flow or if only distal branches are available for anastomosis. Due to their low flow rates of 20 to 35 mL/min,⁹ they are rarely indicated in carotid bypass for tumor resection (Table 1).

SVGs have been widely used for carotid bypass but must be carefully prepared and anastomosed. As they have a relatively large diameter, size should be carefully considered when harvesting the vein. Size mismatch between the vein and donor or

Table 1 Graft Types

Graft	Type	Flow Rate	Indications	Benefits	Drawbacks
Saphenous vein	High	70–140 mL/min	Carotid bypass to arteries > 2 mm	High flow; easy to access	Susceptible to occlusion; fragile endothelium
Radial artery	Medium	40–55 mL/min	Bypass to arteries < 2 mm; SVG failure	Easier to work with; better flow dynamics	Vasospasm; medium flow
Superficial temporal artery	Low	20–35 mL/min	Bypass to and from distal branches	Good for distal EC-IC bypass when collaterals exist	Low flow

EC-IC, external carotid-internal carotid.

recipient vessels can lead to increased turbulence and premature occlusion. The larger the vessel desired, the more proximal the vein must be taken from the thigh/leg. Endothelial denudation can cause thrombosis and graft occlusion, as can low-flow states.

RAGs have some benefits over the saphenous vein when: a medium flow graft can be used, the recipient artery is < 2 mm, or a previous SVG has failed. Cardiac literature shows better long-term patency rates of RAGs than SVGs. This is because the arterial endothelium can support lower flow rates, causing less turbulence and therefore less chance for thrombosis.²¹ There is also less risk of endothelial denudation and the thinner arterial wall makes the anastomosis technically easier to perform. Vasospasm is the largest risk of RAGs due to the reactive arterial wall, but pretreatment with pressure distention can obviate this problem.²¹

INTRAOPERATIVE MANAGEMENT

Anesthesia support is critical for a successful outcome. Preoperative antibiotics, dexamethasone, and anticonvulsants are given after induction of anesthesia. EEG and/or somatosensory-evoked potentials are monitored through the procedure. Mild hypocapnea is maintained with a PCO_2 of 30 to 35 mm Hg. Mannitol may be used to relax the brain during tumor resection. Mild hypothermia is used, keeping the patient's temperature around 33°C . In preparation for temporary vascular occlusion, propofol and/or barbiturates are used for EEG burst suppression. The systolic blood pressure is raised 20% above baseline. Prior to anastomosis, 1 gram of methylprednisolone may be given to protect the venous endothelium upon exposure to arterial blood.⁹ Two to five thousand units of heparin are also given at this time to protect against thrombosis during temporary occlusion. This may be partially reversed at the conclusion of the procedure if excessive oozing persists.

OPERATIVE TECHNIQUE

Graft Harvest

General principles of harvesting apply to all grafts regardless of location. Longitudinal incisions that follow the course of the graft vessel are used (Fig. 3). The exposed graft is not harvested until the proximal and distal anastomosis sites are exposed. We believe that the graft should be left in its natural position with uninterrupted flow as long as possible to reduce the risk of thrombosis or vasospasm. The vessel is covered with gauze saturated in 3% papaverine solution. Small branches of the SVG or RAG are ligated. We leave a cuff of periadventitial tissue intact around the graft to provide more structural support for the vessel, but it is critical that no constricting fascial bands be left in situ. They often become visible only after distention (Fig. 4). The manipulation required to completely skeletonize the graft may increase the risk of endothelial injury and vasospasm. Only 1 to 2 cm of the proximal and distal ends of the graft are denuded to facilitate the anastomoses. After the graft is harvested, it is flushed and distended with heparinized saline. Leaking points are secured with small silk ties.

The saphenous vein is harvested from the groin and thigh for 20 to 30 cm. If a longer graft is anticipated, the harvest can be extended into the proximal leg. The radial artery may be an appro-

Figure 3 Proximal saphenous vein graft harvesting from left thigh.

priate choice if the recipient vessel of the bypass is less than 2 mm in diameter. It is usually harvested from the nondominant upper extremity after confirmation that adequate collateral blood flow is supplied to the hand through the ulnar artery (Allen's test).

