Angioplasty and Stenting for Cerebrovascular Disease: Current Status

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KEYWORDS

• Stent • Intracranial • Cerebrovascular • Stroke
• Aneurysm • Dissection • Pseudotumor cerebri

As with many concepts in medicine, the terms, ''stent'' and ''stenting,'' have had a convoluted process of consecration. Charles Stent (1807–1885), an English nineteenth-century dentist, is remembered for his contributions to the field of denture making, in particular the addition of stearine to improve the stability and plasticity of existing dental impressions materials and the use of talc as a filler and coloring agent of dentures and dental prostheses. ''Stenting'' first was used to describe the action of stiffening garments.

In medicine, a stent is defined as ''an expandable wire mesh or a hollow perforated tube which is inserted in a hollow structure of the body to keep it open.'' Therefore, the word ''stenting,'' which now designates the use of mechanical devices to overcome diameter reductions in human ducts, is somewhat out of place in relation to its initial use. A review of stents for the treatment of arterial disease must start with a mention of balloon angioplasty, which was developed in the 1970s by Andreas Gruentzig in Zurich, Germany, and gradually gained wide acceptance, as many trials demonstrated its effectiveness, and, in many cases, its superiority compared with surgery in various organ systems.[1]

The issue of restenosis, however, which has been amply documented in the coronary, renal, and peripheral circulations, has continued to generate debate as to the long-term effectiveness of angioplasty.[2]

That naturally led to the development of vascular stents. The first device to be used extensively in the vascular system was the balloon-mounted Palmaz-Schatz stent (Johnson & Johnson, Warren, New Jersey).[3]

The use of intravascular stents has exploded in recent years, so that there now are more than 15 companies in the United States and Europe actively involved in the manufacturing, research, and development of stents that have various characteristics.

Despite the lack of formal comparison, in any vascular territory, between the long-term patency rates of stenting and those of surgery, clinical results, including outcomes and long-term arterial patency, are considered better with stents than with angioplasty, particularly in the coronary circulation.[4,5]

TECHNICAL CONSIDERATIONS

Stent Technology

Stents may be either balloon expandable or self-expandable. In addition, self-expandable stents may be delivered alone or after balloon angioplasty.

Each choice of technique has specific advantages and disadvantages. For example, self-expandable stents are relatively easy to deliver and have a low risk for vascular injury. They

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have, however, a relatively low radial force compared with balloon-expandable stents, which have a higher risk for vascular injury because of excessive blood vessel straightening and are harder to deliver because of limited flexibility, whereas balloon angioplasty carries a risk for elastic recoil and dissection.[6]

In addition, stents may be made of inert metal or may be biologically active. Purely metallic stents are made of stainless steel or of complex alloys, such as nickel-titanium, used in most self-expandable and some ballon-expandable stents. Biologically active stents, designed to inhibit neointimal hyperplasia, include coated stents and drug-eluting stents. Stents with a passive coating have been made with diethylstilbestrol (DES), which inhibits the germinal matrix in and around the stent, and with heparin, which acts to inhibit thrombosis.

Drug-eluting stents have a polymer coating that facilitates the release of a drug into the local vascular tissue. Mechanisms of action and drugs used so far include anti-inflammatory (dexamethasone and methylprednisolone), antiproliferative (angiopectin, actinomycin, paclitaxel, and sirolimus), migration inhibitor (batimastat), and healing promoter actions (estradiol, via vascular endothelial growth factor activation).

The most extensively used drugs to date are two immunosuppressants: sirolimus (rapamycine), a macrolide used systemically to treat renal transplant rejection, which binds to a receptor protein to inhibit a regulatory enzyme so that smooth muscle cell proliferation is shut down; and paclitaxel, a derivative of the yew plant, which inhibits the cell cycle and is used in breast, ovarian, and lung cancer.[7]

Technical Limitations

Several limitations remain associated with the use of stents. In-stent thrombosis is a significant risk, despite the use of antiplatelet therapy, which itself adds some degree of morbidity. Once delivered in a blood vessel, stents may interfere with the delayed luminal expansion that accompanies late remodeling.[8] In addition, there is frequent mismatch with the vessel size, often resulting in a smaller lumen after stent implantation. Also, the implantation of a stent may result in the permanent occlusion of side branches (jailing). Although initially considered a major advance in preventing late restenosis, drug-eluting stents also remain associated with subacute and late thrombosis, requiring prolonged antiplatelet therapy for at least 12 months. Further, the polymer used as a vehicle for drug delivery may trigger vascular irritation, endothelial dysfunction, vessel hypersensitivity, and chronic inflammation at and around the stent.[9] Lastly, metallic stents are responsible for significant artifacts on cross-sectional imaging studies (MRI, magnetic resonance angiography, CT, and CT angiography), which are the dedicated noninvasive modalities used to follow these patients.

