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Balloon Angioplasty for the Treatment of Vasospasm: Results of First 50 Cases

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Abstract

OBJECTIVE: To report the results of the first 50 consecutive patients with vasospasm secondary to subarachnoid hemorrhage treated with balloon angioplasty after failure of medical management.

METHODS: Retrospective uncontrolled study of 50 consecutive patients treated with balloon angioplasty between February 1988 and July 1992. Forty-six had objective clinical deterioration despite maximal medical therapy, whereas four were treated on the basis of rapidly accelerating transcranial Doppler velocities and decreased regional blood perfusion detected by technetium-99m-exametazime brain single photon emission computed tomography. All patients had evidence of marked vasospasm demonstrated by angiography. Thirty-two (64%) and 46 (92%) patients underwent angioplasty within 12 and 18 hours, respectively.

RESULTS: Of the patients with clinical evidence of vasospasm-induced ischemia, 28(61%) showed sustained neurological improvement within 72 hours of angioplasty. Three (6%) patients deteriorated within 72 hours after angioplasty, with two (4%) patients dying immediately after angioplasty as a result of vessel rupture and the other patient's Glasgow Coma Scale score decreasing by 2. Two additional patients in poor condition with Hunt and Hess Grade V at the time of angioplasty subsequently died during hospitalization. Two other patients died as a result of unclipped aneurysms that subsequently bled 4 and 12 days after angioplasty, respectively. The improvement demonstrated clinically, angiographically, and by transcranial Doppler after angioplasty was sustained, with only one patient requiring subsequent angioplasty of a previously dilated segment (total, 170 vessel segments dilated). Two patients developed vasospasm in previously undilated segments.

CONCLUSION: Timely balloon angioplasty can reverse delayed ischemic deficit caused by vasospasm in patients for whom medical therapy has failed.

Despite recent advances in the treatment of vasospasm with hypertensive, hypervolemic therapy and with calcium channel blockers and hemodilution, delayed ischemic and neurological deficits secondary to vasospasm continue to be a major cause of morbidity and mortality after subarachnoid hemorrhage(SAH) (13,19). Angiographic vasospasm has been reported to occur in 30 to 70% of patients after SAH, and up to 20 to 30% of patients who develop vasospasm suffer from ischemic neurological deficits (13,19,25).

Zubkov et al. (30), first reported the use of angioplasty for the treatment of vasospasm in 1984. Reports of other small series soon ensued (9,14,19,24). The present report describes the immediate (72 hr) and some limited long-term follow-up results of the first 50 consecutive patients in our institution who underwent angioplasty for the treatment of severe vasospasm. No patients were treated with intra-arterial papaverine infusion in this series.

PATIENTS AND METHODS

Between February 1988 and July 1992, 297 patients were admitted to the University of Washington affiliated hospitals with the diagnosis of SAH. A retrospective assessment of the first 50 consecutive patients who underwent balloon angioplasty for the treatment of vasospasm was conducted by review of the patient records and angiograms. The patients' ages ranged from 19 to 67 years, and there were 28 female and 22 male patients. SAH was confirmed in all patients by cranial computed tomography or lumbar puncture, and a cerebral aneurysm was confirmed by angiography in all except three cases.

Forty-six patients had cerebral berry aneurysms, with nine of these patients having multiple aneurysms. One patient had a mycotic aneurysm of the posterior cerebral artery. Two patients had traumatic SAH, and in one patient, no cause for the SAH was determined despite three angiograms. Twenty-seven had a Hunt and Hess Grade of I or II at admission, and 23 had a Hunt and Hess Grade of III through V (15).

Forty-five patients underwent obliteration of the offending aneurysm before undergoing angioplasty, with 44 undergoing surgical clipping and 1 undergoing endovascular balloon occlusion of a carotico-ophthalmic aneurysm. Of these patients, 26 (58%) had aneurysm occlusion performed within 48 hours of the ictus and 19 (42%) between Days 3 and 10.

