Proposed Paradigms for Patient Selection and Procedural Technique

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Abstract—Carotid artery stenting, compared with carotid endarterectomy, is emerging as an effective and less invasive method of revascularization for extracranial carotid artery stenosis. Carotid stenting is established as the treatment of choice for certain high-risk patient subsets, and ongoing clinical trials are evaluating this method across a broader clinical spectrum, including asymptomatic patients. For carotid stenting to reach its full potential, an acceptable risk of perioperative complications, particularly in low-risk patients, must be ensured (the “3% rule”). The present article provides an in-depth review of carotid stenting, with special emphasis on the process of risk stratification pertaining to clinical, anatomic, and procedural considerations necessary to optimize procedural safety and patient outcomes. (Circulation. 2006;113:2021-2030.)

Key Words: carotid arteries ■ prevention ■ prognosis ■ stents ■ stroke

Carotid artery stenting is now widely utilized worldwide as a less invasive alternative to carotid endarterectomy (CEA) for the prevention of stroke caused by extracranial bifurcation carotid artery stenosis. Recent observational and randomized studies have shown that the risk of procedure-related stroke and death is comparable when skilled operators perform these interventions in well-defined patient subsets. Large-scale, multicenter, randomized trials are in progress to assess the broad-based applicability of carotid stenting to the community at large (Carotid Revascularization Endarterectomy versus Stent Trial [CREST] and the Asymptomatic Carotid Stenosis Stenting versus Endarterectomy Trial [ACT 1]). In this context, a subset of particular importance is that of asymptomatic patients with severe carotid artery stenosis. Considering that the Asymptomatic Carotid Atherosclerosis Study (ACAS) and Asymptomatic Carotid Surgery Trial (ACSST) have demonstrated the superiority of surgical revascularization over medical therapy in asymptomatic patients with carotid artery stenosis, it is noteworthy that the initial studies of carotid stenting that led to device approval by the US Food and Drug Administration and third-party reimbursement in the United States focused on patients considered unsuitable for CEA owing to high surgical risk. With the rapid evolution of catheter-based techniques for carotid revascularization, carotid stenting has become feasible in a wide spectrum of patients, particularly in the treatment of asymptomatic patients, requires definition. To this end, the “3% rule” has been coined by one of the authors (S.L.) to ensure case selection that results in 30-day complication rates of <3%. In this article, we focus on patient selection and technical considerations that are prerequisites for procedural safety and are necessary for the next generation of randomized clinical trials designed to study this therapeutic modality in asymptomatic patients.

Historical Perspective

Carotid revascularization, initially by CEA, was introduced in early 1950s as a method to prevent stroke due to atherosclerosis of the carotid bifurcation and internal carotid artery (ICA). At least 4 prospective randomized trials have demonstrated that CEA compared with medical therapy reduces the risk of stroke in patients with carotid artery stenosis, with the magnitude of clinical benefit dependent on symptom status, lesion severity, and the risk of surgery-related complications. Although perioperative death and stroke rates were low in the highly selected patients enrolled in these trials, the risk for other complications causing significant morbidity was not negligible. For example, in the North American Symptomatic Carotid Endarterectomy Trial (NASCET), cranial nerve damage and serious medical complications occurred in 5.6% and 8.1% of patients, respectively.

Effective experimentation with carotid angioplasty began in the mid 1970s and rapidly developed during the subsequent 2 decades. The contemporary era of carotid stenting began in 1994 when Roubin et al instigated the first rigorous, prospective study of carotid stenting entailing independent neurological evaluation at baseline and at 30 days after procedure. This study, and the experience of others, demonstrated that from the outset, carotid stenting performed by experienced operators produced acceptable outcomes. Although stenting compared with balloon angioplasty signif-
ificantly enhanced the efficacy and safety of percutaneous carotid revascularization, the development of embolic protection methods provided the answer to the problem of athero-embolism from the intervention site. From the early description by Vitek et al.20 of innominate artery angioplasty with occlusive balloon protection of the common carotid artery (CCA), through pioneering work by Theron et al.21 and Henry et al., distal23 and proximal24 antiembolic protection technology has developed rapidly. The availability of multiple embolic protection systems has been shown in many single and multicenter registries to confer a remarkably low risk of embolic complications after carotid stenting.25–29 Thus, the feasibility of carotid stenting, its simplicity compared with CEA, and the low morbidity afforded by distal protection devices have accelerated the acceptance and utilization of this procedure.