Because of the limitations of currently available stents, many operators consider offering balloon angioplasty for intracranial stenotic disease as the first-line treatment, and reserve the use of a stent for failures or complications. Therefore, readers should become familiar with the following lexicon, inherited from the interventional cardiology practice: ''elective stenting'' refers to the planned use of a stent, regardless of the results of preliminary balloon dilatation; ''direct stenting'' is the placement of a stent without the use of a balloon; ''provisional stenting'' is performed only in case of suboptimal dilatation; and, lastly, ''bailout stenting'' is used when there is acute occlusion or dissection of an artery, usually the result of plaque rupture during dilatation.

Future Directions

Significant hopes in research rest on bioabsorbable stents, which, once absorbed, leave the healed vessel with its original structure, allowing restoration of vasoreactivity and other intrinsic properties. Polymers, such as poly-L-lactic acid and polyglycolic acid, which are used widely as delivery vehicles for drug coatings, are currently evaluated as the major components of either self-expanding or balloon-expandable stents.[10,11]

Perioperative Medical Management

Adjunctive pharmacotherapy is used in the perioperative period and as maintenance therapy in a standard fashion in patients treated with intracranial stents. Most treatment algorithms rely on acetylsalicylic acid (aspirin) and clopidogrel (Plavix), used either separately or in combination, although several other agents may be used by some operators, including heparin, warfarin, ticlopidine, and glycoprotein IIb/IIIa inhibitors.

INTRACRANIAL ATHEROMATOUS DISEASE

Clinical Impact and Pathophysiology

Intracranial atherosclerotic disease is increasingly recognized as a serious and potentially treatable cause of stroke. In the United States, Europe, and Asia, stroke poses a significant public health problem, representing the leading cause of disability and the third leading cause of mortality.[12] Several studies demonstrate that intracranial

stenoses are present in approximately 10% of patients who present with cerebrovascular complaints.[13–15] Although it was not clear for a long time how much morbidity is attributable to intracranial atheromatous disease, its significance is increasingly established. In the EC/IC Bypass Study, between 7% and 10% of patients who had proven intracranial stenoses had strokes.[16]

The association between arterial stenoses and ischemic events in cortical territories distal to the lesion has been since amply demonstrated.[17,18]

The first National Institutes of Health (NIH) warfarin-aspirin study concluded that, in patients who had known intracranial stenoses, the annual stroke rate attributable to that specific territory was at least 11%.[19] Current yearly estimates of the American Heart Association are that between 50,000 and 60,000 stokes are attributable to intracranial atheromatous disease in the United States.

Further, subgroups of the general population, such as African Americans, Asian Americans, and Hispanics, are identified as having significantly higher rates of intracranial atheromatous disease.[13–15] Also, intracranial disease was found in one study to be twice as prevalent in men as in women (29% versus 14%).[13] In particular, it is demonstrated that African-Americans are more likely than other populations to develop intracranial stenoses in the anterior circulation than extracranial carotid or vertebral artery disease.[20,21]

General risk factors are the same as for vascular disease in general and include hypertension, hypercholesterolemia, tobacco smoking, and diabetes mellitus in addition to heredity and race.[13–15,18,19,22]

There are several mechanisms by which arterial stenosis may cause a stroke: (1) hemodynamic failure, (2) direct occlusion of perforating arteries at the site of the atheromatous plaque, (3) thromboembolism distal to the stenosis, and (4) thrombosis of the atheromatous plaque resulting from intraplaque hemorrhage, thrombosis, or plaque rupture, similar to what happens in larger arteries, such as the carotid or the coronary arteries.[22–25]

Pharmacologic Therapy

The efficacy of medical therapy to prevent stroke in patients who have intracranial stenoses is not established. This was evaluated in two studies, the Warfarin versus Aspirin for Symptomatic Intracranial Disease (WASID) studies. The first WASID study was a retrospective, multicenter study, the purpose of which was to identify the best medical therapy (warfarin or aspirin) in a relatively small number (n = 68) of retrospective patients who had angiographically documented stenoses (50%–99%) of large intracranial arteries, studied between 1985 and 1991.[26] All patients had experienced a transient ischemic attack (TIA) or a stroke in the territory of the affected artery, which was the intracranial vertebral (n = 31), basilar (n = 28), posterior cerebral (n = 6), or the posterior inferior cerebellar (n = 3) artery. Patients were treated with either warfarin (n = 42) or aspirin (n = 12). Mean follow-up was 14.7 months in the warfarin group and 19.3 months in the aspirin group; the rate of major stroke or death was 8.4% in the warfarin group and 18.1% in the aspirin group (with a 9% stroke rate in the same arterial territory as the intracranial stenosis). Because it was a retrospective study, the first WASID study also provided data as to the natural history of intracranial atheromatous disease, again suggesting a yearly stroke rate of over 10% in untreated patients.[19] Subgroup analysis revealed yearly stroke rates of 10.7% in patients who had basilar stenoses, 7.8% in vertebral stenoses, and 6% in posterior cerebral or posterior-inferior cerebellar artery stenoses.[27]