In general, three incisions must be made during these procedures—one for the craniotomy and exposure of the tumor, the second in the neck to expose the ICA, and the third to harvest the graft. We typically secure the patient in a radiolucent head holder, and a femoral artery sheath is placed to prepare for intraoperative angiography. Of course, the final positioning of the patient depends on the cranial approach chosen to attack the lesion. Ultimately, the positioning must allow access to the tumor, the proximal and distal anastomotic sites, and the graft site. We favor a preauricular subcutaneous tunnel created with a 28 French-sized chest tube. The most critical aspect of tunneling is to prevent the graft from torsion or kinking, lest it occlude.

Anastomosis of the Graft

Therapeutic systemic heparinization is not used routinely, although with SVGs, 2000 to 5000 units IV are given once and not reversed. This is not done with RAGs. Adequate exposure of the recipient and donor vessels is required to perform the graft anastomoses. If the recipient site is limited in exposure or if there is a significant size disparity between the graft and the recipient vessel, an end (graft)-to-side (recipient) anastomosis may be more feasible than an end-to-end anastomosis, and this is the most common scenario. A rubber dam is positioned under a segment of the recipient artery that is free of branches. A flexible microsuction device is secured near the anastomosis site to continuously remove excess cerebrospinal fluid and irrigation fluid. Temporary clips are placed proximally and distally on the recipient vessel. Under high magnification, an arteriotomy that matches the size of the graft opening is initiated with a beaver blade and

Figure 4 Pressure distention of saphenous vein graft. Testing for leaks and eliminating constrictive bands.

completed with microscissors. The end of the graft is usually shaped in a “fish-mouth” to achieve an anastomosis “at-an-angle,” usually pointing incoming blood flow toward the proximal end of the recipient vessel. The other purpose of using the “fish-mouth” shape is the prevention of suture line stenosis. An “elephant foot” configuration is the desirable end result (Fig. 5). The recipient artery lumen is irrigated with heparinized saline so that the walls of the vessel are clearly visible. The suture thickness depends on the vessel size, with 10–0 nylon used for distal MCA branches and 8–0 nylon sufficient for the petrous or supraclinoid ICA. After anchoring the fish-mouth of the graft to the recipient artery with a single suture, the more difficult side of the anastomosis is completed first. Usually, this is the back wall of the anastomosis. We typically use a running suture whose loops are initially loose and subsequently tightened in a

Figure 5 Distal saphenous vein graft—M2 end-to-side anastomosis completed.

Figure 6 Distended saphenous vein graft being prepared for tunneling to the neck.

sequential manner prior to tying the end. The front wall of the anastomosis is sutured in a similar fashion with periodic irrigation of the vessel lumen with heparinized saline. A temporary clip is placed on the graft near the distal anastomosis, and the clips on the recipient vessel are removed (Fig. 6).

After tunneling the graft (Fig. 7), temporary clips are placed on the donor vessel, generally in the patient's neck. Other factors being equal, we generally favor the distal common carotid artery as a donor in view of its large caliber, its position proximal to possible atherosclerotic plaques, and mostly because of the relative absence of cerebral ischemia during its cross clamping (due to external

Figure 8 Proximal saphenous vein graft-common carotid artery end-to-side anastomosis completed.

carotid retrograde flow into the ICA). Repeating the steps above, an end-to-side anastomosis is completed proximally (Figs. 8 and 9). Small leaks in either anastomosis can be reinforced with oxidized cellulose, whereas larger leaks require reinforcement with interrupted sutures.

The patency of the bypass is assessed qualitatively by palpation, but more importantly quantitatively by flow measurements. In addition, intraoperative angiography is always used prior to sacrificing the native artery (Figs. 10 and 11). We favor nonstaged sacrifice of arteries to simplify the complexity of anticoagulation management in the postoperative period and avoid spontaneous

Figure 7 Preauricular tunneling with chest tube.

