Intracranial Stenting for Cerebrovascular Pathology
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Learning Objectives: After reading this article, the participant should be able to:
1. Describe the use of stent-assisted angioplasty for intracranial atherosclerotic disease.
2. Recall the present status of stent-assisted management of intracranial aneurysms.
3. Describe the role of stenting and its technical aspects in the treatment of arteriovenous fistulae and acute stroke.

Only within the past few years have technological advances made it possible to produce stents capable of negoti-ating the tortuosity of the intracranial circulation. Recently, the development of smaller, more pliable stents and delivery devices has greatly broadened the application of intracranial stenting for various clinicopathologic disease processes. In this review, we discuss the utility of stent implantation for the following cerebrovascular diseases: intracranial atherosclerosis and stenosis; arterial dissection; fusiform and wide-necked aneurysms; venous occlusive disease; and acute vessel thrombosis. We also describe some of the basic technical aspects of intracranial stent insertion, along with the periprocedural medical management of patients.

Historical Background
Although intracranial applications for stent technology are merely a few years old, these intravascular devices have been in existence for more than three decades. In a review of neurovascular stenting by Horowitz and Purdy, pioneering work by several groups is described. Dotter and Judkins first proposed the concept of intravascular stenting in 1964. Shortly thereafter, animal research reported by Dotter in 1969 demonstrated long-term patency in the dog popliteal artery following intravascular placement of a coil-spring tube graft. More than a decade later, a second report by Dotter described the use of nitinol stents in the canine vasculature. Throughout the early 1980s, several investigators tested stents in various animal models. In 1987, Rousseau et al. described the use of nitinol stents in the canine vasculature. Throughout the early 1980s, several investigators tested stents in various animal models. In 1987, Rousseau et al. described the first case of stent placement within a human coronary artery.

Category: Vascular

Key Words: Cerebrovascular, Atherosclerosis, Aneurysm, Stroke, Arteriovenous fistula
Atherosclerotic Disease: Stent-Assisted Angioplasty

Before the availability of intracranial stents, symptomatic, medically refractory atherosclerosis of the intracranial vasculature was treated primarily with balloon angioplasty. Although balloon angioplasty has demonstrated efficacy in the coronary vasculature, cerebral vessels have less adventitia and are surrounded by cerebrospinal fluid and, therefore, may be at greater risk for dissection and abrupt closure. Subsequent to the initial report by Sundt et al. in 1980, several advances in both technique and devices have increased the efficacy of intracranial balloon angioplasty. Nearly a decade would pass before Ahuja et al. and other clinicians reported multiple cases with more widespread application of intracranial angioplasty.

In a recent report by Connors and Wojak, a cohort of 50 patients received angioplasty in which the balloons were undersized and were inflated slowly and deliberately. Even though 16% of this group had residual stenosis of more than 50% of the luminal diameter, none of them had strokes or suffered acute occlusion. These authors admit that the slow inflation technique and undersizing may yield suboptimal angiographic results but feel that it greatly decreased the likelihood of intimal damage and acute thrombosis. Although postangioplasty results may appear suboptimal in many cases (because of recoil and residual stenosis), Derdeyn et al. have described restoration of normal cerebral blood flow and oxygen extraction despite residual stenosis of 40% in the setting of supraclinoid internal carotid artery stenosis that caused misery perfusion (inadequate perfusion relative to metabolic demands of the parenchyma ipsilateral to the stenosis). Similar improvements in cerebral blood flow and oxygen extraction have been reported after basilar artery angioplasty.

As suggested by Mori et al., the morphology of the lesion often determines the suitability of balloon angioplasty. Lesions that are tortuous, angulated, or longer than 10 mm (type C) have a restenosis rate of 100% at 1 year and a stroke risk of 87% at 1 year, with only a 33% likelihood for immediate success. Lesions less than 5 mm long with a concentric configuration (type A) are well suited for angioplasty, with a 92% immediate success rate and no restenosis at 1 year. Type B lesions are 5 to 10 mm in length and eccentric, and may or may not be best suited for angioplasty, as evidenced by a 33% rate of recurrent stenosis at 1 year.