These data triggered the initiation of the better-known, NIH–sponsored, multicenter, second WASID trial, which was randomized, prospective, and double-blinded and enrolled 569 patients followed for a mean of 1.8 years.[28] Ischemic stroke in the territory distal to an arterial stenosis (50%–99%) occurred in 12% of the aspirin-treated patients and 11% of the warfarin-treated patients at rates, therefore, that were not statistically different. Much higher rates of major hemorrhage (8.3% versus 3.2%) and death (9.7% versus 4.3%), however, were observed in the warfarin-treated group than in the aspirin-treated group. Also, high rates of ischemic stroke, hemorrhagic stroke, and vascular death were observed in both groups during the same average follow-up period of 1.8 years (21.8% in the warfarin group and 22.1% in the aspirin group). The conclusions of the WASID trial were that aspirin should be used instead of warfarin in patients who had intracranial arterial stenoses, as the risk for hemorrhage was unacceptably high with warfarin; however, neither warfarin nor aspirin was credited with significant protective effects in these patients.[28]

Intracranial Angioplasty Studies

At the same time, experience with the endovascular management of intracranial disease was slowly growing. The first accounts of balloon angioplasty for intracranial disease are attributed to Sundt and coworkers who treated symptomatic basilar stenoses in that fashion, reported in 1980.[29] Because

of the high rate of complications, however, including vessel rupture and acute occlusion resulting from intimal dissection, thrombosis, and elastic recoil, the technique did not expand rapidly.

Terada and colleagues reported on 12 patients who had symptomatic severe intracranial vertebral (n = 7) or basilar (n = 5) artery disease and reported a 17% rate of periprocedural major stroke or death for a total complication rate of 33% (two thromboembolic and two arterial dissections).[30] Further, the degree of angiographic improvement was modest, with mean percentage of intraluminal stenoses improving from 84% to 44% after angioplasty.[30]

Hacein-Bey and colleagues[31] reported on a group of high-risk patients who had symptomatic intracranial disease. Twenty patients were evaluated and an algorithm was applied to ascertain that (1) symptoms lay entirely in the targeted arterial territory, (2) there was evidence of perfusion failure in the symptomatic arterial territory implying high likelihood of improvement after angioplasty, and (3) patients had failed maximum medical therapy (warfarin at the time). Angioplasty was offered to the 12 of 20 patients who fulfilled all criteria, successfully and without complications. During the mean follow-up period of 12 months (range 2–37 months), 16% of angioplasty-treated patients and 37% of medically treated patients had recurrent events.

In 1999, Marks and coworkers reported on 23 patients who had symptomatic intracranial stenoses. Technical success was achieved in 91%, with one treatment failure and one death from arterial rupture. Importantly, this study featured long-term clinical follow-up (mean 35.4 months, range 16–74 months), which revealed an annual rate of subsequent stroke in the territory of the affected artery of 3.2%.[32]

Also in 1999, Connors and Wojak published their experience of almost one decade of practice of intracranial angioplasty in 70 patients.[33] Despite an excellent overall rate of technical success (98%), these investigators reported a major change in their technique, so that in their last 50 patients, they used a balloon that was undersized relative to the arterial dimensions and slow, gradual inflations. They argue, understandably, that the apparent suboptimal angiographic results obtained are amply justified by the increased safety resulting from decreased intimal damage, decreased acute platelet/thrombus deposition, and decreased risk for acute arterial closure. Late restenosis occurred in 9%, and residual stenosis greater than 50% was noted in 16% of their patients. Unfortunately, long-term follow-up is not available in their study.

Gress and colleagues[34] reported on 25 patients who had vertebrobasilar disease who had failed to respond to medical therapy. Stenoses were at the intracranial vertebral (n = 10), vertebrobasilar junction (n = 9), and basilar (n = 6) levels. Although perioperative risk for major stroke or death was 16% in this series, angioplasty was able to restore arterial lumen by a mean of 40% in all patients, establishing, therefore, a reasonable record of technical success.

Indeed, as most intracranial stenotic atheromatous lesions are eccentric, and because of the risk for elastic recoil, angioplasty usually results in subtotal vascular diameter restoration. Until the advent of flexible coronary stents that were mounted on small delivery systems, however, access to the intracranial circulation through the tortuosities of the cervical arteries was not possible.

Intracranial Stenting Studies

The first reported use of a stent in the intracranial circulation, in 1997, was not for atheromatous disease, but for the treatment of a ruptured intracranial aneurysm, and is attributed to Higashida and coworkers[35] who used a 15-mm Palmaz-Schatz stent to treat a broad-based, fusiform, vertebrobasilar artery aneurysm, which had caused repeated subarachnoid hemorrhages in a 77-year-old patient. The stent was used to cover the base of the aneurysm, so as to prevent backward protrusion of electrodetachable coils, which were delivered into the aneurysm sac. The procedure was successful technically and the patient had a good outcome. Several reports then followed on the use of stents for the treatment of intracranial disease.[36–39] In 1999, Horowitz and colleagues reported on the technical feasibility and success with total luminal reconstruction of the basilar artery in three patients using flexible, balloon-mounted coronary stents.[36] Two patients had stenoses and one had complete basilar occlusion; one patient developed a pontine stroke, and made a reasonable recovery. In 2000, Gomez and colleagues[37] reported on successful stent-assisted reconstruction of the basilar artery in 12 symptomatic patients ranging in age between 40 and 82; arterial stenoses were reduced from a mean of 71.4% to 10.3%. There were two perioperative events, including a patient who developed sixth and seventh nerves pareses, with reported total resolution within 8 weeks. Only one patient had recurrence of symptoms at a mean follow-up of 5.9 months (range 0.5–16 months); that patient presented with TIAs, which were attributed to a severe stenosis of the basilar artery proximal to the stent, managed