Figure 9 After completion of both anastomoses, flow reestablished through saphenous vein graft.

Figure 10 Cranial view of intraoperative angiogram prior to carotid sacrifice. Bypass is clearly patent.

thrombosis of the bypass graft when no flow demand is introduced.

The wound closure is focused on avoiding compression of the bypass graft. The dura is left open widely around the bypass intracranial entry site. A wide opening is left in the bone flap and the

Figure 11 Cervical view of intraoperative angiogram prior to carotid sacrifice. The saphenous vein graft is clearly seen anastomosed to the distal common carotid.

Figure 12 Bone flap closure. Sufficient craniectomy is created to avoid compression of the graft.

layers of the scalp are closed loosely over the graft (Fig. 12). Finally, a noncompressive dressing is applied to the wound. The pulse of the bypass is rechecked, and Doppler probing is repeated after wound closure (Fig. 13).

POSTOPERATIVE MANAGEMENT

After carotid bypass, the primary goal of postoperative management is to avoid complications and keep the graft from occluding. Coagulopathy is reversed

Figure 13 Final Doppler check of saphenous vein graft flow prior to leaving the operating room.

as needed. Systolic blood pressure, usually supplemented by crystalloids and 5% albumin, is generally maintained between 110 and 130 mm Hg for several days to prevent ischemia and hyperperfusion. Graft pulse and Doppler are performed as part of the routine vital signs. Patients with arterial grafts are kept on 325 mg of aspirin for 1 week, while those with SVGs take it for life. We have encountered two patients with SVGs who spontaneously occluded their grafts at 2 and 5 years postoperatively, respectively, within days/weeks after inadvertent discontinuation of chronic aspirin. Our patients have a formal angiogram during the postoperative period and Sekhar and Kalavakonda²¹ suggest follow-up with magnetic resonance angiography at 3 months and annually thereafter.

COMPLICATIONS

There are several postoperative complications to be aware of before embarking upon a bypass procedure. Most of these are avoidable if exacting care is used during the procedure. Graft-related problems, stroke, vasospasm, and hematomas have been reported.^{9,13,21}

Intraoperative graft occlusion usually occurs due to technical errors. Poor flow with subsequent thrombosis can be caused by poor proximal anastomosis, kinking at the distal anastomosis, or twisting and compression along the tunneled path. Early recognition is the cornerstone of prevention in these cases. Intraoperative angiography (IOA) has proven invaluable in this arena. Sekhar et al¹³ recommend IOA to evaluate filling of intracranial vessels and the speed of flow through the graft with immediate correction of any problems. In their series, 32% of those undergoing IOA had graft problems, 19 of 23 which were corrected with an initial patency rate of 100%. The most common issues were 7 proximal end problems (suture line stenosis, anastomotic kinking), 4 tunnel compression problems, and 3 distal end problems (size disparity).

Late graft occlusions are less common but can also be caused by kinking at the anastomosis and tunneling errors. Endothelial damage of the graft due to poor harvesting or handling and size disparity between the graft and anastomotic vessels leading to turbulence are other pitfalls to avoid. Implanting a vein graft into a small, distal artery may result in occlusion due to inadequate flow demand. Sekhar and Kalavakonda²¹ report accelerated atherosclerosis with delayed occlusion in 2 of 105 patients with SVGs. Spasm of a RAG is also common if the pressure distention technique is not performed adequately. In the case of SVG failure, the graft must be completely replaced. A RAG with vasospasm may be treated with careful angioplasty of the vessel.²¹

Neurologic injury due to ischemia is rare with good operative technique. Early stroke can be due to hypoperfusion, embolism, or perforator occlusion. Prolonged time of occlusion and inadequate brain protection in those with poor collateral circulation result in hypoperfusion and infarction. Damaged endothelium or inadequate heparinization can result in embolic stroke.^{11,13} Prior radiation can increase susceptibility to perforator-related stroke.⁹ Delayed stroke can be caused by graft occlusion due to low flow or hyperperfusion from overestimation of flow needs.