To date, several small retrospective series have examined the angiographic and clinical outcomes in patients with intracranial atherosclerotic disease treated by use of intracranial stents. In a series reported by Gomez et al., 12 basilar artery stenoses were treated with stents. There were no permanent significant complications such as vessel occlusion, stent thrombosis, or vessel rupture. The mean residual stenosis was 10% (before stenting, the mean stenosis was 71%). Although the results of this series suggest that intracranial stenting is feasible with low morbidity, other clinicians have reported significant mortality and morbidity rates following stenting of the intracranial posterior circulation. In a series of 11 patients treated with stent-assisted vertebrobasilar angioplasty, Levy et al. reported three periprocedural deaths and one delayed death from procedure-related brain stem infarction. Among the survivors, follow-up angiography revealed in-stent hyperplasia in one patient, a new stenosis proximal to the stented lesion in a second patient, and in-stent aneurysm formation in a third patient.

Some atherosclerotic lesions in the intracranial circulation may be less amenable to balloon angioplasty alone because of the bony confinements of the cerebral vasculature. The petrous portion of the carotid artery is especially refractory to balloon dilatation. In a report by Fessler et al., angioplasty of a flow-limiting stenotic lesion of the petrous...
carotid artery resulted in minimal improvement in the diameter of the vessel lumen. This suboptimal result most likely was due to the limited capability for vessel expansion in the carotid canal. In such cases, stenting may improve postangioplasty vessel lumen diameter. Stenting for atherosclerotic stenosis also may be especially useful for stenotic lesions that recur after angioplasty or following minimally responsive luminal dilatation to angioplasty alone. Following angioplasty, restenosis results from intimal injury–induced fibrosis. Stenting of these lesions has a lower risk of embolic shower as compared to stenotic lesions resulting from atheromatous plaques. In addition, these fibrous lesions tend to have better post-stent angiographic results.

Technical performance of intracranial stenting involves the following key stent parameters: sizing of the stent to the vessel lumen; “trackability” (the ease with which the tortuosity of the intracranial vessels can be negotiated); thickness; porosity; flexibility; balloon overhang (length of stent relative to balloon); adherence; rate of balloon inflation; and outward radial force exerted by the stent. Several patient-dependent anatomical considerations often dictate which stent will be used. These factors include vessel tortuosity and flexibility, lumen diameter, distortability of the vasculature, and the inherent curvature of the vasculature (which is extremely low for vessels surrounded by a bony canal). Aortic arches with bovine configurations or a severely angulated vessel off of the aortic arch present even more complexity. All of these anatomic factors must be appreciated when choosing the optimal stent design for the procedure. Stent selection should be based on these parameters as they relate to the individual patient’s anatomy.

For atherosclerotic lesions of the intracranial circulation, it may not be necessary to achieve a vessel diameter equivalent to that of the parent vessel. On the other hand, a poor technical result may increase the possibility for subacute stent thrombosis or restenosis (Fig. 1). These lesions develop slowly, often allowing the brain to obtain supply from the collateral circulation as a compensatory mechanism for augmenting flow to hypoperfused regions. Thus, even moderate improvements in luminal diameter may provide sufficient restoration of flow. As is observed in conjunction with carotid endarterectomy procedures, recanalization of a clinically significant, high-grade intracranial stenosis may cause reperfusion hemorrhage. Oversizing stents in an attempt to achieve minimal residual stenosis also increases the risk of vessel rupture and dissection. In addition, stents that are deployed rapidly and with high outward radial force increase the risk of dissection. Finally, stents differ in trackability. It is important that the stent chosen for stent-assisted angioplasty be able to navigate through turns without significant resistance to increase the likelihood of placement and reduce the risk of vessel injury.

**Stent-Assisted Aneurysm Management**

In the future, endovascular techniques eventually will replace surgery for intracranial aneurysms. Current techniques result in a significant rate of regrowth because of coil compaction and incomplete obliteration. Hemodynamic studies suggest that endosaccular occlusion with current coil technology most likely will not cure aneurysms without complete elimination of neck remnants, especially at the inflow zone. Stents represent a method of neck remodeling with significant diversion of flow from the aneurysm inflow zone. Stenting of fusiform aneurysms, described by Higashida et al. and by our group, may obviate the need for these technically challenging and risky procedures.