successfully with angioplasty. Also in 2000, Gomez and colleagues reported on the successful stent-assisted reconstruction of a symptomatic middle cerebral artery stenosis.[38] Phatouros and coworkers[39] reported on the successful use of coronary stents in two patients who had severe persistent posterior circulation symptoms from severe midbasilar stenoses. In both patients, the basilar artery lumen was reconstructed fully, and there was no recurrence of symptoms at a mean follow-up of 6.5 months. Joseph and coworkers[40] described the successful use of a stent to treat a basilar stenosis, which had resisted several attempts at balloon angioplasty.

Levy and colleagues reported in 2001[41] on 11 patients who had symptomatic posterior circulation disease treated with stenting. There were four periprocedural deaths, including a delayed death after a large pontine stroke, and one pontine stroke resulting in permanent diplopia, for a rate of major complication or death of 36%. Seven patients (64%) had resolution of their symptoms. Of the five survivors, two (40%) had angiographic demonstration of restenosis. In 2004, Jiang and colleagues[42] reported the largest series to date of patients who had middle cerebral artery stenoses involving various locations of the vessel that were treated with stenting. Overall technical success was 97.6%. Lesions were classified by level of expected technical difficulty with vascular access. Type I lesions (n = 17) had mild-to-moderate tortuosity and smooth access, type II (n = 18) had severe tortuosity or irregular arterial wall, and type III (n = 7) displayed excessively severe tortuosity. Technical success was 100% in types I and II lesions and 85% in type III lesions. Mortality was 0% in types I and II lesions and 25% in type III lesions. Follow-up (mean 10 months) was available in 38 of 40 patients, none of whom had recurrence of their presenting TIAs. Angiographic follow-up was reported for only 8 of 40 patients, one of whom demonstrated restenosis.

Clinical Trials for Intracranial Stenting

As this growing body of reported experience continued to provide answers to longstanding questions by establishing the technical feasibility of stenting for intracranial artery reconstruction, the need for anwers to new questions also was becoming apparent. Similar to the concerns of possible increases in iatrogenic neurologic and non-neurologic complications that could be associated with the uncontrolled widespread practice of stenting of carotid arteries, particularly for asymptomatic disease,[43] the lack of scientific legitimacy and of a clear, organized process for the decision making, the control of technical standards, and the perioperative management has generated a degree of consternation.

Several factors, including the prevalence of the disease, the lack of a suitable pharmacologic solution, the increasing experience with endovascular techniques, and the strong impetus from the industry, had to lead to the initiation of clinical trials. Two clinical studies have been conducted to date, both of which were sponsored, however, by manufacturing companies.

The first trial to evaluate the effect of endovascular therapy on intracranial stenotic disease, the results of which were published in 2004,[44] was the Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVIA). The trial was a prospective, multicenter, company-sponsored (Guidant, Indianapolis, Indiana) study aimed at assessing the feasibility of intracranial stenting. The trial was conducted between November 2000 and November 2001, and enrolled 61 patients, ages 18 to 80, who had a single, symptomatic stenosis (>50%) of the intracranial arterial circulation (n = 43) or the extracranial vertebral artery (n = 18). Patients were included if they had a stroke (more than 7 days prior) or a TIA (more than 24 hours prior) and if they could take two antiplatelet agents; patients were excluded if they had a concomitant intracranial tumor, arteriovenous malformation, or aneurysm (larger than 5 mm). Primary endpoints were (1) death or stroke rate within 30 days of treatment and (2) stent success, defined as lack of greater than 50% restenosis, and coverage of no more than the length of the vascular lesion.

Successful placement of the Neurolink balloon-mounted stent (Guidant, Indianapolis, Indiana) was possible in 95%, with a morbidity of 6.6% and no mortality. Significant (>50%) restenosis at 6 months was 32% in the intracranial arterial circulation and 42% in the extracranial vertebral arteries; 39% of all patients who had restenosis were symptomatic. Also, there was a 7.3% rate of late strokes (>30 days after treatment). Based on the results of the study, the Neurolink device was granted humanitarian device exemption status by the Food and Drug Administration (FDA) in 2005 to treat symptomatic patients.

Another trial, also industry-sponsored, was published in 2005.[45] The WingSpan study reported the results of the use of balloon angioplasty followed by the delivery of a self-expanding stent in 15 patients who had symptomatic stenoses that were resistant to medical therapy.[45] Mean arterial stenoses were 72%, and were reduced to 54% after angioplasty, then 38% after the delivery of a stent. There was neither mortality nor permanent

morbidity in any of these patients. An update to the WingSpan study was presented at the 2005 meeting of the American Society of Neuroradiology, which reported a 4.4% combined mortality and ipsilateral stroke rate at 30 days and a 7.1% stroke rate at 6 months.[46]

The WingSpan device also was granted humanitarian device exemption status by the FDA in 2006 to treat symptomatic patients (**Fig. 1**).