As previously stated, epidural or graft site hematomas can occur due to excessive oozing after heparinization. Seromas and lymphoceles can also occur in the graft extraction site and a postoperative drain may be recommended in cases of problematic graft site hemostasis.⁹ Graft infection and blowout are special considerations specific to revascularization in extensive and malignant skull base tumors. As the graft is often left open to the nasopharynx, the potential for infection exists but has not been found to be a large risk.¹¹ Wright and colleagues¹¹ do report one case of hemorrhage from the graft after having a skin graft placed over the vein bypass without intervening muscle. Adequate interposed tissue is important in those with a history of previous neck dissection and radiation.

RESULTS

Final outcome must be measured by graft patency, stroke rate, and tumor control. Sekhar and Kalavakonda²¹ have the largest series to date including cerebral revascularization for skull base tumors. Of 137 bypass procedures, 83 were performed for cranial base tumors and overall, 105 SVGs and 24 RAGs were used. Their overall graft patency rate was 95.6% with late graft patency of 94% from a graft occlusion 5 years postoperatively. This correlates with the series by Lawton et al (early graft patency 95% and late patency 92%).²⁰ Technical advances have improved patency rates since the series by Sundt and colleagues²² in 1986 with only 84% overall patency. Even within the Sekhar-Kalavakonda²¹ series, intraoperative angiogram significantly improved graft patency. Graft patency was 90% without IOA and 98% after IOA became standard for all patients undergoing revascularization.¹³

The risk of stroke after bypass grafting ranges from 0 to 17%.^{11,12,19,21} In the Sekhar-Kalavakonda series, 23 patients (17.3%) experienced cerebral infarction, 17 in the immediate postoperative period. Wright and associates had one early stroke from embolus and one late stroke after a year without graft occlusion in their series of 20 patients.¹¹ This stroke risk is only slightly lower than that of a selective approach to carotid sacrifice.

As the goal of carotid bypass in skull base tumors is complete resection, tumor control in the long term is of utmost importance. Sekhar et al¹³ resected 53 meningiomas and other benign tumors with total resection in 39 patients, 6 recurrences and regrowths, and 2 deaths, with average follow-up of 4 years. Seven chordomas and chondrosarcomas were included with 3 total resections, 3 recurrences and regrowths, and 2 deaths with average follow-up of 2 years. Eleven malignancies were resected with total resection in 9 patients, recurrence and regrowth in 5 patients, and 6 deaths due to disease progression over an average of 2 years.

CONCLUSIONS

A curative resection for skull base tumors involving the carotid artery is most certainly an achievable goal. For benign tumors, carotid preservation is usually possible. Carotid sacrifice should be considered for any benign tumor that can otherwise be completely resected. Resection of malignancies in patients with a reasonable prognosis should also include carotid resection due to the risk of tumor invasion and local complications, primarily hemorrhage. In our opinion, the resection of benign tumors with a preoperative prediction of a subtotal resection should not include carotid sacrifice, as this adds little to disease control. In addition, patients harboring tumors that are partly in the cavernous sinus, and are of an "adhesive" nature, should be considered for planned subtotal resection and postoperative radiosurgery.

In experienced hands, cerebral revascularization is a safe option. It does, however, incur a surgical risk that does not always outweigh the benefits. The overall stroke risk is slightly lower when "selective" revascularization is used as a strategy versus "universal" revascularization or none at all. We advocate using a selective approach to revascularization when considering carotid sacrifice as part of tumor resection, including those patients who are young with moderate to poor collateral flow and Tarr Group II or III. Patient selection is the most important factor in the success of cerebral revascularization in skull base tumors.