![Figure 1. A, Digital subtraction angiogram of a right vertebral artery injection demonstrates bilateral vertebrobasilar junction high-grade stenosis with reflux of contrast down the left vertebral artery. B, Angiogram following stent placement in the distal right vertebral artery demonstrates excellent resolution of the stenotic segment. Reflux of contrast into the left vertebral artery is no longer seen.](image-url)
Initially, both Guglielmi detachable coils (Boston Scientific/Target, Fremont, CA) and a stent were used for the endovascular management of these types of cases. Currently, however, we and other investigators are advocating the use of low-porosity stents (i.e., without coils) as the primary treatment for vertebrobasilar fusiform aneurysms. Using in vitro side-wall aneurysm modeling, Lieber et al. demonstrated that aneurysm flow from the parent vessel lumen and into the aneurysm could be controlled with low-porosity stents. It seems logical that stents with sufficiently low porosity should be able to recreate normal intraluminal laminar flow within the parent vessel with concomitant aneurysm thrombosis. Using laser-based imaging technology, investigators have demonstrated reduced flow vortices into aneurysms, with resultant stagnation and eventual thrombosis following stent placement.

In addition to fusiform vertebrobasilar aneurysms, intimal dissections (which may develop into pseudoaneurysms) can be treated effectively with stents. As detailed by Lylyk et al., dissecting aneurysms can be treated with stent-assisted coiling. In another report, Mericle et al. described stenting of a pseudoaneurysm arising from a dissection of the horizontal petrous internal carotid artery. Stenting of this lesion prevented coil herniation into the parent vessel and obviated the need for tight coil packing into a friable pseudoaneurysm dome. Malek et al. reported on an iatrogenic dissection of the basilar artery following angioplasty and stenting of a stenotic segment. This dissection was managed by deploying a tandem stent that covered the intimal flap, thereby restoring flow through the basilar artery. It has been our experience that low-porosity MagicWall stents (Boston Scientific, Natick, MA) may be sufficient to treat these fusiform and pseudoaneurysms and dissections without the additional need for coils.

Before the development of remodeling techniques and intracranial stents, wide-necked aneurysms posed great challenges to endovascular surgeons. Coil herniation into parent vessels is more likely to occur in aneurysms with fundus-to-neck ratios of less than 2. In addition, tight packing of these aneurysms is more difficult, with follow-up angiography demonstrating coil compaction over time. Stent-assisted coiling of these aneurysms provides a buttress to protect the parent vessel from coil herniation, diverts flow from the aneurysm inflow zone, and allows for tighter coil packing (Fig. 2). Case reports from Sekhon et al., Lavine et al., and others have demonstrated optimal angiographic results following stent-assisted coiling of wide-necked aneurysms.

In a series of 10 patients reported on by Lanzino et al., aneurysms were treated with either stent-assisted coiling or

![Image](Figure 2. A, Anteroposterior (left) and lateral (right) angiographic views demonstrates a large basilar trunk aneurysm. B, Unsubtracted angiogram demonstrates the stent acting as a buttress against the coil mass. C, Angiogram of the basilar artery following successful stent-assisted coiling of the aneurysm with preservation of the anterior inferior cerebellar arteries.)
stenting alone. No permanent complications occurred, and good angiographic results (>90% occlusion) were achieved in patients treated initially with stent-assisted coiling. In a wide-necked aneurysm model created by Imbesi and Kerber, the placement of a stent across the aneurysm neck was sufficient to disturb the slipstreams entering the aneurysm. Additional coil deposition caused even further disruptions of the slipstreams into the aneurysm. Such hemodynamic alteration may result in thrombosis of the aneurysm.