**Short-Term Outcomes and the Impact of Embolic Protection Devices**

Periprocedural neurological complications, the major determinant of the risk-benefit ratio of carotid stenting, are due primarily to embolization of friable atheromatous material from the aortic arch or the carotid lesion that has undergone intervention.28,30,31 Embolic protection devices (EPDs) that eliminate liberated atheromatous debris from the circulation have had a significant impact on the safety of carotid stenting. A number of such protection devices have recently been introduced and are under clinical evaluation.21 Our group’s experience in 1358 carotid stent procedures was reported recently.32 In a prospective registry, carotid stenting procedures with (n=538) versus without (n=775) embolic protection were associated with lower 30-day rates of any stroke (1.9% versus 5.8%, respectively; P=0.0003) and stroke or death (2.4% versus 6.5%, respectively; P=0.001). Utilization of embolic protection was the strongest multivariate predictor of freedom from periprocedural stroke. The impact of EPD use on stroke risk was most pronounced in patients >80 years old (n=220), in whom 30-day rates of any stroke or major stroke were significantly lowered by EPD use (6.6% versus 15.4%, P=0.02, and 0.8% versus 2.3%, P<0.001, respectively). In the European Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte (ALKK; n=1483) registry,33 use of an EPD (n=668) compared with no EPD (n=815) was associated with significantly lower in-hospital rates of stroke (1.7% versus 4.1%, P=0.007) and stroke or death (2.1% versus 4.9%, P=0.004). A meta-analysis of earlier studies has reported similar findings.34 Thus, embolic protection should be considered the standard of care in carotid stenting. When use of an EPD is precluded by anatomic factors, alternative treatment strategies (CEA or medical therapy) must be strongly considered.

**Long-Term Outcomes**

Numerous studies have shown that late neurological events and restenosis after carotid stenting are rare. In the SAPPHIRE trial (Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy), the 1-year rate of major ipsilateral stroke after carotid stenting was 0.0% (versus 3.5% with CEA, P=0.02).4 Roubin et al.1 prospec-tively followed 528 patients after carotid stenting over a 5-year period. Follow-up, available in 99.6%, ranged from 6 months to 5 years (mean 17 months). With 30-day events included, long-term freedom from any ipsilateral strokes was 95%. Freedom from any ipsilateral stroke after the first postprocedural month was ∼99%, and the rate of restenosis that required reintervention was only 3%. Gray et al.35 performed clinical follow-up and serial imaging studies in 136 patients after carotid stenting, demonstrating angiographic restenosis in 4 patients (3.1%) at 6 months and an additional 2 cases between 6 and 12 months, with no further restenosis or any major ipsilateral strokes at 2-year follow-up. Bosiers et al.36 recently reported the long-term outcomes of 2167 patients undergoing successful carotid angioplasty (stenting rate ∼95%). At 5-year follow-up, ∼85% of patients were alive and free from ipsilateral stroke, with restenosis rates <4%. Although the data await publication, other high-volume centers have also witnessed low rates of late adverse events, with long-term freedom from death, ipsilateral major stroke, or restenosis in excess of 95% (K.D. Mathias, MD, unpublished data, 2005). The available data thus demonstrate that in a broad spectrum of patients, the excellent early results of carotid stenting are durable in the long term.

