Following the encouraging and long-lasting results obtained with angioplasty and stenting in the coronary, renal, and peripheral vascular systems, a natural evolutionary step was the application of these new technologies to the cerebrovascular tree. Preliminary clinical series have suggested that carotid angioplasty followed by stenting can be carried out with an acceptable degree of safety and with excellent angiographic results immediately and at 6 months. Although long-term follow-up is not yet available, the low morbidity and mortality associated with these procedures, at least in these early series, make it a viable alternative therapy to carotid endarterectomy (CEA) for a select group of patients.

In this article, we discuss our current indications and diagnostic evaluation for carotid intervention from the viewpoint of neurosurgeons who are experienced in both endovascular and surgical approaches for the treatment of carotid artery disease. In addition, we briefly review the potential applications of angioplasty and stenting to intracranial pathologies.

**Potential Indications for Carotid Angioplasty and Stenting**

Several large cooperative randomized trials have proven the efficacy of CEA for extracranial carotid artery stenosis. According to the study results, however, the benefits of CEA are critically dependent on the surgeon’s rate of perioperative complications. If the perioperative morbidity and mortality exceed 3% in asymptomatic patients and 6% in symptomatic patients, the benefits of surgery are quickly lost.

To evaluate the possible role of carotid stenting for extracranial carotid artery stenosis, it is important to recognize that those patients in whom CEA has been proven beneficial were carefully selected. In the North American Symptomatic Carotid Endarterectomy Trial (NASCET), European Carotid Surgery Trial (ECST), and Asymptomatic Carotid Atherosclerosis Study (ACAS), patients with risk factors that might have created confounding variables in the data analysis were excluded. These factors included age greater than 79 years; heart, kidney, liver, or lung failure; cancer likely to cause death within 5 years; cardiac valvular lesion or rhythm disorder likely to be associated with cardioembolic stroke; previous ipsilateral CEA; angina or myocardial infarction in the previous 6 months; progressive neurological signs; contralateral CEA within 4 months; or a major surgical procedure within 30 days. Thus, a large number of patients with high-risk carotid lesions do not meet the criteria established for enrollment in these studies. For this patient subgroup, the indications and risks of CEA are not yet established. That the results of the above-mentioned studies do not adequately represent the entire spectrum of patients with carotid occlusive diseases is further supported by the observation that although the published mortality in the NASCET series was 0.6%, during the same period, mortality among Medicare beneficiaries undergoing CEA was 3%.

Although widespread use of angioplasty and stenting for the entire spectrum of carotid occlusive diseases is not indicated or recommended, certain patient subgroups could benefit from this technique if it is associated with the low morbidity and mortality suggested by early clinical series. Included in these subgroups are patients with one or more of the following conditions: significant medical comorbidity, recurrent high-grade stenosis, high-grade stenosis with contralateral occlusion, radiation-induced
stenosis, surgically difficult to approach high-cervical stenosis, tandem lesions, and stenosis containing intraluminal clot.

**Patients with significant medical comorbidities.** There is a direct relationship between significant (clinically important) medical comorbidities and complications observed following CEA. This is not only true for non-neurologic complications such as myocardial infarction but also for neurological morbidity and mortality. In a subgroup analysis of the NASCET study, diabetic patients undergoing CEA had a significantly higher perioperative morbidity and mortality (above 10%). Additionally, because patients with significant medical comorbidities were excluded from the previously mentioned CEA trials, the indications for and results of surgery in this subgroup of patients are not well established.

The coexistence of severe carotid artery stenosis and clinically important coronary artery disease presents the physician with a management dilemma. The operative repair of one condition is accomplished at the substantial risk of complication from the other. Coronary artery disease is one of the most important factors in evaluating the perioperative risk of CEA. In addition, when long-term results of CEA were reviewed, coronary artery disease, and myocardial infarctions in particular, were the leading cause of death. Conversely, significant carotid disease places patients undergoing coronary artery bypass grafting (CABG) at increased risk for stroke from hypotension during general anesthesia, air or atheromatous embolization, or both, particularly during cardiopulmonary bypass.

