In addition to extracranial embolic sources, ischemic stroke can also be caused by intracranial atherosclerotic stenosis with resultant chronic hypoperfusion, emboli, or total occlusion. The estimated risk of stroke in the setting of intracranial stenosis varies from approximately 7 to 40% per year with or without medical therapy.\(^2,6,9,22,27,29\)

Intracranial angioplasty can increase perfusion of the at-risk territory, decrease or eliminate ongoing or recurrent neurological symptoms, and potentially delay or prevent secondary occlusion and stroke. With current techniques and equipment, intracranial angioplasty can be performed safely. We present the results of 9 years of experience with intracranial angioplasty leading to the development of our current technique.

**Clinical Material and Methods**

A retrospective analysis was conducted of all patients who had undergone intracranial angioplasty performed by the authors between 1989 and 1998. Over this 9-year period, 70 elective intracranial angioplasties for primary intracranial atherosclerotic disease were performed using three different techniques (early, middle, and current periods). All patients had symptoms referable to the target lesion, and all were referred for treatment when angiographic studies revealed an intracranial stenosis as the source of their symptoms. All patients were referred by neurologists or neurosurgeons.

In addition to the number of cases and the distribution of lesions treated, the presence or absence of immediate complications, the therapeutic maneuvers required to treat the complications, the degree of residual stenoses, and the clinical outcomes were recorded for each of the three periods. The long-term restenosis rate was not the object of this study; it could only have been evaluated accurately by repeated angiography, and these data are incomplete.

Each case was assessed for complications that devel-
oped while the patient was in the hospital. Complications were defined as: 1) direct vascular damage related to angioplasty; 2) problems related to access, either puncture site or vascular route; 3) problems related to anticoagulation; 4) angiographically visible embolic sequelae; or 5) new neurological deficit. Direct angioplasty vascular damage (micro- or macrodissection) was defined as: 1) intimal flap visible on biplanar angiography; 2) vessel wall haziness; or 3) decreasing lumen postangioplasty thought to be related to thrombus/platelet aggregation secondary to intimal damage. Malignant thrombus/platelet aggregation was observed to progress to total occlusion. Procedural success was defined as dilatation of the lesion with resultant increase in luminal diameter without neurological complications. A suboptimal angiographic result was acceptable because of our current practice of undersizing the angioplasty balloon and not recrossing an angioplasty site during an elective procedure. Further dilation can be performed at the first follow-up evaluation if necessary. Therapeutic success was defined as decrease or elimination of ongoing neurological symptoms and/or lack of subsequent stroke (the latter is an ongoing evaluation).

The technique of intracranial angioplasty evolved during the study and can be divided into three periods during which different techniques and materials were used: early (1989–1992), middle (1992–1993), and current (1994–present). We performed all procedures either individually or in association with other physicians. All of the procedures were performed in conjunction with systemic heparinization.

**Early Period**

Initially, a balloon closely approximating, but invariably smaller than the diameter of the vessel was chosen. An early version of the balloon angioplasty catheter (STEALTH; Target Therapeutics, Fremont, CA) was used primarily. The balloon inflation rate was moderate (maximum inflation was achieved in 15–30 seconds). Recrossing the lesion was permitted if the result of the initial dilation was not believed to be adequate. If the initial appearance of the vessel after angioplasty was satisfactory, no further observation was performed. Eight patients were treated using this technique. The distribution of lesion locations for all procedures is given in Table 1.

**Middle Period**

The technique used during this period was somewhat different and was based on the prevailing theory of angioplasty at the time. The balloon size was chosen to be as close to the vessel diameter as possible; oversizing by up to 0.25 mm was allowed. Very rapid and brief dilation (as fast as possible) was performed. The STEALTH system was used for all 12 lesions treated during this period. Recrossing the lesion was permitted. After an educational case (Fig. 1), repeated angiography was performed every 10 minutes for at least 30 minutes to assess for thrombus formation.