**Indications**

Candidates for carotid revascularization include patients with symptoms attributable to an ipsilateral carotid lesion and asymptomatic patients, usually diagnosed as the result of a screening procedure. In general, the indications for carotid revascularization relating to symptomatic status and lesion severity are similar for the endovascular and surgical strategies (Table 1).37–40

It is becoming evident that carotid stenting is particularly suitable for certain patient subsets characterized by specific clinical or anatomic features. The randomized, multicenter SAPPHIRE trial compared CEA with carotid stenting utilizing EPD in 334 patients considered at high risk for open surgical intervention because of coexistent vascular disease or nonvascular comorbidities.4 Enrollment required lesion diameter stenosis ≥50% in symptomatic patients or ≥80% in asymptomatic patients (the latter accounting for ∼71% of the trial population). By intention-to-treat analysis, 1-year rates of the individual major adverse event end points were lower with carotid stenting than with CEA (death [7.4% versus 13.5%, P=0.08], major ipsilateral stroke [0.6% versus 3.3%, P=0.09], and myocardial infarction [3.0% versus 7.5%, P=0.07]), although these differences did not attain statistical significance. However, the composite end point occurred significantly less frequently with carotid stenting than with CEA (12.2% versus 20.1%, respectively; P=0.053). At 1 year, the requirement for repeated carotid revascularization procedures was lower in patients treated with stenting than in those treated with CEA (0.6% versus 4.3%, P=0.04). Nota-bly, carotid stenting was entirely devoid of cases of cranial nerve injury, which occurred in 5.3% of CEA patients. Recent prospective registries of carotid stenting in patients at high risk for CEA are consistent with the SAPPHIRE trial, with reports of 30-day major adverse event rates <8%2–3 Thus, patients who have serious comorbid medical or anatomic
TABLE 1. Indications for Carotid Artery Revascularization

<table>
<thead>
<tr>
<th>Indication Level</th>
<th>Symptomatic Stenosis†</th>
<th>Asymptomatic Stenosis†</th>
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<tbody>
<tr>
<td>Proven</td>
<td>&gt;60% Stenosis</td>
<td>Periprocedural complication risk &lt;6%</td>
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<tr>
<td></td>
<td></td>
<td>Life expectancy &gt;5 y</td>
</tr>
<tr>
<td>Acceptable</td>
<td>&gt;60% Stenosis</td>
<td>Periprocedural complication risk &lt;3%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Planned CABG</td>
</tr>
<tr>
<td>Unacceptable</td>
<td>&lt;60% Stenosis</td>
<td>Periprocedural complication risk &gt;5%</td>
</tr>
<tr>
<td></td>
<td>or</td>
<td>No indication for CABG</td>
</tr>
</tbody>
</table>

CABG indicates coronary artery bypass graft surgery.
*Derived from references 37–40.
†Lesion severity is determined according to the NASCET methodology (ie, the ratio between lumen diameter at the point of maximal stenosis and the lumen diameter of the nontapered segment of the distal ICA).38,44

conditions that increase the risk from an open surgical approach or general anesthesia should be primary candidates for carotid stenting. These conditions include advanced age, significant cardiac and pulmonary disease, prior neck irradiation or radical surgery, restenosis after endarterectomy, contralateral carotid occlusion, high lesions behind the mandible, and low lesions that would require thoracic exposure. Randomized trials comparing carotid stenting and CEA in patients at low surgical risk are in progress.

Carotid stenting has a number of notable relative contraindications. Patients who are intolerant to antiplatelet agents are more safely managed with CEA. Similarly, if the patient has a compelling reason to undergo a major surgical procedure within 3 to 4 weeks that will require the cessation of antiplatelet therapy, CEA may be a better option. A large thrombus burden and specific angiographic findings discussed in detail below should be excluded before carotid stenting. Intracranial arterial stenoses, arteriovenous malformations, or stable aneurysms are not necessarily contraindications for coronary artery stenting; however, in the latter case, stringent control of blood pressure and careful modulation of anticoagulation are mandatory. Although contrast nephropathy is an important consideration in patients undergoing carotid stenting, this seldom represents a contraindication, because experienced operators should rarely require ≥75 mL of contrast material to complete the procedure.