New liquid embolic agents show promise in the stent-assisted management of wide-necked aneurysms. The advantage of these agents over coils is that they can assume the configuration of the aneurysm sac without leaving residual dead space. Ideal coil packing of an aneurysm results in only 30% to 40% occlusion of the fundus volume. Although higher rates of complete endosaccular occlusion might be achieved with the use of liquid embolics, parent vessel protection often is required to prevent extrusion of the agent into the vessel. Parent vessel protection can be achieved by means of balloon occlusion or stent placement. Animal studies by Murayama et al. and by our group have demonstrated excellent results with a combination of liquid embolic agent and stent placement (with and without concomitant deposition of coils) for the treatment of wide-necked aneurysms. Although some fear that jailing of vessels by stent struts may lead to small branch vessel occlusion, the strut diameter that is currently used intracranially is insufficient to occlude these vessels. As demonstrated by Wakhloo et al. in animal and in vitro studies, the pressure differential between parent arteries and their side branches maintains vessel patency across stent struts that do not cover more than 50% of the branch vessel orifice. Risks incurred by treatment of aneurysms with liquid embolics include leakage of the agent into the parent vessel, resulting in ischemia and possible vessel thrombosis.

Arteriovenous Fistulae and Venous Occlusions

Dural sinus thrombosis resulting in intracranial hypertension may cause symptoms ranging from headaches to blindness and even brain death. In many cases, dural sinus thromboses can be treated by using pharmacological thrombolysis, mechanical clot evacuation, or anticoagulation therapy. Complex, symptomatic arteriovenous fistulae may require a combination of these interventions.

Murphy et al. describe a case of a type IV transverse sinus arteriovenous fistula with a transverse sinus thrombosis in which the patient was treated with mechanical thrombolysis followed by the insertion of multiple stents. Stents were placed from the transverse sinus to the internal jugular vein. This procedure re-established normal drainage and simultaneously obliterated the dural arteriovenous fistulae. Malek et al. describe the case of a 13-year-old-boy with aphasia and right hemiparesis secondary to multiple dural arteriovenous fistulae and posterior sagittal, bilateral transverse, and occipital sinus occlusions. After failure of medical therapy, angioplasty of the left transverse sinus and occipital sinus was performed. Over the next few days, the occlusions recurred. Repeat angioplasty followed by stenting of the occipital sinus was performed. At 3 months’ follow-up, the occipital sinus was patent and was the significant conduit for drainage of venous outflow. The patient was asymptomatic at the 12-month follow-up evaluation.

Stenting of occluded venous sinuses should not be considered first-line therapy but, rather, as one option for failed medical therapy or failed thrombolysis (medical and mechanical). The choice of a low-porosity stent in this setting seems preferable, because the stent may function both to maintain vessel patency and to seal arteriovenous fistulae sites.

Stent Placement for Acute Stroke

Despite many novel treatment devices and pharmacotherapies, acute stroke remains a leading cause of morbidity and mortality in the United States. In 1996, there were an estimated 600,000 to 730,000 new strokes. Although intrararterial thrombolytic agents have enabled clinicians to recanalize vessels, maintaining lumen patency—especially in the face of underlying focal stenosis—can be more difficult.

Following failed thrombolysis with intra-arterially administered therapeutic agents such as reteplase or abciximab, balloon angioplasty or microsnares sometimes are used to morselize clot. Should the clot be effectively lysed, the presence of residual stenosis or dissection can present a dilemma. The stenosis can be managed by subsequent stent insertion. In this setting, our group occasionally has used stents intracranially to treat significant residual stenosis. The rationale behind such an intervention is that residual narrowing may induce sluggish flow through diseased portions of the vessel, resulting in recurrent acute thrombosis. Recently, we have inserted stents in vessels that continue to occlude acutely despite maximal pharmacotherapeutic thrombolysis. In this setting, stent insertion has been used as a “last resort” following hours of failed mechanical and pharmacologic thrombolysis. Perhaps stents should be considered sooner for occlusions that seem resistant to conventional intra-arterial thrombolysis. In our experience, excellent angiographic results were achieved, but clinical outcome was poor due to prolonged ischemic time from large vessel occlusion (Fig. 3).