**Current Period**

Since 1994, we have performed 50 elective angioplasties. The balloon is always undersized (generally by 0.2–0.7 mm). The earliest procedures in this period were performed using the STEALTH and FasSTEALTH catheters; since the introduction of the microangioplasty balloon catheter (Stratus; Medtronic, Inc./Microinterventional Systems, Minneapolis, MN; the latter company went out of business in April 1998), we have used this system almost exclusively. Since 1996, all patients have received the antiplatelet drug abciximab (ReoPro; Eli Lilly & Co., Indianapolis, IN) during the actual angioplasty and by continuous infusion for 12 hours thereafter (according to the protocol developed for coronary angioplasty).

### TABLE 1

*Anatomical distribution of intracranial stenoses in 70 patients treated with balloon angioplasty*

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>No. of Patients Treated/Period</th>
<th>Early</th>
<th>Middle</th>
<th>Current</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICA</td>
<td>2</td>
<td>4</td>
<td>17</td>
<td>23</td>
<td>32.9</td>
</tr>
<tr>
<td>MCA</td>
<td>3</td>
<td>2</td>
<td>15</td>
<td>20</td>
<td>28.6</td>
</tr>
<tr>
<td>Distal VA</td>
<td>2</td>
<td>2</td>
<td>11</td>
<td>15</td>
<td>21.4</td>
</tr>
<tr>
<td>VBJ</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1.4</td>
</tr>
<tr>
<td>BA</td>
<td>1</td>
<td>3</td>
<td>5</td>
<td>9</td>
<td>12.9</td>
</tr>
<tr>
<td>PCA</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>2.9</td>
</tr>
<tr>
<td>Total</td>
<td>8</td>
<td>12</td>
<td>50</td>
<td>70</td>
<td>100</td>
</tr>
</tbody>
</table>

* BA = basilar artery; ICA = internal carotid artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; VA = vertebral artery; VBJ = vertebrobasilar junction.
Fig. 1. Angiographic studies demonstrating postangioplasty malignant thrombosis. A and B: Posteroanterior (PA) (A) and lateral (B) left common carotid artery (CCA) angiograms obtained in a 51-year-old man who had symptoms typical of stenosis of the distal ICA or MCA. These included episodic weakness, clumsiness, and numbness of the hand with occasional speech difficulty. The angiogram reveals severe hemodynamic abnormality; note that the superficial temporal artery (arrows) reaches the crown of the head long before intracranial vessels are even perfusing their own territory. C: A magnified view of the focal stenosis after 250,000 U urokinase was infused over 1 hour. This study infusion was given to assess the possibility of thrombus superimposed on the plaque and reveals that the stenotic vessel diameter remains much smaller than 1 mm. D and E: Angioplasty was performed using a 2-mm STEALTH balloon. After the angioplasty was performed, the occluding wire was left in the vessel across the stenosis while the catheter was withdrawn slightly (note the markers on the balloon, short arrows). The angioplasty result appears to be satisfactory (long arrows). Note the excellent antegrade intracranial flow on the lateral view. F: Continued observation was maintained periodically until this image was obtained. The lateral angiogram (obtained 27 minutes after angioplasty and 22 minutes after the studies depicted in D and E) demonstrates that the ICA is now completely occluded (arrows). It was at this time that the patient began to experience some neurological difficulty, a point at which most patients would be in the recovery room. G: Four minutes after the study depicted in F, infusion of urokinase was begun, and after a bolus of approximately 20,000 U had been slowly administered the vessel was again patent. Abundant thrombus is still evident at the angioplasty site (arrow). H and I: In PA and lateral views obtained 1 hour and 25 minutes after the study depicted in G, and after a total of 610,000 U of urokinase had been infused, the vessel and angioplasty site are revealed to be cleared (arrows). Subsequent observation of the site revealed no further evidence of malignant thrombosis, and the patient was neurologically intact.
is continued for 1 hour. The platelet aggregation cascade, if present, usually stops in less than 30 minutes (see exception in Fig. 1) and during the second half hour the angioplasty site either stabilizes or begins to regain the appearance it had immediately after the angioplasty. If the postangioplasty appearance becomes alarming, urokinase can be infused to clear the lumen. Since the institution of abciximab therapy, the need for this maneuver has decreased: five (41.7%) of 12 in the middle period and one (14.3%) of seven in the current period required it before abciximab use, compared with one (2.3%) of 43 cases since abciximab use was begun.

Patients are started on a regimen of 250 mg ticlopidine taken orally twice daily for 1 month after the procedure. Aspirin therapy, 325 mg/day taken orally, is continued for the rest of the patient’s life.