In patients with coexistent clinically significant carotid and coronary artery diseases, there is little debate that revascularization is appropriate for both. The choice of a surgical approach is either a simultaneous procedure or a staged approach in which one operation is performed several days before the other. Published reports on combined CEA and CABG suggest that the risk of stroke or death ranges from 7.4% to 9.4%, which is roughly 1.5 to 2.0 times the risk of each operation alone. On the other hand, patients who undergo CEA as a prelude to CABG, are at the highest risk for complications. In this subgroup of high-risk patients with coexistent severe and often unstable coronary disease in need of prompt myocardial revascularization, angioplasty and stenting of the carotid artery, which avoids the need for a major operation or general anesthesia, represent a valid alternative to CEA. This procedure can be carried out at the same time as coronary angioplasty and stenting or the day before CABG.

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**Figure 1.** (A) Pre-intervention digital subtraction angiography (DSA), left common carotid artery injection, anteroposterior view. The origin of the internal carotid artery (ICA) demonstrates severe stenosis (arrow). The lumen of the proximal ICA is irregular. (B) DSA (left common carotid injection) after injection of 100,000 units of urokinase. The angiographic grade of stenosis is significantly improved (arrow). In the anteroposterior view, the proximal diameter obscures the space between the internal and external carotid arteries. (C) DSA (left common carotid artery injection, lateral view) after high-pressure balloon angioplasty (post-stent deployment). The artery has been uniformly remodeled. Notice the smooth transition at the proximal and distal ends of the stent. (Reprinted with permission from: Guterman LR, Budny JL, Gibbons KJ, Hopkins LN: Thrombolysis of the cervical internal carotid artery before balloon angioplasty and stent placement: report of two cases. Neurosurgery 1996;38:620-624.)
Carotid restenosis. Post-endarterectomy recurrent carotid stenosis has been increasingly recognized because long-term follow-up with noninvasive methods is routinely performed after CEA. Patterns of early and late restenosis are observed after technically successful CEA. Early recurrent stenosis within 24 months after surgery is usually caused by a myointimal fibroplastic reaction, whereas late restenosis results as a consequence of recurrent atherosclerotic formation.

Surgery for recurrent carotid stenosis is technically challenging because dense scar tissue surrounds the carotid bifurcation and its branches, and the planes of dissection are difficult to delineate from surrounding tissue, thus the dissection is more traumatic. In early restenosis with myointimal hyperplasia, there is diffuse thickening of the intima and media with resultant fibrous hypertrophic scarring throughout the CEA site and a definitive cleavage plane is usually impossible to identify. With late recurrent atherosclerosis, the recurrent plaque is often friable and associated with intraluminal clot, which increases the risk of clot embolization during carotid dissection. In addition, the presence of scarring and the lack of a clear plane of cleavage between the recurrent plaque and the underlying media may necessitate excision of the diseased segment and reconstruction with an interposition graft. Because of all of these challenges, the major complication rate of redo CEA approximates 10%, even in the most experienced hands.

Recently, angioplasty and stenting of the extracranial carotid artery has been suggested as a valid alternative to carotid re-exploration for patients with recurrent disease. Angioplasty and stenting obviate the need for dissection, and the presence of periarterial scarring does not pose a problem from the endovascular route because no dissection is required, thus there is no cranial nerve injury (a significant problem in case of re-exploration).

In the past 3 years, we have performed a total of 20 endoluminal revascularization procedures in 16 patients (mean age: 70 years) with recurrent carotid stenosis. The mean interval from the primary CEA was 54 months (range 8 to 202 months). Early in our experience, angioplasty alone was performed to treat 7 arteries; more recently, we learned that, in this subgroup of patients, the addition of stents (13 arteries) prevents the recoil phenomenon that is especially observed with more fibrous lesions. The mean degree of stenosis, measured using strict NASCET criteria, was 73% before treatment and 11% after angioplasty with or without stenting. No major neurological or cardiac complications and no mortality occurred in this patient subgroup. There was only one periprocedural transient neurological event, and one patient developed a femoral artery pseudoaneurysm at the access site. No neurological events ipsilateral to the treated artery occurred in the 13 patients who each had at least 6 months of follow-up (mean follow-up: 20 months). These results and those of other authors indicate that endovascular treatment of carotid restenosis can be safely achieved and that angioplasty and stenting represent a valid alternative to carotid re-exploration in this high-risk group.