Oral nimodipine was administered at admission, and hypervolemic treatment was routinely performed. Hypertensive therapy was commenced if there was laboratory, angiographic, or clinical suspicion of developing vasospasm. All patients were monitored in a neurological intensive care unit, with frequent assessment of vital signs and repeated neurological examination to detect early signs of symptomatic vasospasm. Daily transcranial Doppler (TCD) was performed to detect significant changes in hemodynamic parameters, which provided early warning of symptomatic vasospasm (23). Cranial computed tomography and nuclear medicine technetium-99m-exametazime (Tc99m-HMPAD, Ceretec, Amersham Corp., Arlington Heights, IL) brain single photon emission computed tomography (SPECT) were performed, as indicated, to help evaluate the patient's state (20).

Patients were treated with balloon angioplasty if they met the following criteria: 1) new onset of a neurological deficit not attributable to other causes (e.g., hematoma, swelling, or hydrocephalus); 2) no evidence of established cerebral infarction on the computed tomographic (CT) scans; 3) deficit persisting despite hypertensive, hypervolemic therapy; and 4) angiographic evidence of vasospasm in a distribution that could explain the deficit. In four patients, angioplasty was performed in the face of marked and rapidly rising TCD velocities in dominant hemisphere left middle cerebral arteries (MCAs) without marked clinical deterioration. Every effort was made to treat the patients as soon as possible, within 18 hours of symptom onset in most cases.

Angioplasty was performed via a transfemoral approach using a silicone microballoon (Target Therapeutics, Inc., Fremont, CA/Boston Scientific, Inc., Natick, MA) attached to a variable stiffness microcatheter (Target Therapeutics, Inc./Boston Scientific, Inc.) (Fig. 1). This balloon has a 3-mm diameter and a 12-mm length when fully inflated (at 0.5 atmosphere of pressure) with 0.15 ml of iodinated contrast. The details of performing balloon angioplasty have been previously described (10). Briefly, the angioplasty technique used consists of successive gradual mechanical dilations of the vessel lumen. The spastic vessel is first cautiously opened using 25% of the balloon volume for inflation and is then deflated. Angioplasty is gradually continued using 50 and then 75% of maximum balloon inflation, with intermittent deflations. Eventually, this angioplasty sequence is completed using 100% balloon inflation. This four-step sequence may need to be repeated many times, depending on the chronicity of vasospasm. Because of the concern regarding vessel over-dilation and rupture, no angioplasty was performed in vessels that were smaller than 3 mm in diameter before the onset of vasospasm.

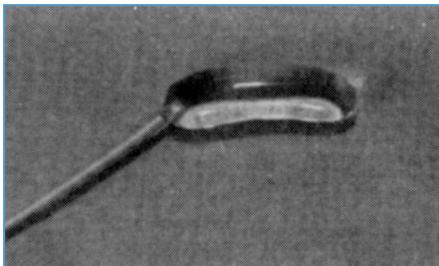


FIGURE 1. Low-pressure silicone angioplasty balloon (Target Therapeutics, Inc./Boston Scientific, Inc.).

All patients were monitored in a neurosurgical intensive care unit after the procedure. Neurological condition was evaluated using the Glasgow Coma Scale (GCS), and any further change in motor power or other neurological deficit was documented. Significant clinical improvement at 72 hours after angioplasty was defined as follows: 1) an increase of 2 or more in the GCS score; 2) an increase in motor power of two grades; 3) an increase in the GCS score of 1, accompanied by complete clearance of a previously clouded sensorium; 4) mild weakness (Grade IV/V) or focal neurological improvement, such as a homonymous hemianopsia, which completely cleared. Patients who had a decrease in the GCS score of 2 or more were classified as deteriorating, and all others were classified as unchanged.

RESULTS

Forty-seven (94%) patients were treated with angioplasty 4 to 14 days after SAH, with only three (6%) patients being treated more than 2 weeks after ictus. These three patients were transferred from other hospitals, where they had previously undergone craniotomy and clipping of their aneurysms. Thirty-two (64%) patients were treated within

12 hours of symptom onset, and 46 of the 50 (92%) patients were treated within 18 hours of symptom onset. However, some were treated as late as 6 days after symptom onset. Delayed angioplasty occurred most commonly in patients who had been transferred from other hospitals.