Periprocedural Medical Management

Catheters, wires, balloons, and stents all have the potential to cause intimal injury and subsequent thrombosis, embolus, and vessel occlusion. In addition, all devices are thrombogenic. When blood encounters foreign substances, a monolayer of platelets and fibrin becomes adherent, depending on the surface charge, chemical properties, and surface irregularities of the foreign body. Therefore, proper anticoagulation and antiplatelet premedication is essential before these devices are introduced within the intracranial circulation. Additionally, clinicians must be careful when using and dosing a variety of anticoagulants in settings of acute stroke or critical stenosis, as intracerebral hemorrhage can occur.

For most intracranial stent procedures, an intra-arterial or intravenous bolus of 70 U/kg of heparin is administered following catheterization of the common carotid artery. All saline flush bags are primed with heparin (1 U/mL). The
activated coagulation time should be kept between 250 and 300 seconds. If possible, patients should be placed on aspirin and clopidogrel for 2 to 3 days before the procedure or given a loading dose of 300 mg earlier that day.

Aspirin is the most widely used antiplatelet medication, but it is a weak platelet inhibitor. The mechanism of action is by inhibition of cyclooxygenase 1. Aspirin does not inhibit platelet aggregation or impede platelet adhesion or secretion. Therefore, thrombin and cellular proliferation from platelet-activated mitogenic activity are uninhibited with aspirin treatment alone.

Clopidogrel is often used synergistically with aspirin. Clopidogrel is a thienopyridine derivative that inhibits the binding of adenosine diphosphate to its platelet receptor and prevents platelet aggregation. The antiplatelet action is irreversible and lasts for 7 to 10 days.

Evidence in the cardiac literature supports the use of combination antiplatelet regimens. In the Stent Antithrombotic Regimen Study, the rate of complications such as myocardial infarction, death, repeat angioplasty, or requirement for coronary bypass at 1 month was reduced by 80% for the aspirin and ticlopidine group as compared with the aspirin-only group. The advantage of a combination antiplatelet regimen is supported in animal models as demonstrated by marked reduction in the deposition of platelets and fibrinogen on stents placed in baboons treated with combination aspirin and clopidogrel versus those treated with aspirin alone.

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Some clinicians use IIb/IIIa inhibitors along with aspirin and either lower-dose heparin (40 U/kg) or no heparin with low rates of intraprocedural complications. A platelet glycoprotein (GP) IIb/IIIa receptor-specific antibody, abciximab, prevents the binding of fibrinogen to these platelet receptors, thus inhibiting platelet aggregation. When administered intravenously, abciximab has a half-life of 10 minutes, but its pharmacologic action lasts for 48 hours (although it is reversed easily with platelet transfusion). Eptifibatide, a cyclic peptide that may be a more specific inhibitor of GP IIb/IIIa receptors, is shorter-acting and may be less likely to result in hemorrhagic complications. Platelet inhibition lasts for 2 to 4 hours following administration of eptifibatide.

The coronary literature supports such anticoagulation regimens; however, there is a paucity of documentation in the neurosurgical literature. Heparin can be reversed with protamine, whereas platelet transfusions are needed to reverse the effects of abciximab.

Several studies in the cardiac literature suggest safety and trends toward benefits with these agents; however, few data have been reported in the neurosurgical literature regarding the use of these agents during and following intracranial endovascular therapies. Our preliminary data suggest that hemorrhage of a chronically ischemic brain is more likely to occur with GP IIb/IIIa inhibitors, so we do not administer these agents in patients with atherosclerotic disease causing high-grade stenosis or chronic hypoperfusion seen on single photon emission computed tomography studies. In patients without ischemic changes on preoperative magnetic resonance imaging, some clinicians administer a combination of intraprocedural heparin (one-time intravenous bolus of
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quent with phosphorylcholine, paclitaxel (Taxol), nitric oxide, and
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 manipulated across the lesion. The microcatheter is with-
removed, and a stiffer 300-cm 0.014-inch exchange wire is
vessel. Routine transfemoral angiography is performed,
place, and a 6-French guide catheter is then placed into the
30 to 60 minutes before the procedure. Following the proce-
dure, abciximab is infused intravenously for 12 hours at a rate
of 10 µg/min. In place of abciximab, epifibatide may be
administered with a loading dose of 135 (g/kg followed by
a 20- to 24-hour infusion of 0.5 (g/kg. Immediately after stent-
ing, patients who might be considered for short-term ther-
apy with GP IIb/IIIa inhibitors must undergo a computed
tomography scan to exclude postprocedural intracranial hem-
orrhage, which would preclude the use of these agents.
Following routine stent placement, heparin therapy is
discontinued but not actively reversed. Patients typically
are maintained on clopidogrel (75 mg daily) for 1 month
and on aspirin indefinitely. Modifications of anticoagula-
tion regimens depend on the indication for stenting (for
acute stroke, some interventionists would not discontinue
heparin immediately following the procedure) and any
complication that may have resulted.