High-grade carotid stenosis with contralateral occlusion. The risk of ipsilateral stroke in medically treated patients with high-grade stenosis of the symptomatic carotid artery and occlusion of the contralateral one is 69.4% at 2 years. Although CEA significantly reduces the overall risk of stroke in this cohort of patients, the perioperative stroke risk is high. In the NASCET study, the perioperative risk of stroke or death in the presence of a contralateral carotid occlusion was 14.3%. Carotid shunting is used in 67% to 83% of patients with contralateral occlusions. However, shunt insertion may be technically difficult, leading to an increased risk of embolic stroke. In addition, there is no

Figure 2. An 80-year-old, otherwise healthy, man experienced transient ischemic attacks (TIAs) consisting of left hand weakness. He was started on antiplatelet agents but continued to experience TIAs, the last of which consisted of left hand plegia and lasted 1 hour. Doppler studies suggested possible right ICA stenosis at the level of the bifurcation. The patient was scheduled for cerebral angiography and possible endarterectomy. Cerebral angiography showed an approximately 50% stenosis of the ICA in the neck (not shown). However, intracranial views revealed a hemodynamically significant stenosis of the main trunk of the right middle cerebral artery (arrows). Anticoagulation was initiated, and the patient has been symptom-free for the past 4 months. Angioplasty of the lesion will be considered should the patient become symptomatic again while on coumadin.
evidence that shunting actually reduces the perioperative risk of stroke, and patients with contralateral occlusions have a high prevalence of other significant risk factors, which is not surprising since a high-risk profile is expect-
Figure 4. DSA post-stent deployment, left ICA injection, anteroposterior (A) and lateral (B) views showing faint opacification of the angular artery origin (arrows). (C) Capillary parenchymal blush seen on DSA (parenchymogram) reveals absence of flow in the territory of the left angular artery. (D) and (E) DSA, left ICA injection after local thrombolysis, shows good filling of the large angular artery (arrows). (F) Parenchymogram following thrombolysis reveals re-established flow in the territory of the left angular artery. (See next page for figures 4E and 4F.)
In those patients with more advanced grades of atherosclerosis. Angioplasty followed by stenting in this subgroup represents a valid alternative to CEA, obviating the need for even temporary occlusion in the presence of reduced cerebrovascular reserve.

Radiation-induced carotid stenosis. Human and animal studies have shown that concentrated radiation therapy to the cervical region damages large arteries and leads to an atherosclerosis-like occlusive disease. The spectrum of the disease is variable and not necessarily dose-related. With the increased survival associated with current therapeutic regimens for systemic and head and neck malignancies, radiation-induced carotid stenosis is not infrequently observed.

Patients with symptomatic carotid occlusive disease occurring as a result of cervical irradiation often require treatment because the disease often has a rapid progression. Although successfully treated with direct endarterectomy or bypass of the involved arterial segments, these lesions undoubtedly present surgical challenges. Angiographic findings in this disorder include disproportionate involvement of the distal common carotid artery and lesions that, although confined to the field of radiation, are unusually long and difficult to resect during conventional CEA because of extensive periarterial scarring and fibrosis that obscure the dissection planes. Periarterial scarring, probably related to radiation effects in the microvasculature, is a common finding and makes dissection of the large vessels extremely tedious. In addition, the arterial wall is weakened as the vasa vasorum, which provide nutrients to the outer vessel wall, are extremely vulnerable to radiation-induced damage. Infections and wound problems are increased by previous radiation. Further, the risk of airway obstruction (as a result of endotracheal tube trauma to the fixed, irradiated vocal cords and laryngeal edema caused by surgical dissection in an irradiated field) can be increased in these cases. The potential advantages offered by the endovascular approach for these patients are obvious.

High cervical stenosis and tandem lesions. A very high bifurcation near the skull base, especially in a patient with a short or thick neck, or a long stenotic lesion extending high in the neck is difficult to expose surgically. Dissection of the carotid artery in these cases can be troublesome and at times quite traumatic. These patients should be considered for stenting. High lesions are also more likely to be smoother than their lower counterparts with less atherosclerotic debris and dense calcifications and therefore more suitable for the endovascular approach.

Tandem lesions have long been considered an angiographic risk factor for perioperative neurological events. The coexistence of carotid siphon disease, for example, has been proposed as a contraindication to CEA for fear of postoperative occlusion from decreased flow velocity during endarterectomy. As mentioned, these cases were excluded from the NASCET. Patients harboring such lesions could also benefit from angioplasty and stenting as only a few seconds of occlusion time are needed to perform the procedure. If the distal stenosis is severe, angioplasty of both lesions can be carried out at the same sitting.