One hundred seventy vessel segments underwent balloon angioplasty in these 50 patients (Table 1). Each of 48 patients underwent one session of angioplasty, and each of 2 patients underwent two sessions, but the segments treated during subsequent angioplasty were, with the exception of a single segment in a single case, different from those treated initially.

Circulation	
Anterior	
Internal carotid artery	45
M1 segment of MCA	61
M2 segment of MCA	12
A1 segment of ACA	5
A2 segment of ACA	1
Posterior	
Basilar artery	16
Vertebral artery	14
P1 segment of PCA	14
PCoM	1
P2 segment of PCA	1

^a MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; PCoM, posterior communicating artery.

TABLE 1. Vessel Segments Undergoing Angioplasty (n = 170)^a

Twenty-eight (61%) of the 46 patients with clinical objective evidence of vasospasm-induced ischemic deficit improved objectively within 72 hours (Table 2 and Figs. 2-4). Fifteen patients did not improve in this 72-hour time frame after angioplasty, and three others deteriorated, two of whom died. The GCS scores at admission (Table 3) and immediately before angioplasty (Table 4) did not predict response to angioplasty, although there was a trend in patients in spasm with GCS scores less than 12 to be less likely to improve after angioplasty.

Results	No. of Patients
Improved objectively within 72 h (61%)	28
≥2 GCS scores	12
≥2 motor grades	7
4/5 improvement to normal	5
>1 GCS score and clearing of sensorium	3
Clearance of homonymous hemianopsia	1
Patients remained unchanged (33%)	15
Patients deteriorated within 72 h (6%)	3
Died	2
Decreased by 2 GCS scores	1

^a Four other patients had refractory vasospasm detected by TCD without obvious clinical deterioration who were treated with balloon angioplasty with marked TCD and angiographic evidence of improvement at 72 hours. GCS, Glasgow Coma Scale; TCD, transcranial Doppler.

TABLE 2. Results of Balloon Angioplasty in 46 Patients with Clinical Evidence of Vasospasm-induced Ischemic Deficit^a