The NIH-sponsored SAMMPRIS (Stenting versus Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis) trial, scheduled to start late 2008, will target high-risk patients, who presented with TIA or stroke and 70–99% intracranial stenoses.[47] A relative benefit of at least 35% of intracranial angioplasty and stenting combined with intensive medical therapy over maximum medical therapy alone is expected over a two-year follow-up period. Patients will be treated with the Wingspan intracranial stent and the Gateway balloon (Boston Scientific, Natick, Massachusetts). As there is growing concern about recurrent stenosis and occlusion in relation to the use of the Wingspan stent,[48] the data from the SAMMPRIS trial will no doubt be awaited with great interest.

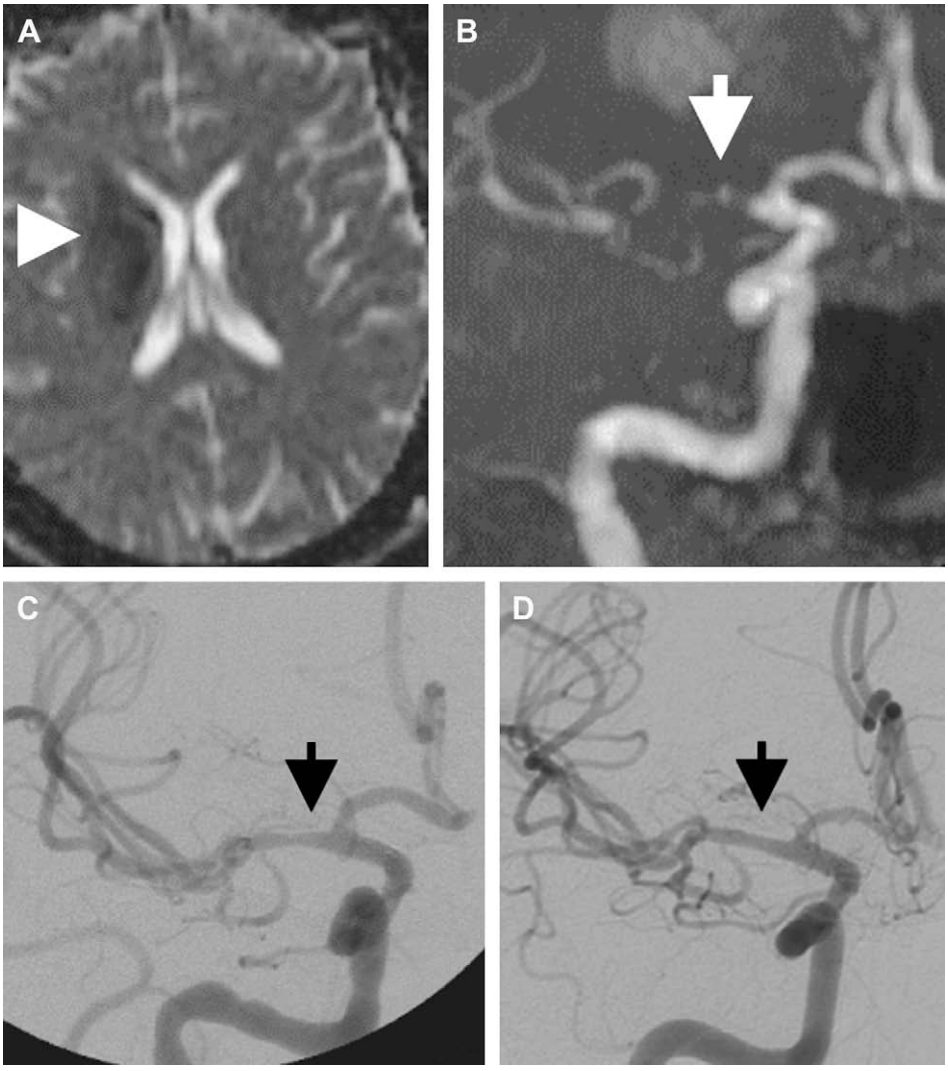

Fig. 1. A 57 year old diabetic, hypertensive male smoker s/p car accident due to transient loss of consciousness. (*A*) MRI shows a right caudate/subcortical infarct (*arrowhead*). (*B*) MRA suggests a tight right middle cerebral artery (MCA) stem stenosis (*arrow*), confirmed at angiography. The patient was placed on Aggrenox, but motor symptoms recurred. (*C*) The right MCA was treated with a Wingspan® stent, with a persistent "waist" in the middle of the stent (*arrow*). (*D*) A few weeks later, further stent expansion has resulted in total MCA reconstruction (*arrow*).

Biologically Active Stents

To date, little experience exists with the use of biologically active or drug-eluting stents in intracranial disease. Significant expectations are placed in these agents, designed to inhibit intimal proliferation and ensuing arterial stenosis.