**Patient Selection**

The clinical advantages afforded by any therapeutic intervention are obviously dependent on the natural history of the condition without intervention, the risks inherent to the procedure itself, and the clinical course after a successful intervention. Although the risk of stroke due to carotid artery stenosis treated medically is determined primarily by angiographic lesion severity and symptom status,5,6,40,41 the risks of periprocedural complications after CEA appear to be largely independent of symptoms or the degree of the stenosis,8 and the available data suggest that the same holds true for carotid stenting (unless the lesion contains a large thrombus load).1,36,42

**Symptomatic Patients**

Given the demonstrated benefit of revascularization over medical therapy in the management of severely stenotic lesions (70% to 99% diameter stenosis by the NASCET criteria39,43,44) clearly associated with symptoms attributable to the ipsilateral carotid distribution, CEA in these patients has been considered indicated when the periprocedural risk of death or stroke is <6%.38 The same is applicable to carotid stenting. Available data demonstrate that the rates of periprocedural death or disabling stroke after carotid stenting are generally below 6%, even without the universal use of EPDs1,45 and in patients at high risk for CEA.2–4

The risk of recurrent ipsilateral neurological events with medical management is much lower for moderate stenosis (50% to 69% by the NASCET criteria) than for severe carotid lesions.8 Because the potential benefit of any revascularization procedure is inversely related to angiographic lesion severity,40 in patients with lesions of moderate or borderline severity, the risk-benefit ratio of carotid stenting should be weighed accordingly.

**Asymptomatic Patients**

Stroke prevention in asymptomatic patients requires special consideration. The risk of stroke in the territory of an asymptomatic carotid stenosis has been shown to be strongly dependent on angiographic lesion severity.41 In the European Carotid Surgery Trial (ECST), the 3-year rates of ipsilateral stroke with asymptomatic lesions of less than or greater than 70% stenosis were approximately 2% and 5.7%, respectively.41 (It is noteworthy in this regard that the methods for the measurement of the degree of carotid stenosis have varied among trials, so that application of the ECST methodology results in greater degrees of stenosis for a given lesion than the NASCET methodology.39,44) In the medical treatment arms of the ACAS and ACST, 5-year rates of death or ipsilateral stroke were similar at ≈12%.4 Thus, for clinical benefit to be derived by an asymptomatic patient with a severely stenotic carotid lesion, periprocedural rates of death or stroke after carotid revascularization must not exceed 3%. Given the high prevalence of asymptomatic carotid disease,46,47 the optimal application of carotid stenting in this patient subset must be defined rigorously (see “The 3% Rule” below).
Owing to the very low event rates in patients with asymptomatic lesions of moderate severity (<60% diameter stenosis), it is unknown whether currently available interventional techniques can improve long-term outcomes over those achievable with optimal medical management. Also unresolved are the indications for carotid stenting in asymptomatic individuals with contralateral carotid occlusion and those undergoing major cardiac or vascular surgery.

The 3% Rule

As stated above, carotid artery revascularization can be justified in the asymptomatic patient only if the procedure can be accomplished with a complication rate ≤3%. With the widespread availability of CEA and carotid stenting, candidates for carotid revascularization have generally been selected for either procedure on the basis of the presumed surgical risk (the “conventional paradigm,” depicted in Figure 1). Low-risk surgical patients would usually be referred for CEA or be enrolled in a randomized clinical trial of surgery versus stenting. Patients considered at high risk for open surgery were often referred for carotid stenting, arbitrarily considered a low-risk intervention because little attention had been given to definition of the risks associated with the latter procedure. However, it is of crucial importance to recognize the risks of carotid stenting and to realize that in certain patients (easily identified by readily available clinical and angiographic features), particularly those with asymptomatic lesions, the risks of procedure-related major adverse events might exceed the long-term risk of ipsilateral stroke with medical therapy. We believe that for the full clinical potential of carotid stenting to be realized, a paradigm shift needs to be implemented in the process of procedural risk stratification and selection of patients for revascularization. This applies both to everyday clinical practice and to the design of randomized trials. Clinical decision making that incorporates these principles is depicted in Figure 1.

