Intracranial Angioplasty and Stent Placement for Cerebral Atherosclerosis

H. Christian Schumacher, MD, Alexander V. Khaw, MD, Philip M. Meyers, MD, Rishi Gupta, MD, and Randall T. Higashida, MD

Intracranial atherosclerotic stenoses have been estimated to account for 8%–10% of all ischemic strokes. A substantial number of patients fail the best medical treatment, which includes control of vascular risk factors and administration of antithrombotics (platelet-active drugs or warfarin), statins, and angiotensin-converting enzyme inhibitors. In these patients, angioplasty with stent placement is one reasonable treatment option for preventing massive ischemic stroke. Herein, we discuss basic pathophysiologic concepts and their effect on endovascular revascularization procedures.

EPIDEMIOLOGY AND RISK FACTORS FOR INTRACRANIAL ATHEROSCLEROSIS

INTRACRANIAL atherosclerosis accounts for about 8%–12% of all ischemic strokes (1,2). In the United States, approximately 40,000 strokes a year are due to intracranial atherosclerosis. Typically, intracranial atherosclerosis occurs in the setting of widespread atherosclerosis. Risk factors associated with intracranial atherosclerosis include insulin-dependent diabetes mellitus; hypercholesterolemia; cigarette smoking; hypertension; and black, Hispanic, or Asian descent (1,2). Intracranial atherosclerosis may be either stenotic or dilatative.

PATHOLOGY AND DYNAMICS OF ATHEROSCLEROTIC INTRACRANIAL ARTERIAL STENOSIS

Intracranial stenosis is usually detected in patients presenting with acute ischemic events, and most of our knowledge about the natural history of intracranial atherosclerosis had been obtained from patients examined with either conventional angiography or transcranial Doppler ultrasonography (US). Intracranial stenoses are dynamic lesions and may undergo progression, undergo regression, or remain stable after the initial diagnosis (3–5), a process that is currently poorly understood and difficult to predict in the individual patient. More important, a substantial portion of intracranial stenoses diagnosed in the setting of acute ischemic events will regress with medical treatment alone (5–7).

Most knowledge about the pathology of intracranial atherosclerosis and associated stenoses is based on autopsy studies (8–14). Atherosclerotic stenoses of the intracranial arteries typically occur in the petrous and cavernous siphon portions of the internal carotid artery (ICA) (Fig 1), the main trunk of the middle cerebral artery (MCA, M1 segment, Fig 2), vertebral arteries distal to the transverse foramen of C2 (V3 and V4 segments, Fig 3), and the basilar artery (Fig 4). In most cases, the narrowing is due to atherosclerotic plaques, and pathologic evidence has shown that plaque morphology in intracranial vessels resembles that of other vascular territories. Little is known about plaque pathology in acute ischemic stroke due to intracranial atherosclerosis because, unlike patients who experience coronary events, most patients survive. Plaque morphology appears to be important in the setting of acute coronary syndromes, and, by extension, this might also apply to acute cerebrovascular syndromes. There are two types of plaque: white and yellow. White plaques are also known as stable plaques, and yellow plaques are known as unstable plaques. Yellow plaques have been associated with acute coronary syndromes; white plaques have not. Noninvasive and invasive imaging enables the in vivo diagnosis of the two plaque types in coronary and extracranial carotid arteries (15–19). The use of similar imaging techniques in intracranial arteries, however, is currently not pos-
Figure 1. Symptomatic tandem intracranial carotid artery stenosis. A, Angiogram of the right ICA obtained in the frontal projection during the arterial phase demonstrates a high-grade tandem stenosis (arrows) in the ICA. The proximal stenosis is 80%-90% of the transluminal diameter. B, Angiogram obtained after stent-supported angioplasty with a 3.5 × 12-mm-diameter coronary stent for the proximal lesion and a 3.5-mm balloon catheter for the distal lesion. The vessel diameter is markedly increased at both sites (arrows), and the patient’s neurologic symptoms resolved.

Figure 2. Images in a 48-year-old man with intermittent aphasia and weakness in his right arm. A, Angiogram of the left carotid artery obtained in the frontal projection during the arterial phase demonstrates a high-grade stenosis (arrow) in the middle portion of the left MCA (M1 segment). B, Angiogram obtained after stent-assisted angioplasty with a 2.5 × 8-mm coronary stent (arrow). Complete normalization of the vessel lumen was achieved. The patient remained asymptomatic following treatment.
sible, and clinical judgment in conjunction with conventional angiography remains the standard of reference for treatment considerations.