In our current series, clinical and angiographic follow-up evaluations are conducted at 3 months, 6 months, and 1 year to check for restenosis.

Results

The results for all three treatment periods are summarized in Table 2.

Early Period

Of the eight patients treated during this period, seven experienced symptomatic relief (no further transient ischemic attacks [TIAs] or ischemic symptoms). One patient who was thought to have VB insufficiency did not improve after angioplasty of the distal vertebral artery (VA). Residual stenosis was present in all patients; this was 25 to 50% of the diameter in five cases (62.5%) and 50 to 66.7% in the remaining three (37.5%). Minimal dissection was identified on angiographic studies obtained immediately after angioplasty in four (50%) of the eight lesions, all without flow restriction or sequelae. No neurological complications occurred. Symptomatic restenosis occurred in one patient and was treated with repeated angioplasty in the middle period (this patient thus represents one procedure each performed during two periods).

Middle Period

Of the 12 patients treated, only one had a completely uneventful course. There were three intimal dissections (25%) that did not require treatment; these patients had good outcomes. Five dissections were thought to be associated with thrombus formation and required urokinase infusion (41.7%; total occlusion in two cases and partial occlusion in three). Four of these were successfully treated, with excellent results. In the fifth patient the artery remained occluded and this resulted in a stroke. One lesion (8.3%) demonstrated rebound stenosis, necessitating immediate repeated angioplasty, with a good result. Dissection with immediate vessel occlusion occurred in one case (8.3%) and occlusion secondary to attempted recrossing of the angioplasty site in another (this is the case in which urokinase was administered because thrombus was also thought to be present, but the vessel remained occluded); both were associated with poor outcomes (one stroke, one death). The lesion that occluded secondary to attempted recrossing of the fresh angioplasty site was a delayed (3 years) restenosis of a lesion treated during the early period. Of the lesions treated successfully in this middle period, none demonstrated a residual stenosis of greater than 50%. Ten (83.3%) of the 12 patients eventually achieved good outcomes.

Current Period

We performed 50 elective angioplasties in the current period. Of these, seven were performed before use of abciximab became routine, and 43 were performed with abciximab therapy. Angiographic evidence of dissection was present in seven (14%) of these 50 cases. Continued observation demonstrated that five of these were stable; only two required urokinase infusion (resulting in complete clearing of thrombus and no sequelae in both). Of the two lesions requiring urokinase infusion, one (14.3%) of seven was treated before the routine use of abciximab and one (2.3%) of 43 occurred after. There were no instances of abrupt vessel occlusion or stroke associated with intimal dissection.

In these 50 elective angioplasties the following complications were seen: one superficial temporal lobe hemorrhagic conversion (2%) of a prior subacute infarction occurred at 18 hours postprocedure (the patient recovered uneventfully); one vascular perforation with the occlusion wire (2%) that resulted in delayed intracranial hemorrhage and death; one small postoperative occipital intraparenchymal hematoma (2%; this patient had a preexisting field cut that was unchanged after the bleed); and two episodes of intraprocedural TIAs (4%), both of which resolved completely in less than 24 hours. The vessel perforation occurred prior to the use of the 0.010-in angioplasty system and before the routine use of abciximab.

Residual stenoses of greater than 50% were present in eight instances (16%); none was greater than 70% and the lumen was larger than 1 mm in diameter in every case. There were four instances (8%) of late restenosis di-
agnosed at the time of follow-up angiography (3–12 months); all were successfully treated with repeated angioplasty.

Of the 50 elective intracranial angioplasties, in 49 (98%) we achieved good angiographic and short-term clinical results (stable or improved neurological status and resolution of progressive or recurring symptoms).

Definitive long-term follow up of all patients has not been possible because of the nature of this retrospective evaluation, the several institutions involved in the performance of procedures, and the widespread geographic locations of the patients (from at least seven states). At least one follow-up angiographic examination has been obtained in 44 of the 50 patients treated in the current period. There was one stroke in a contralateral carotid artery (CA) territory. We are aware of no subsequent late-term strokes in the treated vascular territory in any patient who underwent angioplasty.