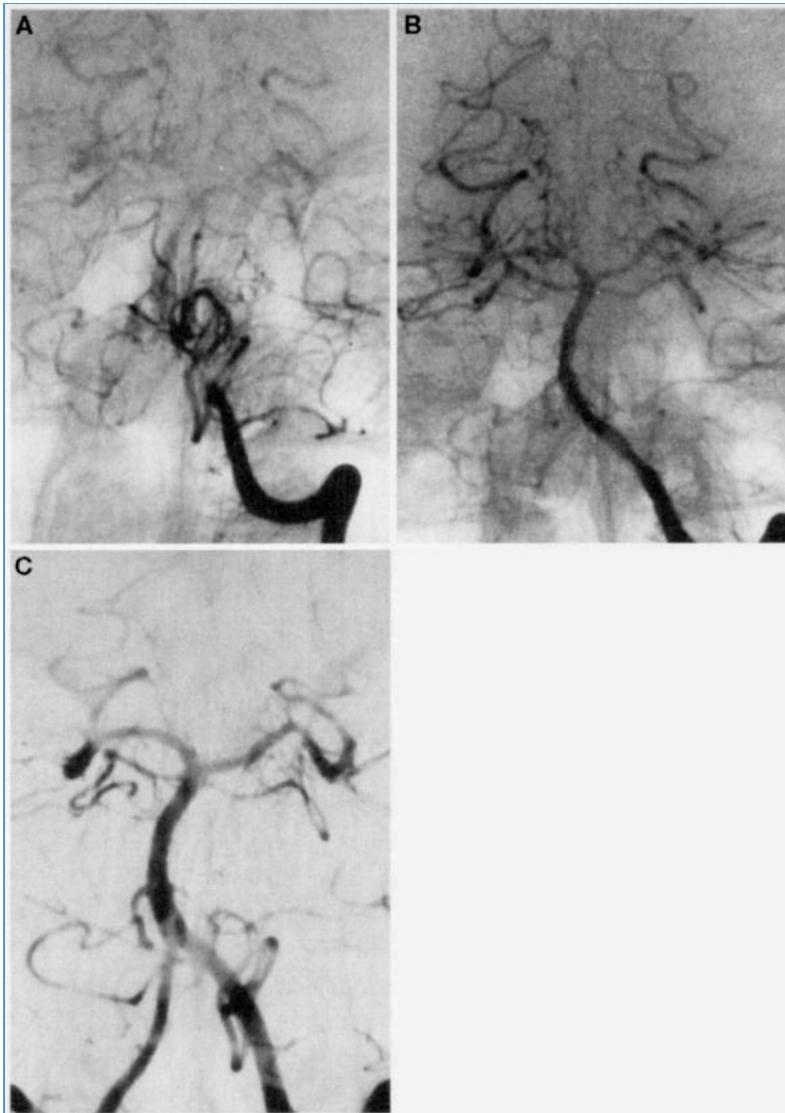


FIGURE 2. *A*, frontal view left vertebral arteriogram demonstrating severe vasospasm involving the distal vertebral and the proximal basilar arteries. The patient was comatose at this point. *B*, frontal view left vertebral arteriogram obtained immediately after angioplasty of the distal vertebral and basilar arteries. The vertebrobasilar system is normal in caliber. Angioplasty was performed at 10:00 PM. The next morning, at 7:00 AM, the patient was extubated. By 8:00 AM, the patient was neurologically intact. *C*, frontal view left vertebral arteriogram obtained 18 months after angioplasty. The vertebrobasilar system is normal, without evidence of long-term damage. The patient remains normal neurologically and is fully functional.

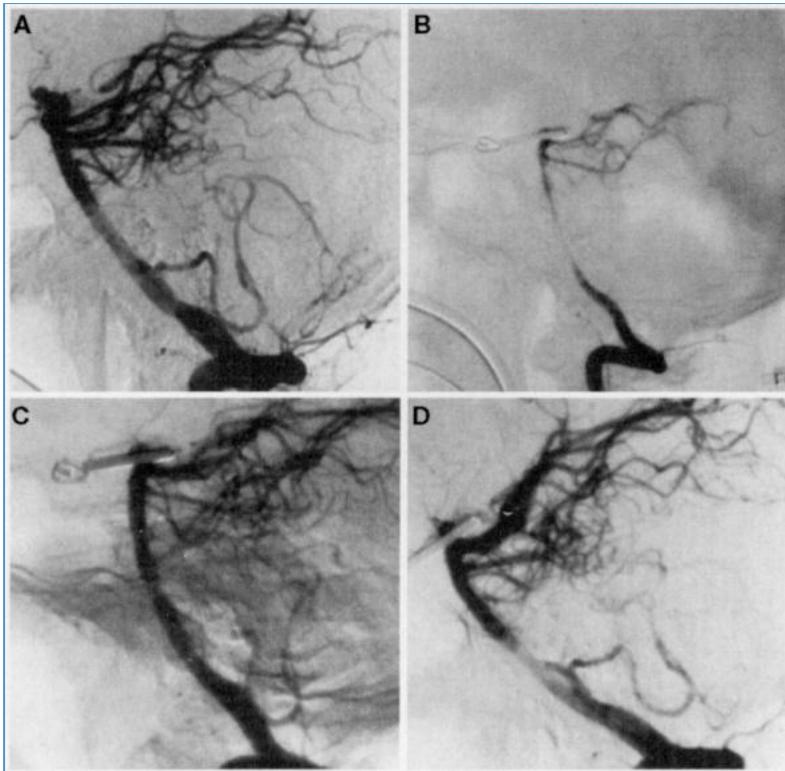


FIGURE 3. A, lateral view left vertebral arteriogram demonstrating a basilar tip aneurysm. The aneurysm was successfully clipped the day after the onset of SAH. B, 6 days after SAH, the patient had a respiratory arrest and slipped into a coma. Lateral view left vertebral arteriogram demonstrates severe basilar artery vasospasm. C, arteriogram obtained immediately after angioplasty shows the basilar artery to be normal in caliber. The patient's GCS score improved from 7 to 10 within 72 hours, and he gradually recovered during the next 2 weeks to be neurologically intact at discharge. D, lateral view left vertebral arteriogram obtained 18 months later, showing that the vertebrobasilar system is normal. There is no evidence of long-term damage. The patient remained normal neurologically and returned to work.