PATHOPHYSIOLOGY OF STROKE IN INTRACRANIAL ATHEROSCLEROSIS

Ischemic strokes in intracranial atherosclerosis can be due to perfusion failure, local thrombosis at the site of the stenosis, arterioarterial thromboembolism, or occlusion at the origin of small penetrating arteries.

Depending on both stenosis grade and adequacy of collateral circulation, stenosis of cerebral arteries may lead to reduction of blood flow distal to it. In cerebral angiography, hemodynamic effects are usually demonstrated as delayed flow or border-zone shift. The hemodynamic effects of cerebrovascular stenoses have been categorized into three stages (see Derdeyn et al (20) for a review), as follows: stage 0 = normal cerebral hemodynamics, stage 1 = reflex vasodilation in response to inadequate collateral vessels and a decrease in perfusion pressure with an increase in cerebral blood volume and prolongation of mean transit time but preservation of cerebral blood flow and a normal oxygen extraction fraction (OEF), and stage 2 = misery perfusion with a decrease in the cerebral blood flow and an increase in the OEF.

Recently, this more simplified stage model of hemodynamic failure has been revised (21), as follows: An increase in the OEF occurs after slight decreases in cerebral perfusion pressure, and an increase in the OEF and cerebral blood volume distal to an occlusion or stenosis are high-risk factors for ipsilateral ischemic stroke. Overall, the presence of a high-grade stenosis or even occlusion does not necessarily imply that there is actual perfusion failure, and every measure must be taken to document this in a given patient. Important factors for perfusion failure are location of the stenosis and presence of sufficient collateral flow. For example, a high-grade stenosis in the intracranial carotid artery may be well compensated if there is a fully functioning circle of Willis with good supply of the ipsilateral MCA through retrograde flow from the ipsilateral anterior communicating artery or the posterior communicating artery. Conversely, a similar stenosis in the proximal part of the MCA may lead to overt perfusion failure due to the lack of substantial collateral vessels for that arterial segment. Hemodynamic compromise as defined with neuroimaging is associated with and seems to be an independent risk factor for ipsilateral stroke (21–23). Patients with hemodynamic compromise due to intracranial atherosclerosis may represent a subgroup of patients for whom endovascular revascularization may have the greatest chance of being proved effective.

Findings of several autopsy studies (9,10,14,24,25) suggest that thrombosis complicates intracranial atherosclerosis associated with a preexisting stenosis. In some cases, intramural hemorrhage due to fibrinoid degeneration of the capillaries in the plaque could be responsible for cerebral arterial thrombosis (25,26). Local thrombosis may also result in distal arterioarterial embolism (24,27,28). The detection of microembolic signals with transcranial Doppler US has been associated with symptomatic intracranial stenoses and may be a marker for a local unstable plaque (7,29). In these patients, treatment strategies may primarily focus on plaque stabilization and anticoagu-

Figure 3. Images in a 62-year-old woman with episodic visual disturbances and dysarthria. A, Angiogram of the right vertebral artery obtained in the frontal projection during the arterial phase demonstrates a high-grade focal stenosis (arrow) in the intracranial portion of the left vertebral artery. B, Angiogram obtained after stent-assisted angioplasty with a 2.5 × 12-mm coronary stent demonstrates complete restoration of the luminal diameter (arrow). The patient's symptoms resolved.
lation, whereas endovascular techniques for revascularization may be considered secondarily.

Stenotic atheromatous plaque can include the origin of small arteries. The main point is that stenotic atheromatous plaques in large intracranial arteries might cause symptomatic stenosis or occlusion of small penetrating arteries.

Figure 4. Images in an 82-year-old man with signs of vertebrobasilar insufficiency. A, Frontal and B, lateral angiograms of the vertebral artery obtained in the frontal projection during the arterial phase demonstrate an irregular and eccentric severe stenosis in the middle third of the basilar artery (arrows). C, Frontal and D, lateral angiograms obtained after stent-assisted angioplasty with a 3 × 12-mm-diameter coronary stent show that the luminal diameter has been restored (arrows) without additional perforator artery ischemia. The patient’s symptoms resolved immediately. Because the patient presented before permanent parenchymal injury, his recovery was rapid, with discharge in 3 days.
arteries (30,31). Careful correlation of the ischemic clinical syndrome with neuroimaging is necessary in these cases because angioplasty of the stenotic plaque may result in complete obstruction and subsequent ischemic stroke in the territory of the penetrators, resulting in an adverse outcome to the procedure.