Technique for Stent Placement
The technique used for stent placement varies slightly
from institution to institution. The following is a general
outline of the steps our group follows to place stents in the
intracranial circulation for stenting for most indications.
The procedure is performed in a suite with biplane digi-
tal subtraction angiography and fluoroscopic imaging capa-
ibilities. Following femoral artery puncture, a 6-French sheath
is inserted. A 5-French catheter is advanced over a 0.035-
inch hydrophilic wire into the aortic arch, and the artery of
interest is catheterized. Road-mapping technique is used.
The sheath and catheter are removed with the wire left in
place, and a 6-French guide catheter is then placed into the
vessel. Routine transfemoral angiography is performed,
and a 3-French catheter is advanced coaxially over a 0.014-
inch microwire into the intracranial vessel. The wire is
removed, and a stiff 300-cm 0.014-inch exchange wire is
then placed through the microwire. This system is then
manipulated across the lesion. The microwire is with-
drawn, and a balloon-mounted, over-the-wire stent is navi-
gated across the area of interest where it is deployed at 6
to 8 atmospheres of pressure. Angiograms are obtained fol-
lowing balloon deflation.

Future Directions
Novel modifications of stent surfaces using biomaterial
science and surface-coating techniques have opened a new
area in stent research. Investigators have demonstrated
some promising results in both human and animal studies
with the use of heparin-coated stents. Although rates of
subacute thrombosis with heparin-coated stents are lower
in some series, consistently significant reductions in resteno-
sis rates have not been demonstrated. Stents that have
shown promise in the cardiac literature are those coated
with phosphorylcholine, paclitaxel (Taxol), nitric oxide, and
rapamycin (sirolimus).

Of the many drug-coated stents under clinical investi-
gation, rapamycin-coated stents seem exceptionally promis-
ing. Zohlnh"fer et al. have shown that gene-expression
patterns of human neointima demonstrate the upregula-
tion of FKBP12 at the mRNA and protein level of human
neointima. FKBP12 is involved in controlling transform-
growth factor (TGF) beta receptor I signaling. Rapamycin,
which acts at the FKBP12 receptor site, has been shown to reduce neointima formation in animal mod-
els. It may be that arrest of neointimal formation and resid-
tant in-stent restenosis are due to the rapamycin effects of
blocking protein synthesis and concomitant induction of
cell-cycle arrest. Recent human trials suggest that
rapamycin-coated stents reduce restenosis rates. In one of
the first human trials of sirolimus-coated stents, reported
by Sousa et al., none of the patients treated with the coated
stents demonstrated restenosis at the 4- to 6-month follow-
up evaluation. The results from the Randomized Study
with the Sirolimus-coated Stent (RAVEL) European study of
220 patients were recently disclosed. RAVEL was a ran-
domized, multicenter, double-blinded study of sirolimus
balloon-expandable stents in patients with single lesions
in the coronary vasculature. According to the results of this
study, no restenosis was found at 6 months in patients
treated with the coated stents, and neointimal volume was
2% versus 37% in those treated with non-coated stents.
Although these data come from findings following stent-
ing of cardiac vessels, it may be that the same response will
be demonstrated in the intracranial vasculature.