Implementing the 3% Rule

In determining the risk of death or stroke associated with carotid stenting, it is of critical importance to recognize 4 factors that have been associated with increased procedural complications (Table 2). The most important of these factors is advanced age. In the lead-in phase of the multicenter CREST trial, the risk of 30-day stroke or death among 749 patients was directly related to age (<60 years, 1.7%; 60 to 69 years, 1.3%; 70 to 79 years: 5.3%; and >80 years, 12.1%; P=0.006). Although the risk attributable to advanced age in this analysis appeared to be independent of other clinical (eg, gender or symptom status), angiographic (eg, lesion severity), or procedural (eg, use of distal protection devices) factors, it is likely that the increasing prevalence of the other factors listed in Table 2 with advanced age accounts, at least in part, for this association. Decreased cerebral reserve is another important factor when one considers the risk of carotid stenting. Carotid revascularization (carotid stenting or CEA) is usually associated with some degree of cerebral embolization that is generally well tolerated in patients with good cerebral reserve; however, patients with prior strokes, lacunar

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**TABLE 2. Clinical and Angiographic Features Associated With Increased Procedural Risks After Carotid Stenting**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Features</th>
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<tbody>
<tr>
<td>Clinical</td>
<td>Advanced age ≥80 y</td>
</tr>
<tr>
<td></td>
<td>Dementia</td>
</tr>
<tr>
<td></td>
<td>Prior (remote) stroke</td>
</tr>
<tr>
<td></td>
<td>Multiple lacunar infarcts</td>
</tr>
<tr>
<td></td>
<td>Intracranial microangiopathy</td>
</tr>
<tr>
<td>Angiographic</td>
<td>Excessive tortuosity ≥2 90° bends within 5 cm of the lesion</td>
</tr>
<tr>
<td></td>
<td>Heavy calcification Concentric circumferential calcification Width ≥3 mm</td>
</tr>
</tbody>
</table>
infarcts, microangiopathy, or dementia of varying stages are much more likely to experience neurological deficits after carotid stenting. This risk is markedly amplified in the presence of an isolated hemisphere with lack of good collateral support.

Although some lesion characteristics (eg, degree of stenosis and length) indicate potential technical difficulties, the 2 most important anatomic findings portending an increased procedural risk are vascular tortuosity and heavy concentric calcification. Excessive tortuosity is defined as \( \geq 2 \) 90° bends (arrows) within a 5-cm segment spanning a lesion (arrowhead) in the ICA. LCCA indicates left CCA.

Figure 2. Extreme vascular tortuosity, indicating an increased risk for major complications after carotid stenting, is defined as \( \geq 2 \) 90° bends (arrows) within a 5-cm segment spanning a lesion (arrowhead) in the ICA. LCCA indicates left CCA.

in a satisfactory angiographic outcome, the risk of neurological adverse events exceeds the 3% rule and is thus prohibitive.

Procedural Considerations

The protocol for carotid stenting has been described in detail previously.51 The following technical and procedural factors have proved important in ensuring a facile and complication-free carotid stenting procedure.