**PROGNOSIS OF PATIENTS WITH INTRACRANIAL ATHEROSCLEROSIS**

The prognosis of patients after stroke associated with intracranial stenoses seems to be dependent on the location and extent of intracranial atherosclerosis. Most of our knowledge about prognosis in intracranial atherosclerosis is based on retrospective studies. An overview of reported annual stroke and death rates is given in Table 1. Most of these annual stroke and death rates, however, were gathered from retrospective studies with highly selected and very heterogeneous cohorts. Unfortunately, prospective, population-based data about the prognosis of intracranial atherosclerosis are lacking.

<table>
<thead>
<tr>
<th>Disease Distribution</th>
<th>Death Rate per Annum (%)</th>
<th>Any Stroke per Annum (%)</th>
<th>Isotopic Stroke per Annum (%)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid</td>
<td>9.5–17.2</td>
<td>3.9–11.7</td>
<td>3.1–8.1</td>
<td>(72–76)</td>
</tr>
<tr>
<td>MCA</td>
<td>3.3–7.7</td>
<td>2.8–4.2</td>
<td>4.7</td>
<td>(28,77,78)</td>
</tr>
<tr>
<td>Vertebrobasilar</td>
<td>6.1–9.7</td>
<td>2.4–13.1</td>
<td>0.8–7</td>
<td>(79–82)</td>
</tr>
</tbody>
</table>

**IDENTIFICATION OF THE SYMPTOMATIC PATIENT WITH INTRACRANIAL ATHEROSCLEROSIS**

Currently, angioplasty and stent placement are usually performed in patients with symptomatic intracranial stenoses. It is important to evaluate all patients before performing angioplasty to correlate the patient’s symptoms and clinical findings with the presumed symptomatic vessel and to exclude other potential diagnoses (eg, cerebral vasculitis) with alternative treatment options. Determination of the type and duration of medical therapy should be part of the evaluation, and medical treatment should be adjusted as necessary.

The usual vascular work-up of patients with stroke includes extracranial and transcranial Doppler or duplex US, computed tomographic (CT) angiography, or magnetic resonance (MR) imaging with MR angiography. Intracranial stenoses in the arteries accessible to angioplasty and stent placement can be easily identified with any of those studies. In individual cases, it might be necessary to verify the findings of the noninvasive studies with conventional cerebral angiography. Impaired cerebrovascular reserve as an indicator for perfusion failure distal to the stenosis can be diagnosed with several methods, each of which has its own advantages and disadvantages. Currently, the diagnosis of stage 2 hemodynamic compromise (increased OEF) is only possible with positron emission tomography and an oxygen 15-labeled radiotracer. To what extent the information obtained with these studies might be helpful for determining the indication for and timing of angioplasty remains to be established with future studies.

**MEDICAL TREATMENT OF INTRACRANIAL ATHEROSCLEROSIS**

The medical treatment of intracranial atherosclerosis is similar to that of atherosclerosis in other vascular territories and includes the control of vascular risk factors and the prescription of antithrombotics (platelet-active drugs or warfarin), statins and angiotensin-converting enzyme inhibitors (32–36). Patients are usually referred for elective intracranial angioplasty if “maximal medical” therapy has been unsuccessful. The term “maximal medical therapy,” however, is not clearly defined. In most instances, maximal medical therapy failure is considered to occur when a patient experiences a transient ischemic attack or recurrent ischemic stroke while receiving therapeutic doses of aspirin (≥81 mg per day), ticlopidine (500 mg per day), clopidogrel (75 mg per day), warfarin (international normalized ratio ≥2.0), or intravenous administration of heparin (prolongation of partial thromboplastin time more than 1.2 times the baseline value).

**ENDOVASCULAR REVASCULARIZATION: ANGIOPLASTY**

During the past decade, dramatic improvements in microcatheter angioplasty balloon and stent technologies have enabled more innovative interventional neurovascular procedures to be performed. The successful use of balloon angioplasty for the treatment of intracranial atherosclerosis has been reported by an increasing number of investigators, predominantly those in academic centers and high-volume medical centers with substantial neurovascular expertise. Results to date are encouraging, yet the procedure is technically demanding at many levels and carries substantial risk. In general, most practitioners reserve endovascular revascularization for patients who are refractory to maximal medical therapy.