Carotid stenosis with intraluminal clot. Patients with intraluminal thrombus superimposed on an atherosclerotic plaque often present with crescendo transient ischemic attacks (TIAs) and have a high-risk of imminent stroke; thus, they need prompt treatment. However, CEA in these cases has a significant perioperative neurological morbidity. In a subgroup of 53 patients enrolled in the NASCET study with intraluminal clots identified by angiography, the 30-day risk of stroke or death was 10.7% in those randomized to medical treatment and
12% in those who underwent CEA.4 The high morbidity in this group is related to the presence of fresh clot and the substantial risk of emboli dislodgement during surgical dissection of the carotid artery. An endovascular approach to these cases may offer a lower risk alternative.

To reduce the possibility of emboli from fresh thrombotic material while crossing the lesion with an angioplasty microguidewire, we inject 100,000 to 200,000 units of urokinase into the lesion; and in a few cases, we have observed widening of the vessel lumen following local thrombolysis, suggesting clot lysis5 (Figure 1). We think that especially in this subgroup of patients local injection of urokinase before angioplasty and stenting increases procedural safety by digesting the loose portion of the thrombus (i.e., friable, fresh thrombus), which has a high potential for distal embolism.

Diagnostic Evaluation and Cerebrovascular Rescue of Patients Undergoing Carotid Angioplasty and Stenting

Patients scheduled to undergo angioplasty and stenting must have a recent baseline neuroimaging study, preferably brain magnetic resonance imaging (MRI), to ensure that post-procedural infarction is not attributed to preexisting disease and to exclude the presence of an intracranial process (e.g., tumor) manifesting with symptoms indistinguishable from TIAs.

Before proceeding with angioplasty and stenting, we obtain an angiographic study of both carotid arteries and the posterior circulation with corresponding intracranial views. We believe that a complete angiographic study provides critical information regarding the presence of the following: collateral circulation, a factor that plays a significant role in determining how to proceed; coexistent intracranial disease that may be responsible for the clinical symptomatology (Figure 2); and intracranial aneurysms that may need to be excluded from the circulation prior to any carotid intervention (Figure 3). In addition, preprocedural intracranial views are particularly useful for baseline comparison should embolic complications occur during the procedure.

The patient must understand the potential complications of the procedure and give informed consent. A detailed neurological examination should be carried out immediately prior to the procedure. It is important for the endovascular surgeon to be familiar with the patient’s neurological status, including level of awareness and basic language function, so that subtle changes in his or her condition will be noticed should they occur during the procedure. Constant surveillance and monitoring of the patient’s neurological status should also be carried out by nurses and other paramedical assistants. The patient should not be over-sedated.

The most feared complication of carotid angioplasty and stenting is cerebral embolism. No matter how carefully the procedure is carried out, a small percentage of patients will experience embolic complications. The same is true of CEA. The difference is that with an endovascular approach one has the ability to immediately diagnose and treat cerebral emboli more effectively than is the case with CEA. Patients are awake during interventional procedures and their neurologic and angiographic status can be closely monitored. In some cases, a thromboembolic phenomenon can be diagnosed by angiography before the manifestation of clinical symptoms.

The key to cerebrovascular rescue for the treatment of a thromboembolic complication occurring during angioplasty and stenting is preparation. There are number of procedures we can perform to prevent these complications, and, in addition, pharmacological agents we can administer in anticipation of possible cerebral ischemia; thus, treatment can be initiated even before a complication occurs.

To minimize thrombotic complications, patients receive aspirin, 325 mg daily, and ticlopidine, 250 mg twice daily, starting 2 days before the procedure. Immediately prior to the procedure, the patient should be fully hydrated and blood glucose should be maintained in a normal range. Elevated blood sugar may potentiate ischemic injury. Other preoperative medications include Atropine and a mild sedative, as well as a calcium-channel blocker (i.e., 60 mg of oral nimodipine) and corticosteroids, which may offer limited protection for cerebral ischemia.6 During the procedure, flush packs should be placed on every intravenous line entering the patient. Double flush technique should be utilized frequently, particularly each time a guidewire is used. Attention to detail will help minimize embolic complications. Factors that will strongly influence the results of iatrogenic cerebral embolism include the timing of treatment or revascularization, characteristics of the embolism (soft clot vs. atheroma), collateral circulation, technology and technique, and localization of the embolus (eloquent vs. noneloquent brain).57