Abou-Chebl and coworkers[49] reported on eight patients who had symptomatic intracranial stenoses (>70%) involving the intracranial internal carotid artery (n = 3), the middle cerebral artery (n = 2), the basilar artery (n = 2), and the vertebral artery (n = 1). In four patients, a sirolimus-coated stent (Cypher, Cordis, Johnson & Johnson, Warren, New Jersey) was used, and the other four received a paclitaxel-coated stent (Taxus, Boston Scientific). As discussed previously, sirolimus (rapamycine) inhibits smooth muscle proliferation by enzymatic blockade, whereas paclitaxel acts by inhibiting the cell cycle. All procedures technically were successful, despite one complication (one retinal artery embolus). All patients were followed with vascular imaging at 3, 6, and 12 months. There were no restenoses at a mean follow-up of 12 months (range 2–17.3 months).

Although the technical feasibility of intracranial arterial stenotic disease treatment using biologically active stents is demonstrated clearly, what is still lacking is an understanding of which lesions are at the most risk for restenosis, from either neointimal hyperplasia or recurrent atheromatous disease. Also, little is known currently of the long-term biologic effects of drug-eluting stents.[50] Biologically active stents may represent a major advancement in the treatment of intracranial atheromatous disease, which requires further investigation (**Fig. 2**).

Summary of Current Evidence

Currently, reported follow-up times are long enough that some reliable data are available. A large retrospective, multicenter study of 120 patients who had a mean follow-up of 42.3 months demonstrates annual stroke rates of 3.2% in the treated arterial territory and of 4.4% for all strokes.[51] Another series of 12 patients who had basilar stenoses treated with stents provided a median follow-up of 24 months (range 6–36 months), during which there was no recurrence of symptoms or of arterial stenotic disease.[52]

In conclusion, available evidence to date is sufficient to recommend the performance of endovascular intracranial revascularization in patients who have symptomatic intracranial stenoses that are resistant to medical therapy. Such recommendation has been officially embraced by medical societies that represent interventional physicians involved in the management of cerebrovascular disease (the American Society of Interventional and Therapeutic Neuroradiology, the American Society of Neuroradiology, and the Society of Interventional Radiology).[22] As these are high-risk patients, because of their presenting disease and their significant associated comorbidities, it is crucial that they be managed by skilled multidisciplinary teams and equally imperative that the level of expertise and experience of the treating interventionalist be significant, so as to offer the highest possible benefit for the lowest possible periprocedural risk. All such concepts, as apply to extracranial carotid disease, are stated by the Stroke Council of the American Heart Association.[53]

STENTING FOR INTRACRANIAL ANEURYSMS

As discussed previously, the first clinical report on the use of a stent to treat a broad-based intracranial aneurysm was published in 1997; the investigators successfully addressed a ruptured distal vertebral artery aneurysm with the help of a Palmaz-Schatz stent.[35]

In vivo animal studies demonstrate that complete thrombosis can be induced in sidewall aneurysms after placement of a stent across the aneurysm neck, by remodeling of the arterial lumen as an endothelial layer forms on the surface of the stent. In most aneurysm configurations encountered in living humans, however, stents alone are not sufficient to eliminate aneurysms because of complex interactions between flow dynamics and mechanical properties of stents, including high porosity inherent to currently manufactured stents, and perioperative use of anticoagulation or antiplatelet therapy.[54,55] Hemodynamic studies of aneurysm models demonstrate that stents placed across aneurysms induce complex flow changes, including stagnation and reduction of flow vortices within the aneurysm sac and decreased flow in the parent vessel.[54–56] Further, the precise assessment of flow dynamics in and around aneurysms using computational fluid dynamics (CFD) simulations could lead to the optimization of stent designs, tailored to various configurations so as to maximize flow stagnation.[56,57] The Pipeline Neuroendovascular Device (Pipeline NED; Chestnut Medical Technologies, Inc, Menlo Park, California) is a new stent which is raising great expectations.[58,59] It is a braided, tubular, bimetallic microstent which has been shown to create significant flow disruption along aneurysms necks. However, despite having very small struts, its biocompatibility and hemocompatibility allow the preservation of both parent arteries and small,

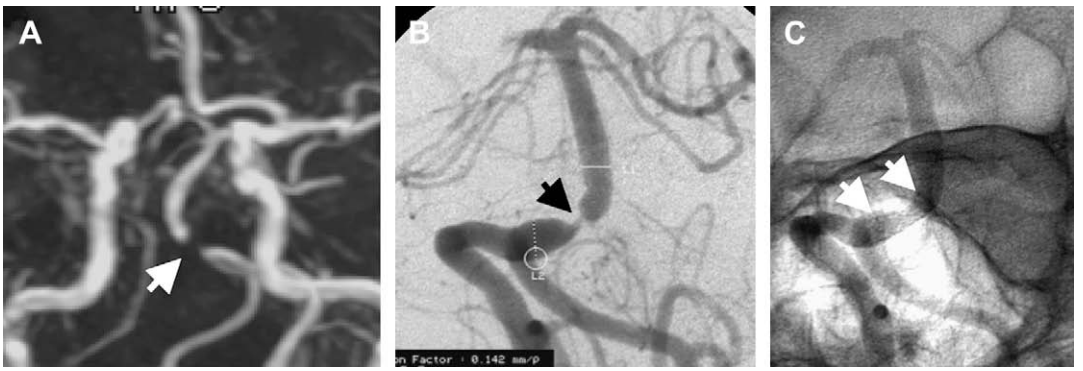

Fig. 2. A 74 year old male with crescendo TIAs and small cerebellar infarcts. (*A*) Severe mid-basilar stenosis revealed by MRA (*arrow*), (*B*) confirmed by angiography (*arrow*). (*C*) Stent-assisted reconstruction of the basilar artery with a balloon-mounted sirolimus coated stent (*arrows*) results in excellent clinical response with disappearance of ischemic episodes (16 month follow-up).