Not all patients with cerebrovascular stenoses are equal: Careful neurologic and imaging assessments are mandatory for planning a successful individualized treatment strategy. Moreover, a multidisciplinary approach to treatment is necessary because weakness in any link of the chain can have devastating consequences. An important concern is the customized approach needed to ad-
dress each patient. No single operator works in a vacuum. Technically successful revascularization of cerebrovascular stenosis is only one step toward the achievement of acceptable treatment outcomes.

Mori et al (37,38) developed an arteriographic classification system to predict the outcome of cerebral revascularization with primary angioplasty alone. Lesions were categorized with thin-section digital subtraction arteriography according to length and geometry, as follows: type A lesions were short (<5 mm in length), concentric or moderately eccentric, and nonocclusive; type B lesions were tubular (5–10 mm in length), extremely eccentric, and moderately angulated (curved); and type C lesions were diffuse (>10 mm in length), extremely angulated (>90°), or have a very tortuous proximal segment.

In general, Mori et al (37,38) found that the more complex the target lesion, the less satisfactory the immediate and long-term outcomes with the currently available devices. For instance, the treatment success for type A lesions was 92%, whereas treatment success for type C lesions was only 33%, with a 100% restenosis rate at 1 year. The use of currently available stent technology resolves certain limitations inherent to angioplasty alone.

### GENERAL PRINCIPLES OF THE INTERVENTION

One to three days before the procedure, patients are routinely treated with a combination of clopidogrel and aspirin. Angioplasty and stent placement are performed with local (39–41) or general anesthesia (42,43). During the procedure, heparin is intravenously administered to achieve an activated clotting time of 250 to 300 seconds. Angioplasty and stent placement are performed by using a conventional transfemoral approach. Most applications have related to the vertebrabasilar system (40,44–46), with limited reports of their use in the MCA (39) and intracranial ICA (40,47).

There are some important principal points due to the intrinsic features of cerebral arteries: (a) The cerebral vessels are—compared with the coronary vessels—relatively tethered by branching arteries and suspended in cerebrospinal fluid. Several small perforating arteries with diameters of 250 μm or smaller arise from the larger cerebral arteries and enter the brain tissue. Deformation of these perforating arteries, which are invisible even with thin-section digital subtraction angiographic equipment, may easily result in their avulsion and lead to catastrophic intracranial hemorrhage. In general, endovascular devices for neurointerventional use are more flexible and softer than those for coronary applications. Most available angioplasty and stent devices, however, are designed for coronary use and remain excessively rigid for cerebral application (see Table 2 for an overview of endovascular devices used off-label for angioplasty of and stent placement in intracranial stenoses between January 1998 and June 2003). Tremendous research efforts are under way to design more malleable devices for intracranial applications. These efforts have led in August 2002 to U.S. Food and Drug Administration (FDA) approval of the Neurolink system (Guidant, Menlo Park, Calif), which was specifically designed for intracranial use (48), under the Humanitarian Device Exemption. The Neurolink system was evaluated in a prospective, multicenter, feasibility study (the “Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral and Intracranial Arteries [SSYLVIA] study). Although publication of the study results in a peer-reviewed journal is pending, the main re-