There are significant differences between the coronary and cerebral circulations. Most complications of coronary artery revascularization procedures come from closure of the coronary artery, rather than distal embolization. The run off in the coronary circulation is but a fraction of that in the cerebral circulation. The high flow in the carotid arteries predisposes to embolic events. Even small bits of clot or debris liberated during angioplasty can result in significant neurologic dysfunction if the embolus lodges in vessels supplying eloquent brain. Considering the large size of the carotid artery and its high flow, all efforts must be aimed at minimizing or treating embolic complications. On the other hand, the risk of acute or subacute vessel closure during carotid revascularization is quite small relative to that associated with coronary revascularization procedures.
It is important to have a good understanding of the basics of cerebral blood flow, cerebral circulation, and cerebral ischemia. A thorough understanding of cerebral collateral circulation through the circle of Willis and other anastomotic pathways is important. A great deal can be learned from a thorough three- or four-vessel preoperative angiogram, which may demonstrate significant collateral blood vessels to the compromised hemisphere. In patients with well-developed collateral circulation, an ischemic complication may be well tolerated with no treatment at all. Of major importance, is the need for immediate pre-treatment anteroposterior, lateral, and oblique projections of the involved hemisphere. These images should be placed on a nearby view box during the procedure so that subtle changes and vessel dropout (i.e., missing vessels) can be immediately discerned by simply comparing intraprocedural projections to the preoperative images.

Should any change in the patient’s neurological status occur, an immediate angiogram should be carried out to look for vessel dropout (Figures 4A–C). Comparison to the immediate preoperative angiogram will likely help pinpoint the area of embolic vessel occlusion. The equipment and personnel necessary for accessing and treating an intracranial embolus must be available during carotid angioplasty and stenting if we are to effectively manage embolic complications. Evidence of vessel occlusion necessitates the immediate preparation of a microcatheter and microguidewire and selection of the best projection for identifying the occluded vessel. In the meantime, proximal flow must be established or re-established in the common and proximal internal carotid artery to facilitate access as well as re-establishment of distal flow. If the patient is cooperative and not moving excessively, the microcatheter can be advanced into the intracranial circulation and into the occluded vessel using angiographic road mapping. Local intra-arterial thrombolysis can then be carried out using mechanical as well as chemical disruption of the clot. If these maneuvers are unsuccessful, consideration can be given to intracranial angioplasty and/or other pharmacologic maneuvers such as the use of glycoprotein (GP) IIb/IIIa platelet inhibition. If the patient is becoming uncooperative and moving uncontrollably, consideration should be given to rapid intubation and deep barbiturate anesthesia, which will add a measure of cerebral protection. During the procedure, it may be feasible to intermittently pass a microcatheter through the area of occlusion and temporarily perfuse the distal circulation with oxygenated blood from the femoral artery in order to minimize cerebral ischemia. In most cases, it should be possible to reopen a clotted vessel fairly quickly due to the hyperacute access that is possible with the endovascular approach. A detailed description of these intracranial maneuvers is beyond the scope of this monograph. It is important to recognize, however, that the intracranial circulation is extremely delicate and that extreme care must be exercised to avoid vessel perforation, which would likely result in a major hemorrhage given the lytic agents used during cerebrovascular rescue procedures.

Another important consideration is the routine use of post-operative angiograms immediately after completion of carotid angioplasty and stenting. Side-by-side comparison of the postoperative and immediate preoperative films should be carried out; and if there is any suggestion of missing vessels on the post-operative views, a more detailed neurological examination should be carried out immediately. We recently experienced a case of very subtle speech hesitation that occurred after stent deployment but did not seem significant at the time. However, because the patient’s preliminary neurological examination did not reveal any speech abnormality, we thought that this new, very subtle change could be related to a procedural complication. Angiographic views after stent deployment demonstrated occlusion of the left angular artery (Figures 4A–C). Rapid catheterization of this artery with chemical and mechanical thrombolysis resulted in rapid reopening (Figures 4D–F) of the angular artery with immediate restoration of completely normal speech on detailed neurological examination. In this instance, a minor stroke was avoided simply by paying careful attention to comparison pre- and post-operative angiograms. We believe this patient’s deficit would have subsequently worsened significantly if left untreated.