**Discussion**

Indications for any therapy are based not only on the risks of the disease process itself, but also on the safety and efficacy of the therapeutic treatment or procedure. Generic evaluation of oral medications may be possible, but evaluation of a procedure is dependent on patient selection and surgical personnel. There is no way to separate the actual intracranial angioplasty technique from the safety and efficacy of this therapeutic procedure. This then determines the risk/benefit ratio and the subsequent indications for treatment, regardless of the “natural history.”

**Goals of Therapy**

Intracranial angioplasty of atherosclerotic lesions is performed to prevent stroke secondary to hypoperfusion or subsequent occlusion. In addition, it can be performed for the relief of recurrent or ongoing neurological symptoms such as VB insufficiency (Figs. 1–3). Although the model of cervical CA endarterectomy for stroke prevention is similar because of the mechanical nature of that therapy, the actual procedure is different. Increased arterial diameter with subsequent increase in flow and possible remodeling of an atherothrombotic surface is the goal of any angioplasty procedure.

**Natural History of Intracranial Atherosclerotic Disease**

The incidence and implications of hemodynamically significant atherosclerotic intracranial vascular disease are underappreciated clinically and diagnostically. In one study, 22.6% of patients who underwent cerebral angiography were found to have intracranial stenoses. In another study, 8% of patients suffering from TIAs were found to have intracranial stenoses. According to the authors of the Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) Study, as many as 5 to 10% of all ischemic strokes are caused by intracranial large-vessel atherosclerotic disease.

Typical intracranial atherosclerotic disease presents in five primary locations: the internal carotid artery (ICA) siphon, the main trunk of the middle cerebral artery (MCA), the distal VA, the VBJ, and the midportion of the BA. The BA is reported to be the most common site, accounting for 8% of all brachiocephalic lesions. The natural history of intracranial stenoses is not as well known as that of extracranial CA stenoses. Intracranial disease often presents with a stroke rather than TIAs; patients with intracranial atherosclerotic stenoses presenting with TIAs have a high incidence of subsequent stroke in a short period of time (within months). In the WASID study, patients with symptoms of TIA or stroke in conjunction with 50 to 99% stenoses of major intracranial arteries were treated with aspirin or warfarin (adjusted to maintain prothrombin time at 1.2–1.5 × control). Of the 63 patients treated with aspirin, nine (14.3%) subsequently suffered strokes in the distribution of the stenosis, and six had strokes in another territory during the follow-up period (median 19.6 months), for a combined stroke rate of 23.8%. Of the 88 patients receiving warfarin (mean duration of follow-up 14.7 months), five had ischemic strokes in the same territory and one in another territory. As part of the WASID study, 68 patients with 50% or greater stenoses in the posterior fossa were followed for a mean of 13.8 months. Of these, 42 were treated with warfarin, whereas 26 received aspirin therapy, according to the physician’s choice. During that time, 15 patients had strokes, of which 10 were in the territory of the target vessel, for failure rates of six of 42 for warfarin and four of 26 for aspirin, which were statistically equivalent. The rates of stroke in the same territory were two (40%) of five for bilateral VA stenoses, two (8%) of 26 for unilateral VA stenosis, five (18%) of 28 for BA stenosis, and one (11%) of nine for posterior cerebral artery (PCA) or posterior inferior cerebellar artery (PICA) stenosis. In addition, neither warfarin nor aspirin stops the progression of atherosclerosis (Fig. 2).

In another study, the investigators found no significant difference in outcome between patients with symptomatic lesions and those with lesions that were asymptomatic at the time of diagnosis. In African American, Chinese, and Japanese patients, the incidence of intracranial disease is greater than that of extracranial disease, and thus represents a major source of ischemic strokes. Intracranial stenoses tend to affect younger patients more often than extracranial stenoses, possibly resulting in a higher cumulative risk of stroke.

Data concerning stenoses of the MCA indicate a stroke risk of at least 8% per year. Patients with MCA disease were younger, had fewer TIAs, and were more often African American and female than patients with extracranial ICA disease.

The natural history of intracranial ICA stenosis has received some attention. In a study of patients with intracranial ICA stenoses reported by Marzewski, et al., intracavernous lesions were most common, followed by petrous lesions and supraclinoid lesions. During the study’s follow up (average 3.9 years), 12.1% of patients had TIAs and 15.2% had a stroke, for an event rate of 27.3%. The rate of stroke in patients younger than 65 years of age who had intracranial ICA stenoses was 300 times the rate in a normal population.