### Table 2

<table>
<thead>
<tr>
<th>Catheter or Stent</th>
<th>Company</th>
<th>References</th>
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<tbody>
<tr>
<td>PTCA dilatation catheters</td>
<td>Boston Scientific/SciMed</td>
<td>(85)</td>
</tr>
<tr>
<td>NC Bandit</td>
<td>Boston Scientific/SciMed</td>
<td>(40,83,84,86,87)</td>
</tr>
<tr>
<td>NC Ranger™, Quantum Ranger™</td>
<td>Target/Boston Scientific</td>
<td>(52,54,55,66,67,84,88–91)</td>
</tr>
<tr>
<td>Stealth, FasStealth</td>
<td>Target/Boston Scientific</td>
<td>(87)</td>
</tr>
<tr>
<td>Maverick®</td>
<td>Medtronic AVE</td>
<td>(83)</td>
</tr>
<tr>
<td>Stealth, FasStealth</td>
<td>Guidant</td>
<td>(83)</td>
</tr>
<tr>
<td>D1</td>
<td>Guidant</td>
<td>(87)</td>
</tr>
<tr>
<td>CROSSAIL®</td>
<td>Cordis</td>
<td>(92)</td>
</tr>
<tr>
<td>OPENSAIL®, HIGHSAIL®</td>
<td>Cordis</td>
<td>(39)</td>
</tr>
<tr>
<td>SAVVY®</td>
<td>Cordis</td>
<td>(84,87)</td>
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<tr>
<td>NINJA™</td>
<td>Cordis</td>
<td>(40,83,84,86,87)</td>
</tr>
<tr>
<td>Endovascular stents</td>
<td>Boston Scientific Corporation, Natick, MA; Cook Cardiology, Bloomington, IN; Cordis Corporation, Miami, FL; Guidant Corporation, Indianapolis, IN; SciMed Life Systems Inc, Maple Grove, MN; Target Therapeutics, Freemont, CA; Medtronic AVE, Santa Rosa, CA.</td>
<td>(45,47)</td>
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<td>Bx-VELOCITY®</td>
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<td>(40,42,43,84,86,87)</td>
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<td>GR-II</td>
<td>Medtronic AVE</td>
<td>(44)</td>
</tr>
<tr>
<td>AVE GFX™</td>
<td>Medtronic AVE</td>
<td>(83,84)</td>
</tr>
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<td>AVE Micro Stent™ II</td>
<td>Guidant</td>
<td>(37,39,40,43,44,46,87)</td>
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<tr>
<td>GFX S670</td>
<td>Boston Scientific</td>
<td>(84,87,93)</td>
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<tr>
<td>Multi-Link®</td>
<td></td>
<td></td>
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<tr>
<td>NIR® ELITE</td>
<td></td>
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</tbody>
</table>

Note.—Boston Scientific Corporation, Natick, MA; Cook Cardiology, Bloomington, IN; Cordis Corporation, Miami, FL; Guidant Corporation, Indianapolis, IN; SciMed Life Systems Inc, Maple Grove, MN; Target Therapeutics, Freemont, CA; Medtronic AVE, Santa Rosa, CA.
sults are presented in the summary of safety and probable benefit published by the FDA or as an abstract (49,50).

(b) Soft flow-directed catheters for distal catheterization of the tortuous cerebral arteries, as used for endovascular treatment of arteriovenous malformations, are not applicable for navigation across cerebrovascular stenoses. For that reason, all intracranial angioplasty procedures must be performed with guide wire navigation. Typically, an appropriate guiding catheter is placed into the cervical carotid or vertebral artery. With use of biplane roadmaps, the intracranial stenosis is traversed with a floppy-tipped guide wire. Subsequently, a microcatheter is advanced across the lesion and correct intraluminal position confirmed with the injection of a small volume of contrast material. An exchange-length floppy-tipped hydrophilic guide wire is then positioned with its tip sufficiently distal to the lesion to provide sufficient support for the stent. Typically, this requires the tip to be in the insular (M2) branches of the MCA for M1 stenoses (39), in P2 segments of the posterior cerebral artery for basilar artery stenoses (44), or in the M1 segment of the MCA for intracranial ICA stenoses (47). The risk of dissection and subintimal passage of the microguidewire in the cerebral circulation is substantial owing to the delicate structure of cerebral arteries.

(c) Compared with coronary arteries, intracranial arteries are more tortuous and located more distally from the orifice of the guiding catheter to the target lesion. This leads to a “bow-string” effect, as additional pressure is required to advance the microcatheter over the guide wire and makes endovascular navigation extremely challenging.

(d) The use of a microcatheter and a soft 0.010-inch or 0.014-inch microscopic guide wire to cross the stenosis initially and then exchange the treatment device (balloon catheter or balloon-mounted stent) by using the over-the-wire technique is technically superior and preferable to the primary crossing of the stenosis with the treatment device. This technique considerably reduces the risk of catastrophic vessel dissection (see b).

(e) Angioplasty is performed with an undersized balloon very slowly, over several seconds to minutes. Tearing of the vessel should be avoided, and the goal here is just to stretch the vessel because small increases of the vessel diameter result in large increases in perfusion. This principle is completely different from that used with the procedure performed in coronary or peripheral arteries.

(f) Thrombus formation during the procedure is possible and makes the administration of thrombolytic agents (eg, urokinase, tissue plasminogen activator) or glycoprotein IIb/IIIa antagonists (ie, abciximab) necessary. In neurointerventional procedures, the administration of these drugs is reported to result in hemorrhagic complication rates that exceed those reported in the coronary literature (51). This seems to be especially true for patients who have experienced recent cerebral ischemic events.