Cerebral embolization will occur in a small number of patients. If handled promptly and properly, a potential neurological disaster can be avoided. Our experience includes more than 140 patients with 3 TIAs, 2 minor cerebrovascular accidents (by MRI), and 2 procedural emboli lysed successfully with urokinase. Our patients were at high risk for CEA and most would have been excluded from the NASCET and ACAS. None have any sustained neurological deficit. Proper preparation and thorough understanding of the cerebral circulation as well as access to available technology and neuroendovascular skills and techniques of cerebrovascular rescue are essential.

Potential Applications of Stents to Intracranial Diseases

With advancements in stent technology and the availability of stents exclusively designed for neurovascular use, select intracranial pathologies might be successfully treated with such devices. These include intracranial occlusive disease, prevention of acute and subacute vessel occlusion and as an adjunct to intra-arterial thrombolysis for acute stroke, and intracranial aneurysms.

Angioplasty and stenting of intracranial occlusive disease. Stenotic lesions located in surgically difficult areas such as the intracranial carotid artery, the intracranial vertebralbasilar system, and the proximal middle
cerebral artery are responsible for 5% to 10% of ischemic strokes.40 Although different extracranial-to-intracranial bypass procedures (superficial temporal artery to middle cerebral artery, occipital artery to posterior inferior cerebellar artery, and superficial temporal artery to superior cerebellar or posterior cerebral artery) have been advocated for the treatment of stenotic lesions in these areas, they are technically demanding and difficult to perform. Additionally, there is no clear-cut evidence supporting their efficacy.

The potential advantages of endovascular revascularization of these surgically inaccessible intracranial vessels by angioplasty and stenting are immediately intuitive. Previous experience with intracranial angioplasty alone for atherosclerotic lesions has shown some promising results.41-44 However, significant risks related to intimal dissections and subacute thrombosis have been reported;45-46 this is reminiscent of the initial experience with coronary angioplasty prior to the introduction of stents and antiplatelet agents. New stents with improved flexibility specifically designed for intracranial use might improve the efficacy of cranial vessel repair and enhance the safety of angioplasty by reducing subacute thrombosis and re-establishing lumen patency following vessel dissection.47

Angioplasty and stenting in the setting of acute stroke. One more potential application of angioplasty and stenting is for acute stroke related to intracranial vessel occlusion following intra-arterial thrombolysis. Several reports have now suggested the potential advantages of superselective intra-arterial thrombolysis over other methods of delivery. The Prolyse in Acute Cerebral Thromboembolism Trial (PROACT) was a randomized, controlled, double-blind trial that tested the rate of recanalization, safety, and clinical efficacy of local intra-arterial recombinant prourokinase given within 6 hours of stroke secondary to occlusion of the middle cerebral artery.48 Recanalization of the middle cerebral artery was found in 58% of patients given the study drug as opposed to only 14% given placebo, and this was statistically significant. The frequency of intracranial hemorrhage, the most feared complication of acute thrombolysis, was not significantly different in the two groups.

Unfortunately, thrombolytic agents, although effective on thrombus lysis, have a paradoxical effect on platelet activation. There is far more experience in the coronary vessels, and the cardiac literature suggests that rethrombosis occurs more commonly in vessels with significant underlying stenoses.49,50 Thus, in stenotic vessels, immediate or delayed reocclusion after successful revascularization is a major concern. This phenomenon has been observed in the anterior circulation as well as in the posterior circulation: the incidence of reocclusion after successful vertebrobasilar recanalization is as high as 30% and is uniformly fatal.51 Therefore, a select subgroup of patients with acute ischemic stroke, namely those with an atherosclerotic lesion with superimposed acute thrombosis, is likely to benefit from the addition of stenting to emergency thrombolysis.

Another consideration is the potential use of GPIIb/IIIa antiplatelet blockade in association with thrombolysis, angioplasty, and stenting. Rethrombosis is initiated by platelet activation and may be minimized by platelet inhibition. The benefits of GPIIb/IIIa inhibition must be weighed against the risk of intracranial hemorrhage.52

Occlusion of the ostia of small perforating vessels during angioplasty and stenting, with the obvious risk of ischemia or infarction in branches of the basilar, anterior, or middle cerebral arteries, is a poorly understood risk and may limit stent applicability in the cerebral circulation. The feasibility and relative safety of intracranial angioplasty has been reported by several investigators.53-56 Additional experimental and clinical experience suggest that the risk with intracranial stents is only a theoretical one. In experimental studies, lateral carotid branches that mimic the size and angle of origin of human perforating branches tend to remain patent after stenting.54 Additionally, in one patient with a fusiform aneurysm of the basilar trunk a segment notoriously rich in critical small perforating branches) treated by stenting, no problems with perforating branch occlusions were reported.55