Extracranial–intracranial bypass was originally proposed as a treatment for intracranial stenoses. In the report by the EC/IC Bypass Study Group, 1377 patients were evaluated, with an average follow-up period of 55 months. Of these patients, 714 were assigned to a group that received optimal medical therapy and 663 to one that...
Fig. 2. Angiographic studies obtained in a 53-year-old man who presented to the emergency room with acute discoordination, ataxia, dysarthria, and dizziness. Computerized tomography studies were unremarkable.

A: Admission angiogram (PA view of the left CCA injection) demonstrating abnormal flow not only to the ipsilateral PCA, but also to the BA through a very small posterior communicating artery (PCoA) (long thin arrow). Indeed, there is flow into both superior cerebellar arteries (small arrows) as well as inferiorly into the mid-BA (large arrow), a most abnormal distribution through such a small PCoA. This implies essentially no normal antegrade perfusion of the posterior fossa.

B: No right VA is present; PA view of the left VA injection reveals an apparent occlusion of the distal VA, just past the origin of the PICA (large arrow). There is reconstitution of the BA via anastomoses with the left anterior inferior cerebellar artery (AICA). Note filling of the opposite (right) AICA (small arrow). The patient was placed on intravenously administered heparin. No angioplasty was desired at this time.

C: Repeated angiographic study obtained after 1 week of intravenous heparin administration revealing that the left VA has opened enough that angioplasty was thought to be unnecessary (arrow). The size of the vessel is surprisingly good and the flow is excellent. Note the retrograde flow into the right VA to its PICA (confirming proximal occlusion of this vessel), and the absence of a right PCA (which filled via the right PCoA). Because of the apparent reversal of the pathological narrowing, the patient received warfarin anticoagulation therapy and was scheduled to be followed in clinic.

D: The patient presented on an emergency basis 3 months later with recurrent severe ataxia and dysarthria. Due to the changing nature of this lesion, an unknown amount of thrombus was suspected and the procedure was delayed for 5 days, during which time the patient was maintained on intravenously administered heparin.

E: Angiogram obtained 5 days after the study depicted in D. Due to the possibility that some of the continued narrowing was caused by additional thrombus, an infusion over approximately 25 minutes of 100,000 U urokinase was administered. This resulted in a smooth but very tight lumen. Note the continued absence of the left PCA because of hemodynamic insufficiency.

F: Left VA angiogram obtained after angioplasty revealing an excellent result with good hemodynamics and no evidence of vascular damage from angioplasty.

G: Angiogram obtained 1 hour postangioplasty, following our usual routine of continued observation of the angioplasty site, exhibiting some early narrowing and vessel wall irregularity (arrow), which was thought to be due to platelet aggregation. This was not believed to warrant any intervention, but rather continued observation. No further progression was seen and the procedure was terminated.

H: Follow-up angiographic study obtained 9 months after the procedure revealing the angioplasty site to be remodeled; it is widely patent with excellent flow. The patient has remained clinically stable for 5 years.
rate of fluid infusion and thus of the balloon inflation rate. A transvenous pacemaker was placed, but he achieved no relief from his symptoms. A: Cerebral angiogram revealing a threadlike remnant of the left VA at the atlantooccipital junction (arrow), a typical location for stenosis (as well as idiopathic dissections). The right VA was occluded. B: Follow-up left VA angiogram obtained after angioplasty of this lesion demonstrating an excellent angiographic result (long arrow) with resultant resolution of his episodic dizziness and ataxia. The angiogram demonstrates excellent posterior fossa perfusion, but extremely poor perfusion of the left occipital lobe (asterisk) beyond the threadlike origin of the left PCA. No true infarct was visible on computerized tomography scanning (repeat magnetic resonance imaging could not be performed because of the patient’s pacemaker). His vision was very poor even with his glasses (he could not read a clock from across the room), but his visual fields were full to confrontation. The decision was made to perform angioplasty to treat the PCA stenosis at a later date. C: Preangioplasty appearance of the left PCA origin stenosis (long white arrow). The normal portion of the vessel distal to the lesion measures only 1.6 mm, but was successfully treated with angioplasty. D and E: Postangioplasty appearance at 9-month follow up. The appearance of the origin of the PCA is good (white arrows, D). There is now bilaterally symmetrical perfusion of the occipital lobes (arrows, E). The day after the angioplasty of the origin of the left PCA, the patient commented that he could watch television for the first time in 3 years and he now reads without glasses.

received the same regimen with the addition of a superficial temporal artery–MCA branch bypass. Nonfatal and fatal stroke occurred more often and earlier in the patients who received the bypasses, and patients with severe MCA stenosis were among those with the worst outcomes.