(g) The safety profile and efficacy of stent-assisted angioplasty in the peripheral, extracranial cerebral, and coronary circulation has been shown to be superior to that with balloon angioplasty alone. Stents limit vessel wall recoil and the extent of introgressive dissection by compressing the intimal flap. Stent placement, however, is always more traumatic to the vessel wall than is angioplasty alone, and the barbs at the end of the stent may actually perforate intracranial vessels. Images obtained before and after stent-supported angioplasty are shown in Figures 1–4.

(h) Most interventional neuroradiologists prefer general anesthesia over local anesthesia because it maximizes safety, reduces motion artifacts, and reduces overall procedure time (52–54). In a patient with already compromised cerebral perfusion, the maintenance of adequate perfusion and cerebral protection is crucial. Great care must be taken to prevent relative hypotensive episodes during anesthesia induction. Burst suppression-inducing doses of etomidate may help protect the brain from ischemia, especially while the treatment device transverses the stenosis, as flow-arrest is often seen for high-grade or tortuous stenoses.

**COMPLICATIONS**

Table 3 summarizes typical complications of endovascular revascularization procedures for intracranial atherosclerosis and the frequency of their occurrence. In most series, the number of acute major complications reported for experienced interventional neuroangiologists is well below 10%. Immediate complications of balloon angioplasty without stent placement include plaque and/or vessel recoil, vessel dissection, acute closure, perforation, and rupture. In the SSYLVIA study (50), which, to our knowledge, is the only prospective multicenter study conducted so far, only four of the 61 patients had a stroke (two minor strokes, one major intraprocedural stroke, and one major postprocedural stroke), and no patient died within 30 days. This corresponds to a 30-day composite stroke and death rate of 6.6%.

**POSTTREATMENT IMAGING EVALUATION**

Subacute-to-late restenosis is related to intimal hyperplasia (cellular proliferation) and vascular remodeling. For noninvasive surveillance, MR angiography is useful (52,55); however, not all stenoses are detected with these modalities. Furthermore, measurements of stenosis severity can be inaccurate. The role of CT angiography in the accurate depiction of restenosis remains to be established but may be limited due to the beam-hardening artifacts that occur with stent implantation. Some equipment vendors have begun to advertise imaging systems that overcome artifacts related to metallic prosthetics. In the in-

<table>
<thead>
<tr>
<th>Type of Periprocedural Complication*</th>
<th>Range (%)</th>
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<tbody>
<tr>
<td>Ischemic stroke</td>
<td>0–20</td>
</tr>
<tr>
<td>Intracranial hemorrhage</td>
<td>0–4</td>
</tr>
<tr>
<td>Death</td>
<td>0–8</td>
</tr>
<tr>
<td>Vessel dissection</td>
<td>0–10</td>
</tr>
<tr>
<td>Vessel rupture</td>
<td>0–4</td>
</tr>
<tr>
<td>Acute vessel occlusion</td>
<td>0–4</td>
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</tbody>
</table>

* More than one complication may occur in a given series.
terim, certain authors advocate rigorous angiographic follow-up beginning at 3 months (38,53,56) given the 33% and 100% incidence of restenosis for Mori type B and type C lesions, respectively, at 1 year (38). It is recommended that conventional angiographic follow-up be performed initially at 3 months, at which stage additional endovascular treatment can be undertaken if required. Depending on patient age, medical condition, and other angiographic risk factors, the value of aggressive arteriographic surveillance must be weighed against the risk of complications (57–59).

CONCLUSION

The poor prognosis of patients with symptomatic intracranial atherosclerotic stenoses despite best medical management has defined a new role for endovascular revascularization of the intracranial circulation. However, the procedure is associated with a morbidity and mortality rate up to 10%–20% in minimally symptomatic patients and is possibly much higher in neurologically unstable patients (37,39,42,52,55,60–71). We recommend using the smallest angioplasty balloon possible to minimize complications. Stent-assisted angioplasty is now feasible with recent stent technology, allowing improved restoration of vessel diameter while reducing the incidence of some local complications. Restenosis after stent placement appears less problematic at early follow-up compared with angioplasty alone, but the adaptation of coronary technology and long-term patency of intracranial stent placement has yet to be determined. A multidisciplinary approach to patients with symptomatic intracranial atherosclerosis is imperative to achieve appropriate outcomes across the spectrum of disease.

References

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