Periprocedural Monitoring and Management

With respect to preprocedural therapy, adequately dosed dual-antiplatelet therapy is key. Patients must receive either a combination of clopidogrel 75 mg and aspirin 325 mg for 5 days before carotid stenting or, alternatively, loading doses of clopidogrel (600 mg) and aspirin (650 mg) at least 4 hours before the procedure. On the day of the procedure, oral antihypertensive therapy is withheld, and adequate volume status is ensured. Mild sedation may be offered to anxious patients, but for the vast majority, reassurance and adequate local anesthesia are all that is necessary. The avoidance of sedatives enhances neurological monitoring and limits hypotension. Continuous monitoring of pulse oximetry, intraarterial pressure, and heart rhythm is essential, as is meticulous control of hemodynamics. Intravenous atropine (0.6 to 1.0 mg) should be administered after placement of the sheath in the CCA to suppress bradycardic responses to balloon inflation and stent implantation. Hypotension is invariably noted after balloon dilation of the stent, particularly in elderly patients with heavily calcified stenoses, and is generally benign. However, aggressive volume expansion, intravenous phenylephrine, and occasionally dopamine infusions are sometimes necessary. Blood pressure elevation after the relief of the stenosis can also occur and should be treated with intravenous nitroglycerine, nitroprusside, or labetalol. If distal protection is with an occlusion-aspiration system, blood pressure should be lowered before the occlusive balloon is deflated to prevent the potential consequences of hyperperfusion.52 Anticoagulation therapy with carotid stenting is vital, but it is equally important to note that modest anticoagulation levels should be targeted. Either heparin (70 IU/kg initial bolus, targeting an activated clotting time of 200 to 250 seconds) or bivalirudin (0.75 mg · kg\(^{-1}\) · h\(^{-1}\)) is followed by a maintenance infusion of 1.75 mg · kg\(^{-1}\) · h\(^{-1}\) is administered immediately with sheath insertion. Prolonged infusion of anticoagulant drugs is unnecessary, and these are stopped immediately after stent deployment. Glycoprotein IIb/IIIa antagonists are not routinely used.53

The use of 6F femoral sheaths and arteriotomy closure devices allows for early ambulation. This counteracts the bradycardia and hypotension commonly associated with carotid stenting. Postprocedural intensive care monitoring is unnecessary, although patients should be followed up in a monitored environment by staff familiar with the postprocedural course and with groin access site management. Remaining sheaths should be removed as early as possible, once the activated clotting time has fallen below 150 seconds. Hypotension should be treated aggressively, and causes unrelated
Procedural Stages
The extent of diagnostic angiography is determined by the anatomic information obtained by preprocedural noninvasive studies but should at the very least include an accurate evaluation of lesion severity, the carotid bifurcation, ipsilateral intracranial anatomy, and the anatomy of the CCA. If a balloon-occlusive EPD is to be used, it is mandatory to ensure adequate collateral flow from the contralateral carotid or posterior circulations. For diagnostic angiography, a double-curved 5F catheter (VTK, Cook Inc, Bloomington, Ind) and a 0.038-inch angled-tip hydrophilic coated wire are used. In >98% of patients, this system enables safe selective catheterization of the CCA, ICA, and external carotid artery (ECA), both subclavian arteries, and at least 1 vertebral artery. The same catheterization technique is used to introduce a 6F 90-cm sheath (Shuttle, Cook Inc) into the CCA, generally delivered over a soft-tipped, stiff, 0.035-inch guidewire (eg, Supracore, Guidant Inc, Indianapolis, Ind) positioned in the ECA. The tip of the sheath is positioned in the distal CCA. Guiding shots of the lesion immediately after sheath placement are performed, because ICA tortuosity might be more pronounced by the sheath (Figure 3). Next, the lesion is crossed with a 0.014-inch guidewire, usually that of the EPD. The EPD is deployed in a distal segment of the cervical ICA. Next, the lesion is dilated with an undersized coronary balloon (“predilation”). The stent is the deployed and subsequently “postdilated” with a conservatively sized, low-profile balloon. Finally, the EPD is removed, and final angiography is performed. With contemporary rapid-exchange (“monorail”) systems, the entire process should take as little as 10 to 15 minutes.

Special Considerations
Catheter Placement
Modifications of the catheter placement technique may be required when the lesion is located in the distal segments of the CCA or if the ECA cannot be catheterized. In these cases, the tip of the 5F catheter and guidewire (Amplatz Super Stiff J-wire, MediTech, Natick, Mass) assembly over which the 6F sheath is placed in the CCA is kept below the lesion or
bifurcation. In cases of significant aortic arch elongation or CCA tortuosity, inability to access the ECA might result in insufficient support for sheath placement. Placing guidewires and catheters at or across the lesion to provide adequate support markedly increases the risk of embolic complications.