Intracranial aneurysms. The primary objective in the treatment of intracranial aneurysms is exclusion of the aneurysm from the circulation to prevent aneurysm rupture and/or growth, without compromising flow through the parent artery. With the recent approval of Guglielmi Detachable coils by the United States Food and Drug Administration, endovascular treatment is now a valid alternative to direct surgical exclusion for high-risk patients.54 Advantages of endovascular therapy include increased accessibility to areas with difficult and demanding surgical access such as the basilar trunk and a “minimally invasive” approach that allows treatment of the aneurysm without the need for craniotomy and brain dissection. With increasing experience, however, several limitations of this appealing approach have emerged. Included are frequent aneurysm revascularization because of coil compaction, inability to pack tightly wide-necked aneurysms without the risk of compromising the parent artery, and the continued risk of bleeding in incompletely occluded aneurysms.

With the development and refinement of endovascular stents, the potential of these devices in the treatment of intracranial aneurysms has become apparent and their use for this application has been tested in several experimental studies.55-58 A few clinical cases
of aneurysms of the cervical internal carotid artery, vertebral artery, basilar artery, and intracranial internal carotid artery treated by stent placement have been reported, suggesting that this approach is a feasible, safe alternative to surgical exclusion. Stents effectively treat intracranial aneurysms by diverting flow from the aneurysm orifice, thereby promoting stasis and inducing thrombosis within the aneurysm. Additionally, the aneurysmal sac can still be filled with coils by introducing a microcatheter through the stent mesh. In this situation, the stent prevents herniation of the coil mass into the parent vessel. Thus, this technique has the potential of allowing even large, wide-necked aneurysms (notoriously the ones that are hardest to approach surgically) to be tightly packed. Further, stent placement in this setting may prevent coil compaction by diverting the inflow hemodynamic forces.

Conclusions

As new technologies and operative experience evolve, it may be reasonable to consider carotid angioplasty and stenting as an alternative to CEA for high-risk patients in whom the complications of CEA may exceed the potential benefits. This includes patients at risk for carotid occlusion, prior cerebral infarction, or tandem lesions, those with concomitant unstable angina requiring surgery or other significant medical comorbidities, those with intraluminal clot, and those with lesions difficult to access surgically (e.g., very high cervical stenosis, radiation-induced stenosis, restenosis). The endovascular technique is well tolerated as it can be performed with minimal sedation and without general anesthesia, affording easy communication with the patient and serial assessments of neurological status and avoiding anesthesia-related complications. Because no postoperative anticoagulation is required, patients are usually discharged after 24–48 hours.

An adequate radiological workup includes an axial imaging study, preferably a brain MRI to exclude an intracranial process that at times can present with transient neurological deficits, and a complete angiogram of both carotid arteries and the posterior circulation with proper intracranial views.

Significant potential for improvement of the devices employed exists. The catheter, balloon, and stent technologies used in preliminary studies were all adapted from coronary, renal, or peripheral vascular applications; none were specifically developed for carotid use. Future developments in stent technology may produce devices suitable for treatment of intracranial pathological processes such as occlusive disease and aneurysms, and prevention of acute or subacute vessel occlusion following successful thrombolysis.

References

Panel Discussion

JIRI VITEK: I want to welcome Dr. Hopkins first, the cardiologists, and the vascular surgeons, radiologists, neuroradiologists and the International Andreas Grünzigt society. It is so important to hear a neurosurgeon’s perspective. From his presentation, I understand that this is a more disciplined approach to carotid stenting. I think it is very important to form a team having somebody who can immediately go ahead with the universal rescue and these things. It is the first lecture presentation about carotid stenting and we now have a little...
opportunity to talk about it. Do you have some questions for Dr. Hopkins?

THOMAS MCNAMARA: I just have one question having to do with the treatment with thrombolysis before you would stent. That is an easy question but it would take a long time to answer, but which patients do you do that on without question? What is your end point? If it is improving appearance, do you continue on until it stops changing?