adjacent perforating arteries. The device is widely anticipated as a significant improvement in the treatment of broad-necked aneurysms, more particularly dissecting aneurysms, and intracranial dissections.

In 2000, Phatouros and coworkers[60] reported on the use of balloon-mounted coronary stents in a series of seven patients who had wide-neck and fusiform aneurysms and pseudoaneurysms of the cervical and vertebrobasilar arteries, which were not amenable to unsupported coil embolization. There were no clinical complications in their series, and all procedures were technically successful, despite recognized difficulties advancing stents into the intracranial circulation.

Lylyk and colleagues[61] reported in 2002 on a large series of patients who had various conditions, 72 of whom had complex intracranial (broad-based, dissecting, or fusiform) aneurysm, 82% of which were treated with a combination of stent and coil obliteration. Again, results were favorable and overall morbidity acceptable. Vanninen and colleagues[62] reported in 2003 on the use of two stents, one within the other, to induce thrombosis successfully within a previously ruptured intracranial vertebral aneurysm.

Technical difficulties associated with advancing balloon-mounted coronary stents in intracranial circulation and subsequent risks for thrombosis associated with the devices remained limiting factors with the technique.

In 2004, the Neuroform (Boston Scientific/Target Therapeutics) microstent was released and granted humanitarian device exemption status by the FDA to treat wide-necked intracranial aneurysms.[63]

The Neuroform device is a nickel-titanium, self-expandable, ultraflexible stent, with diameters ranging between 2.5 and 4.5 mm and lengths ranging between 10 and 30 mm. The interstices of the stent measure slightly less than 1 mm (2–2.5 F), allowing the placement of a microcatheter through the stent into the aneurysm sac. Also, the Neuroform stent exerts minimal radial force on the surrounding artery, estimated at 10 mm Hg, approximately one order or magnitude less than that of self-expanding stents used in the coronary or carotid circulations.

Patients receiving the stent had to be treated with clopidogrel for 48 hours before, and at least 3 months after, the placement of the device, which limited its use in freshly ruptured aneurysms.

The Neuroform device was embraced immediately, and several series of successfully treated patients soon were reported.[64–66]

Despite its easy use and relative safety, complications soon were reported, including delayed in-stent thrombosis and infarction,[66,67] delayed in-stent stenosis in a middle cerebral artery attributed to neointimal hyperplasia,[68] and several technical difficulties with delivery,[69] addressed partially by improvements in the delivery design.

The Neuroform stent is shown to be safe and compatible with MRI, including at field strengths of 3 Tesla.[70] The Cordis Enterprise Vascular Reconstruction Device and Delivery System (Cordis, Johnson & Johnson, Warren, New Jersey) was granted humanitarian device exemption status by the FDA in December 2006. The Cordis Enterprise microstent is a particularly flexible, polymer-coated, Nitinol stent with a closed cell design, which is easy to deliver as it comes pre-loaded on the delivery wire inside an introducer.[71,72] One current limitation of this stent is that it cannot be used safely in arteries that are wider than 4.5 mm.[73] In addition to its extreme flexibility (**Fig. 3**), the Enterprise microstent is reconstrainable. This property has allowed the stent to be used in an occluded

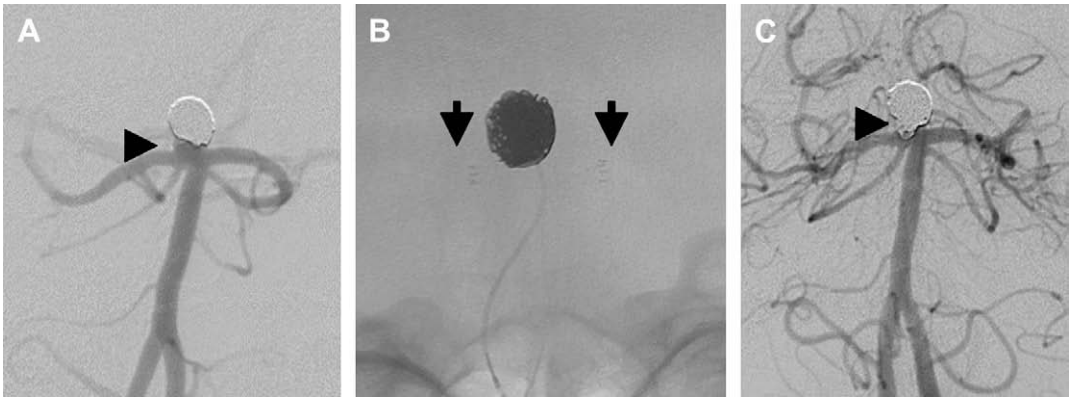

Fig. 3. (*A–C*) A 62 year old female with severe headaches from unruptured basilar apex aneurysm. Initial treatment resulted in total aneurysm obliteration. (*A*) There is recurrence of headaches 12 months later, due to neck recurrence (*arrow*). Placement of a highly flexible stent (Enterprise, Cordis, Johnson & Johnson) through the posterior communicating artery across the basilar terminus (*B*) (*arrows*) permits dense aneurysm coil re-packing (*C*) (*arrow*), resulting is elimination of headaches.