**Crossing the Lesion**

Wiring the lesion and device delivery can be technically challenging. It is critical to minimize the number and volume of contrast injections into the brain, because this alone predisposes to neurological events. At times, because of extreme angulation at its takeoff, the ICA might not be amenable to wiring. In more complex and calcified lesions, a 7F sheath will provide superior support. At all times, the position of the sheath should be monitored to prevent its prolapse back into the arch. Appropriately shaped 5F catheters (125-cm right Judkins or internal mammary catheters) can be advanced through the guiding sheath so that the tip points into the ostium of the ICA, facilitating wire entry. The EPD must be placed at least ≥2 cm cephalad to the stenosis to accommodate the tip of the stent delivery system and to provide satisfactory coverage of the lesion. With heavy calcification, it can be technically difficult or even impossible to advance the EPD beyond the lesion. In these situations, placement of a second (“buddy”) wire and gentle dilation of the lesion with an undersized balloon can facilitate delivery of the system. Anticipating this situation and having the necessary equipment available minimizes cerebral ischemia. Frequent use of this purpose are 0.014-inch coronary guidewires (eg, Balance, Guidant Inc, Santa Clara, Calif) through an over-the-wire low-profile angioplasty balloon (eg, Maverick, 2.0×40 mm, Boston Scientific, Natick, Mass). After inflation, the balloon catheter is used to exchange the wire for a more supportive type (eg, Stabilizer-Plus, Cordis Inc, Miami, Fla). This guidewire will usually straighten the ICA to permit delivery of the EPD beyond the lesion, although it might result in significant spasm that reduces flow (Figure 3). The tip of any wire used is placed close to the skull base, so the operator must ensure its control to avoid distal vessel trauma. Depending on the severity of ICA tortuosity, “buddy wires” can be removed after the protection device has been placed. Alternatively, the buddy wire can be withdrawn after the stent has been positioned, after stent deployment and postdilation (“jailed buddy wire”), or even after retrieval of the protection device. This can be important, because resistance to stent delivery might cause the sheath to prolapse into the arch, a problem that can be eliminated by the buddy wire.

**Predilation**

Lesion dilation before stenting is strongly recommended. Experimental work has shown that more debris is liberated from the lesion site when predilation is not performed, and clinical experience is concordant. Atheroembolism is increased when predilation is performed with large (0.035-inch compatible) balloons, so low-profile coronary balloons should be selected. When full deflation is ensured, these balloons “rewrap” well without residual winging, so that the risk of vessel-wall trauma during balloon withdrawal is reduced. If the lesion is preocclusive, it is preferable to gradually step up the balloon size to minimize plaque disruption and distal embolization. In these situations, predilation is first performed with a 2.0-mm balloon followed by a second inflation of a 3.5- to 4.0-mm balloon. In rare cases, mainly in heavily calcified lesions, a 5-mm balloon might be required to enable stent delivery. Long balloons (30 to 40 mm in length) are preferred to avoid a “watermelon seed” effect.

**Stent Selection and Deployment**

Self-expanding stents are routinely used because balloon-expandable stents are prone to deformation by external compression. The nominal diameter of the self-expanding stent chosen should be at least 1 to 2 mm larger than the largest diameter of the treated segment, usually the CCA, and 10-mm stents are used in almost all cases (oversizing the stent relative to the diameter of the ICA produces no adverse effects and provides effective trapping of plaque, thereby reducing the risk of embolization). Stent length should be adequate to cover the entire lesion, typically located at the...
origin or proximal segment of the ICA, such that it usually extends from the distal CCA to a healthy segment of the ICA, covering the origin of the ECA. For the vast majority of cases, a stent 30 mm in length by 10 mm in diameter will provide complete lesion coverage and facilitates facile, accurate placement with “road mapping” or bone landmarks. The use of contrast injections for stent positioning should be avoided, because embolic events from air trapping may occur.