Pathophysiological Rationale for the Current Angioplasty Technique

We believe that if a mechanism were to be invented to cause vascular damage and intimal dissection intentionally by utilizing catheter techniques, rapidly inflating a large noncompliant balloon inside an atherosclerotic lesion would be an ideal method to achieve this goal. We therefore diligently attempt to avoid this by two means: 1) extremely slow inflation (to allow the vessel to stretch rather than crack); and 2) undersizing the balloon. Whereas the luminal area is proportional to the square of the radius, flow is proportional to the fourth power of the radius. Thus, any small improvement is magnified.

It has been our observation that extremely slow (over minutes), gradual inflation of the balloon during angioplasty results in fewer large intimal tears, flaps, and dissections than the more commonly used method of “blow it up, let it down.” Allowing the vessel to stretch slowly (as opposed to tearing the vessel wall purposefully, as with the rapid inflation technique) seems reasonable. A screw-type inflation device is used for accurate control of the rate of fluid infusion and thus of the balloon inflation rate. Its secondary purpose is for regulation of angioplasty inflation pressure. Vascular dilation is dependent on the rate of balloon expansion, not on the rate of pressure increase.

A coronary angioplasty trial in which a similar slow inflation technique was used (one that was still far more rapid than ours) revealed that using this technique can decrease the incidence of major dissection to one third of that associated with the “standard” technique (3% compared with 9%).25 We believe that our technique results in even fewer significant dissections than this. In an intracranial setting, this difference can be extremely important. A major dissection can result in stroke, if not death, and can change the risk/benefit ratio to a negative value. A surgical rescue is not possible, nor can the lesion currently be repaired using a stent. Therefore, these tears should be avoided at all costs, even to the extent of accepting a less visually appealing angiographic result. These dissections can lead to platelet aggregation that can in turn lead to short-term occlusion or long-term restenosis.

The reasons that an angioplasty procedure may fail acutely are elastic recoil, intimal dissection with resultant flap, or platelet/fibrin/thrombus formation, as has been determined in the coronary arteries,10 and confirmed by the apparent responsiveness to platelet glycoprotein IIb/IIIa receptor inhibitors such as abciximab. Acute vessel occlusion caused by vasospasm and thrombosis of the damaged artery has been reported to complicate femoropopliteal angioplasty in up to 40% of cases.19 Late restenosis secondary to angioplasty is related to intimal hyperplasia (cellular proliferation) and vascular remodeling; it occurs in approximately 30% of coronary artery angioplasty
sites. The mechanisms for these complications are fairly well understood and require specific means of prevention.

Undamaged vascular endothelium has an anionic surface charge that, along with a constant release of endothelium-derived relaxing factor (a powerful antiplatelet agent), yields an antithrombotic environment. Any damage to this endovascular covering decreases the antithrombotic nature of the surface and exposes the subendothelial matrix to the circulating blood. This matrix is thrombogenic, and its exposure begins the thrombotic cascade. It has been shown in experimental angioplasty models that platelets are deposited rapidly at sites of intimal injury from angioplasty and that this deposition is nearly complete after 10 minutes. The extent of aggregation is determined by local hemodynamic forces that are directly related to flow velocity and indirectly related to the third power of the arterial diameter, as well as to the severity of the vessel wall injury. Intracranial vessels are relatively small and can be extremely thrombogenic. Thus, these angioplasty sites must be observed after the procedure to watch for this delayed malignant platelet cascade. We have observed progressive thrombosis to the point of occlusion more than 30 minutes postangioplasty (Fig. 1).