NICK HOPKINS: First of all, it is very controversial. Lots of people don’t believe in it, but we give local intra-arterial urokinase in certain cases, for example, let’s say you have a patient referred with carotid stenosis and he has had a Doppler study and then a repeat Doppler and the plaque characteristics have changed. If you have a previous angiogram, and a follow-up angiogram or an angiogram at the time of your procedure and you see that the plaque has actually changed its morphology in a period of months. The patient who has a very very ugly irregular lesion, the patient with a big ulcer, these are the people in whom I know, at surgery I will find a fresh clot in the plaque. So what we do is just infuse some urokinase, usually 100–200,000 units of urokinase with a microcatheter right into the lesion. And then if we see any significant change in the plaque, we infuse another 100,000 units and we will go as high as 3 or 400,000 units if we need to. I’ve never gone more than 300,000 so far, because you get to a point where you don’t see any more change in the plaque and then we go ahead with angioplasty. So we are not going to get rid of all the clot, but if we lyse the loosest, most dangerous part of that clot, then I think that is a possible benefit. Last week I treated a patient who had a stroke in evolution and whom the neurologist was really scared to treat. They first asked for an endarterectomy, because they felt a stent was too dangerous. If I do an endarterectomy in the face of a known symptomatic fresh thrombus what is my risk of major morbidity? It has got to be in the 10–15% range in that particular kind of patient. The patient already had a fixed neurologic deficit and we opened his severe carotid stenosis up with a stent and within an hour the patient was neurologically normal. That kind of patient with stroke in evolution I would almost always stent rather than do endarterectomy because they are very often associated with a fresh clot. They are often in the process of thrombosing the carotid artery.

TED FELDMAN: Recurrent Symptoms?

NICK HOPKINS: Recurrent symptoms, absolutely. That’s the kind of patient who is symptomatic and has clearly got something loose in that plaque and we know that most TIAs are embolic and I think that is a good patient to consider some thrombolysis pre-angioplasty.

FRANK CRIADO: Would you do that on a patient who is not a high risk for surgery as you define it?

NICK HOPKINS: If the patient is not a high risk and is not symptomatic and doesn’t have ..... FRANK CRIADO: Symptomatic or not, just a garden variety carotid patient, not a particularly high risk for CEA, would you still do this including urokinase?

NICK HOPKINS: We are basing it more on the angiography appearance and the clinical history of multiple TIAs.

FRANK CRIADO: So the answer is yes, you still would do it?

NICK HOPKINS: Yes. if the patient is having recurrent symptoms or has a markedly irregular plaque.

FRANK CRIADO: Do you talk to these patients about the totally unproven experimental nature of such strategy?

NICK HOPKINS: I’m very comfortable, because I can say to the patient “Look I’ve done hundreds and hundreds of endarterectomies and I know that in the kind of patients that I’m recommending angioplasty, endarterectomy is a higher risk”. If the patient has a risk that I think is greater than 6%, then statistically I cannot justify endarterectomy and in that patient I think angioplasty stent has a better risk.

FRANK CRIADO: Is your institution nervous about the medical legal risk of what you are doing?

NICK HOPKINS: Well it’s all done under IRB protocol and I’d say, yes, they are nervous and so am I, but on the other hand, I’m very comfortable in that I have either procedure to offer. I don’t have any axe to grind and I don’t have any financial incentive either way, so I try to recommend the procedure which I feel is safest and best for the patient.

FRANK CRIADO: You don’t get paid for carotid stenting in the US.

NICK HOPKINS: If I do it in a study, then I’m going to get paid. And if the patient is not medicare, I’m going to get paid.

JUAN PARODI: We do open procedures, and also stenting and I had quite a different experience.
TED FELDMAN: Just a brief question regarding pre-treatment with anticoagulant drugs. I think that pre-treating patients with either ticlid or coumadin for a few weeks prior to elective carotid stent implantation may achieve the same thing. Have you taken that approach as an alternative?

NICK HOPKINS: Yes, we pretreat these patients with aspirin and ticlid.

MODERATOR: No, I would like to make a comment about the comment of Dr. Parodi. I would like to point it out that national experience with carotid endarterectomy in Argentina is not as low as that. There are mortalities, maybe 10%. The excellent experience personally of Dr. Parodi is not the whole experience of the vascular surgeon in Argentina.

MICHAELE COWLEY: I just wanted to comment that as a matter of historical perspective it is worth remembering that 18 years ago we had these same discussions about bypass surgery versus PTCA. I'll stop there. Thank you.

SPEAKER: Actually bypass surgery was not proven then and it isn't proven now. So it is a totally different scenario.