middle cerebral artery as a ''temporary bypass'' to disrupt and circumferentially displace an acute thrombus, producing immediate revascularization of the distal territory; the stent was reconstrained and removed a few minutes later, after successful thrombolysis.[73]

Ruptured dissecting aneurysms, in particular those of the basilar artery, remain a major challenge, for which the advent of stenting in addition to coil-packing, albeit representing progress, may not be the final solution because of the extensive nature of the disease.[74–76] Although successful treatment of a dissecting aneurysm with a stent alone is reported,[75] some have cautioned that the treatment of dissecting aneurysms with stents is not effective, as there is a significant chance of recurrence of hemorrhage.[76] The Pipeline microstent[58,59] might represent a major advance in dissecting aneurysms.

STENTING FOR INTRACRANIAL ARTERIAL DISSECTIONS

As for the extracranial circulation,[77] the treatment of intracranial dissections with stents makes a lot of sense. Lanzino and colleagues[78] and Malek and coworkers[79] reported on the successful use of coronary stents to treat iatrogenic dissections of the basilar artery resulting from attempted balloon angioplasty performed for symptomatic ischemia.

The successful management of spontaneous posterior circulation dissections using angioplasty and stenting[80] or stenting alone[81] increasingly is reported. One particular concern compared with the extracranial circulation is the presence of perforators, as precise delineation of the intimal flap is difficult to assess. In intracranial dissections too, a small strut and highly hemocompatible device such as the Pipeline stent[58,59] might offer great promise.

STENTING FOR INTRACRANIAL VENOUS OCCLUSIVE DISEASE

Pseudotumor cerebri, also referred to as benign intracranial hypertension or idiopathic intracranial hypertension, is defined as a condition of raised intracranial pressure in the absence of a mass lesion or cerebral edema. It is characterized by headache and visual deterioration that usually resolve with time, although it may culminate in blindness and major disability.

Various mechanisms are implicated, including increased cerebrospinal fluid (CSF) production, decreased CSF absorption, idiopathic brain edema, and idiopathic intracranial venous hypertension.[82]

Dural sinus venous obstruction increasingly is implicated as the cause of the syndrome in some patients, including those who have morbid obesity, in whom it is believed that increased abdominal pressure is transmitted through the thorax to the cerebral draining veins. This usually is suspected on noninvasive imaging, such as magnetic resonance venography or contrast-enhanced CT of the head. Confirmation requires manometric measurements within the intracranial dural venous sinuses, most commonly the transverse sinuses, demonstrating considerable proximal to distal pressure gradients. Support for this mechanism comes from growing accounts of improvement of the condition after stenting of the dural sinuses.

Marks and colleagues[83] were the first to report on successful use of stenting to relieve pseudotumor cerebri in two patients.

Several subsequent reports and small series have demonstrated further the effectiveness of venous reconstruction in pseudotumor cerebri using stents in selected patients.[84–86] As in the arterial circulation, the possibility of restenosis from neointimal hyperplasia exists and is reported.[87]

Despite accumulating evidence of effectiveness of venous stenting in selected patients who have pseudotumor cerebri, the lack of large clinical trials is raising legitimate concern.[88]

SUMMARY

The use of stent-assisted techniques to manage a variety of cerebrovascular conditions has exploded in recent years. Although the safety of devices is expected to continue to improve, the absence of scientific validation remains an issue in many indications. In posterior circulation arterial disease, considering the absence of valid and reasonable surgical options and the results of the WASID trial, there is widespread consensus that endovascular therapy will become the main option. In anterior circulation intracranial disease, surgical revascularization (EC/IC bypass) may continue to remain an option in selected patients, although it is unclear whether or not randomized clinical trials would be either useful or feasible. The treatment of wide-necked intracranial aneurysms has benefited greatly from the advent of stenting. Intracranial arterial dissections are uncommon and life threatening enough for stenting to remain a major, if not the only, treatment option in many; flexible covered microstents may become the preferred treatment in arterial segments that do not harbor perforators. Concerning the endovascular management of pseudotumor cerebri, as more insight is gained into the epidemiology and the pathophysiology of the disease, it is likely that validation against conventional surgical shunt techniques will be required, at least in subgroups of patients. There is no doubt at this point that large numbers of patients will continue to benefit from the technique. A strong focus on patients' specific needs, a thorough multidisciplinary approach, and continuing efforts in research are necessary to help maintain procedural risks as low as possible.

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