Intimal hyperplasia and late restenosis result from the interaction between blood components and the vessel wall. Damaged endothelium, activated platelets, and neutrophils at the angioplasty site generate reactive intermediary agents. It has been observed that decreasing intimal damage and reducing platelet aggregation also decrease late restenosis. Therefore, avoidance of platelet aggregation is beneficial both acutely and over the long term. In the coronary circulation, stents are used to treat vascular damage from angioplasty and improve immediate results but are known to have a restenosis rate of at least 20 to 40% in as few as 6 months; this is unacceptable in an intracranial location. In addition, reangioplasty of a stented lesion is difficult if not impossible, but reangioplasty of simple restenosis has been uniformly successful in our recent series.

At least a portion of our lack of procedural complications is due to the extremely gentle nature of the actual catheterization and angioplasty; this is in part possible because of the evolution of equipment. The balloons on double-lumen high-pressure coronary microangioplasty catheters, although potentially having a low profile, are currently too long and stiff for intracranial use. Almost all lesions that underwent dilation were less than 10 mm long (usually 2–4 mm), and thus very short balloons are adequate, indeed necessary. When inflated, longer balloons will actually straighten these intracranial vessels, resulting in possible damage to vital perforating vessels. Therefore, all lesions are treated with dedicated intracerebral single-lumen angioplasty catheters, most typically 2 mm by 10 mm in size. In addition, we prefer the softest system available, incorporating an 0.010-in occlusion guidewire. Improvements in these angioplasty catheters are continuing to be developed.

Avoidance of Complications and Modification of Risk/Benefit Ratio

In this series, complications were almost entirely caused by improper tools or technique. Improper technique can result in vessel rupture (which did not occur in our series), vessel perforation (one case, caused by the occlusion wire from the STEALTH system), and vascular damage/intimal dissection resulting in direct mechanical occlusion (one case), or extensive platelet/thrombus stimulation with subsequent partial or total occlusion or emboli (seven of 70 cases). Using the current technique for elective intracranial angioplasty, we have had good angiographic results and clinical outcomes in 49 (98%) of 50 patients. The one major complication in our series (vessel perforation by the occlusion wire) would probably not have occurred if we had used the Stratus 0.010-in occlusion wire system. Therefore, improvements in technique and equipment have rendered this procedure far safer than previously reported (major morbidity and mortality rates ranging from 11.1–40%).

We believe that when using optimal techniques and equipment, the major potential complication of intracranial angioplasty is vascular dissection that can lead to acute vessel occlusion. Our primary means of avoiding this is by gentle and extremely slow vascular stretching combined with undersizing the angioplasty balloon. The goal of therapy for an intracranial angioplasty is to increase the absolute flow through the vessel by increasing the lumen size to an acceptable cross-sectional area and, secondarily, possibly to remodel the lumen to be less thrombogenic.

Absolute evaluation of long-term restenosis was not the goal of this study and its occurrence is identified only by repeated angiography (now routinely performed). All patients in the current period who were found to have recurrent stenosis (lumen < 1 mm in diameter in four of 44; recurrent symptoms in one of 44) underwent uneventful repeated angioplasty with excellent results in all cases.

Delayed (semiacute, periprocedural) occlusion secondary to thrombosis at the angioplasty site is another potential major complication. There are three components in the effective management of this problem: 1) prevention, 2) detection, and 3) treatment. Prevention is achieved by technical proficiency on the surgeon’s part as well as adequate use of anticoagulant and antiplatelet agents. Detection is accomplished by patiently observing for a malignant platelet/thrombosis cascade. Delayed (semiacute) occlusion is treated with fibrinolytic agents.

An angioplasty site should not be recrossed after dilation. This maneuver has resulted in one stroke secondary to abrupt vessel occlusion by production of a large intimal flap (this procedure was performed in the middle period on a restenosis from the early period). Almost any angioplasty result will be better than the preangioplasty state; the lesion can be redilated at the first (3-month) follow-up review if necessary, after intimal healing from the original angioplasty has occurred.

Conclusions

Intracranial atherosclerotic vascular disease is a common condition that causes significant rates of morbidity and mortality in large numbers of people. Endovascular therapy can be performed safely by using optimal equipment and techniques. Further study is warranted.
Evolution of intracranial angioplasty for atherosclerosis

References


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