REFERENCES

1. Conley J. Free autogenous vein graft to the internal and common carotid arteries in the treatment of tumors of the neck. *Ann Surg* 1953;137:205-214
2. Loughheed W, Marshall B, Hunter M, et al. Common carotid to internal carotid bypass venous graft. Technical note. *J Neurosurg* 1971;34:114-118
3. Fisch UP, Olding DJ, Senning A. Surgical therapy of internal carotid artery lesions of the skull base and temporal bone. *Otolaryngol Head Neck Surg* 1980;88:548-554

4. Glascock M, Smith P, Bond A, Whitaker S, Bartels L. Management of aneurysms of the petrous portion of the internal carotid artery by resection and primary anastomosis. *Laryngoscope* 1983;93:1445-1453
5. Sekhar L, Sen C, Jho H. Saphenous vein graft bypass of the cavernous internal carotid artery. *J Neurosurg* 1990;72:35-41
6. Spetzler R, Fukushima T, Martin N, et al. Petrous carotid-to-intradural carotid saphenous vein graft for intracavernous giant aneurysm, tumor and occlusive cerebrovascular disease. *J Neurosurg* 1990;73:496-501
7. [No authors listed]. Failure of external carotid-internal carotid arterial bypass to reduce the risk of ischemic stroke. Results of an international randomized trial. The EC/IC Bypass Study Group. *N Engl J Med* 1985;313:1191-1200
8. Muhm M, Grasl M, Burian M, et al. Carotid resection and reconstruction for locally advanced head and neck tumors. *Acta Otolaryngol* 2002;122:561-564
9. Kalavakonda C, Sekhar LN. Cerebral revascularization in cranial base tumors. *Neurosurg Clin N Am* 2001;12:557-574; viii-ix
10. Lawton M, Hamilton M, Beals S, Joganic E, Spetzler R. Radical resection of anterior skull base tumors. *Clin Neurosurg* 1995;42:43-70
11. Wright G, Nicholson R, Schuller D, Smead W. Resection of the internal carotid artery and replacement with greater saphenous vein: a safe procedure for en bloc cancer resections with carotid involvement. *J Vasc Surg* 1996;23: 775-782
12. Meredith S, Shores C, Carrasco V, Pillsbury H. Management of the carotid artery at the skull base. *Am J Otolaryngol* 2001;22:336-342
13. Sekhar LN, Bucur SD, Bank WO, Wright DC. Venous and arterial bypass grafts for difficult tumors, aneurysms and occlusive vascular lesions: evolution of surgical treatment and improved graft results. *Neurosurgery* 1999;44:1207-1223; discussion 1223-1224
14. Tarr R, Jungreis C, Horton J, et al. Complications of preoperative balloon test occlusion of the internal carotid arteries: experience in 300 cases. *Skull Base Surg* 1991;1:240-244
15. Origiano TC, al-Mefty O, Leonetti JP, DeMonte F, Reichman OH. Vascular considerations and complications in cranial base surgery. *Neurosurgery* 1994;35:351-362; discussion 362-363
16. Drake CG, Peerless SJ, Ferguson GG. Hunteria proximal arterial occlusion for giant aneurysms of the carotid circulation. *J Neurosurg* 1994;81:656-665
17. Gonzalez CF, Moret J. Balloon occlusion of the carotid artery prior to surgery for neck tumors. *AJNR Am J Neuroradiol* 1990;11:649-652
18. Roski R, Spetzler R, Nulsen F. Late complications of carotid ligation in the treatment of intracranial aneurysms. *J Neurosurg* 1981;54:583-587
19. Lawton MT, Hamilton MG, Morcos JJ, Spetzler RF. Revascularization and aneurysm surgery: current techniques, indications and outcome. *Neurosurgery* 1996;38:83-92; discussion 92-94
20. Roberts B, Hardesty W, Holling H, Revich M, Toole J. Studies on extracranial cerebral blood flow. *Surgery* 1964;56:826-833
21. Sekhar LN, Kalavakonda C. Cerebral revascularization for aneurysms and tumors. *Neurosurgery* 2002;50:321-331
22. Sundt T, Piepgras D, Marsh R, Fode N. Saphenous vein bypass grafts for giant aneurysms and intracranial occlusive disease. *J Neurosurg* 1986;65:439-450