A second drug-coated stent that has recently showed clin-
cal promise for inhibiting in-stent restenosis is the pacli-
taxel-coated stent. Paclitaxel inhibits restenosis is by altering
the stability of microtubules. This alteration leads to the
inhibition of cell replication and intracellular signaling. In
animal models, reductions in neointimal proliferation at
blood concentrations 100 times lower than antineoplastic
levels have been shown. In a recent pilot clinical trial, none
of the 21 patients demonstrated restenosis of stented coro-
nary lesions. The Asian Paclitaxel-Eluting Stent Clinical Trial
(ASPECT) trial compared the safety and efficacy of high-
dose and low-dose paclitaxel-coated stents versus uncoated
stents in patients with single lesions in coronary arteries. At
6 months, restenosis rates had dropped from 27% in the con-
trol group to 4% in the high-dose paclitaxel group. These
findings are attributed to reductions in volume of neointi-
mal hyperplasia. It will be interesting to see how the
rapamycin- and paclitaxel-coated stents will be applied in
the future for stenting of intracranial stenotic lesions.

Much interest exists in radioactive stents; however, the
results have been variable and seem to be dependent on the
animal model, time to sacrifice, and the radioactivity of the
stents. It is hoped that development and further research of
coated stents will provide clinicians with prosthetic devices
that locally inhibit platelet-fibrin deposition, mitigate clot for-
mation and restenosis, and promote local endothelialization.

Caveats
Intracranial stenting is a novel technique, still in its early
developmental stages. Although intracranial stents are being
used with increasing frequency, it is important to remem-
ber that there are no long-term data regarding rates of
patency, restenosis, or vessel injury. Additionally, the effects

70 U/kg for a target activated coagulation time of 200 sec-
onds) and a 0.25 mg/kg intravenous bolus of abciximab over
10 to 60 minutes before the procedure. Following the proce-
dure, abciximab is infused intravenously for 12 hours at a rate
of 10 µg/min. In place of abciximab, epifibatide may be
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and on aspirin indefinitely. Modifications of anticoagula-
tion regimens depend on the indication for stenting (for
acute stroke, some interventionists would not discontinue
heparin immediately following the procedure) and any
complication that may have resulted.
of stent-induced intimal hyperplasia are not known in the cerebral vasculature, and the treatment for in-stent steno-
sis may be problematic.

Stenting of intracranial vascular pathology may provide clinicians with therapeutic interventions where none existed previously. The subset of patients who are not surgical can-
nidates, for reasons such as severe cardiopulmonary prob-
lems, now has alternative nonsurgical options for intracranial
revascularization for severe stenoses or aneurysm occlusion.
Clearly, long-term, prospective data are needed to better understand and define the efficacy of intracranial stenting
for diverse cerebrovascular disease processes.

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1. Stenotic lesions that are less than 5 mm long with a concentric configuration are best suited for angioplasty.

   True or False?

2. During a stenting procedure for intracranial occlusive disease, a moderate improvement in luminal diameter is usually sufficient to adequately improve cerebral blood flow.

   True or False?

3. Oversizing stents to achieve minimal residual stenosis has no risk of reperfusion hemorrhage.

   True or False?

4. Stent-omitted coiling of intracranial aneurysm has the potential advantage of protecting parent vessels from coil herniation, especially when used with wide-neck aneurysms.

   True or False?

5. Stent placement for acute stroke has no role when thrombolytic therapy is used.

   True or False?

6. There is no evidence that stenting could help in the treatment of venous sinus thrombosis.

   True or False?

7. Ilb/IIa inhibitors seem to predispose to hemorrhage after reperfusion of cerebral regions in patients with chronic hypoperfusion.

   True or False?

8. There is no evidence from the cardiac literature that combination antiplatelet regimens reduce the rate of periprocedural complications and the need for repeat angioplasty.

   True or False?

9. Rapamycin (sirolimus) has been used in Rapamycin-coated stents to inhibit restenosis by altering the stability of microtubules and thus inhibiting cell replication.

   True or False?

10. The use of paclitaxel-coated stents in coronary stenting trials decreased the 6-month restenosis rates from 27% in the control group to 4% in the paclitaxel group.

    True or False?