Positioning the distal end of the stent in kinks and tortuositites of the ICA should be avoided. These tortuositites can rarely be eliminated and tend to be displaced distally and to be exaggerated by the stiff stent. Covering the origin of the ECA with the stent is not associated with adverse clinical consequences. Follow-up arteriograms have shown that the ECA remains patent with only few exceptions.

**Postdilation**

This is a critical step and requires careful attention, because it is at this stage that embolic events are most likely to develop. The risk of embolization is minimized by conservative sizing of the balloon (5 mm) and by performing a single inflation. The balloon should be deflated slowly. Mild residual stenoses (10% to 20%) or persistence of an ulcer at the lesion site should be accepted, because overzealous stent dilation driven by these findings can worsen embolization.

**EPD Removal**

Removal of balloon-occlusive EPDs is preceded by aspiration of 50 to 60 mL of blood with a dedicated catheter. Filter-based EPDs are removed with a dedicated retrieval catheter. Rarely, the filter can become obstructed by large amounts of embolic material, and blood flow in the ICA is interrupted. Facile technique and optimal antiplatelet therapy prevent this complication in most cases.

**Final Angiographic Assessment**

Careful attention must be paid to the segment of the ICA that contained the EPD. Occasionally (1% to 5% of cases), the embolic protection device can cause dissection in the ICA. The risk of this eventuality is greater with balloon-occlusive devices than with filter-based devices. It is not unusual to encounter spasm and kinks in the cephalad segment of the ICA, particularly in tortuous vessels. These are generally alleviated by guidewire removal and withdrawal of the guiding sheath to the proximal CCA. A small dose of intra-arterial nitroglycerine (100 to 200 μg) is occasionally needed. Stent-related distal edge dissections are rare.

**Postdischarge Monitoring and Treatment**

Patients are discharged with instructions to take clopidogrel (75 mg/d) for 1 month, except for patients treated for lesions related to prior neck irradiation, in whom clopidogrel treatment is extended to 1 year. In the absence of contraindications, aspirin (325 mg/d) is prescribed indefinitely. Patients should have a baseline ultrasound duplex study within 1 month after carotid stenting. This serves as a reference for later follow-up evaluations. Not infrequently, flow velocities within the stent are elevated despite documented good angiographic results. Evidence to date suggests that this finding neither predicts excessive progression of neointimal proliferation nor restenosis. Magnetic resonance angiography is not useful for follow-up purposes because of signal dropout due to the metallic stent. Computed tomographic angiography has shown some promise and may prove to be the modality of choice for follow-up after carotid stenting. Significant angiographic restenosis (>80%) is an uncommon finding, occurring in 3% to 6% of patients. Restenosis is more common in patients initially treated for radiation-induced or post-CEA lesions and can usually be managed by balloon dilation or repeated stenting.

**Conclusions**

Carotid stenting with the use of an EPD is an efficacious method for carotid revascularization. In high-risk CEA patients, carotid stenting has proved superior to CEA. For standard-risk CEA patients, randomized trials comparing both procedures are in various stages of progress. Careful patient selection, based on readily available clinical and angiographic features, and facile technique must be implemented for this procedure to fulfill the tremendous promise it holds for both primary and secondary stroke prevention. Most importantly, it is essential that carotid stenting operators understand how to select patients in whom the procedure entails a low risk for serious complications (<3%). Patients not considered ideal candidates for carotid stenting should be offered well-validated surgical or medical alternatives.

**Disclosures**

Dr Roubin has served as a consultant to or on the advisory board of Abbott Vascular Devices and the Kensey Nash Corporation. Dr Iyer has served as a consultant to or on the advisory board of Abbott Vascular Devices and the Boston Scientific Corporation. Dr Brennan has served as a consultant to Abbott Vascular Devices.

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