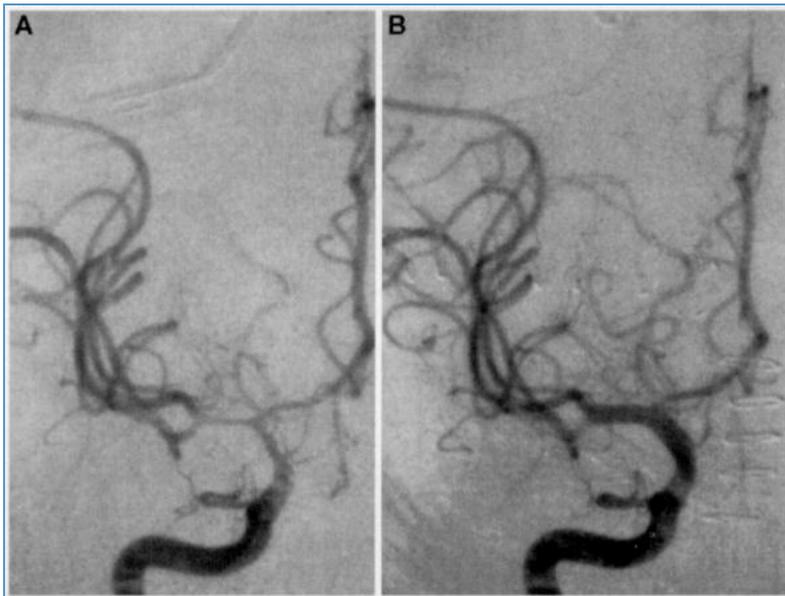


FIGURE 4. A, frontal view right internal carotid arteriogram showing moderate vasospasm of the supraclinoid carotid and severe vasospasm of the proximal MCA. The patient had left hemiparesis. B, angiogram obtained immediately after angioplasty, showing the supraclinoid carotid and proximal MCAs to be normal in size. The hemiparesis resolved within 24 hours.

No. of Patients	Better (n = 28)	Unchanged (n = 15)	Worse (n = 3)
15-13	15 (54%)	6 (40%)	1 (33%)
12-7	7 (25%)	5 (33%)	1 (33%)
<7	6 (21%)	4 (27%)	1 (33%)

TABLE 3. Glasgow Coma Scale Scores at Admission of 46 Patients with Clinical Evidence of Vasospasm Resulting in Balloon Angioplasty

No. of Patients	Better (n = 28)	Unchanged (n = 15)	Worse (n = 3)
15-13	9 (32%)	4 (27%)	1 (33%)
12-7	16 (57%)	6 (40%)	1 (33%)
<7	3 (11%)	5 (33%)	1 (33%)

TABLE 4. Glasgow Coma Scale Score of 46 Patients with Clinical Evidence of Vasospasm-induced Ischemic Deficits at the Time of Angioplasty

A total of six patients died, with two dying immediately after vessel rupture that resulted from angioplasty. One of these two patients had been transferred from another hospital with two aneurysms. The patient had SAH, and the anterior communicating artery aneurysm was partially clipped. Angiography had shown vasospasm and residual filling of the anterior communicating artery aneurysm. During angioplasty of the supraclinoid carotid, the patient became hypertensive and bradycardiac and subsequently died. No autopsy was performed. The supraclinoid carotid may have ruptured as a result of balloon dilation (presumed) or the partially clipped aneurysm may have subsequently bled during angioplasty. In the other patient who died immediately after angioplasty, the basilar artery was being dilated and the balloon extended into the P1 segment of the posterior cerebral artery. Because of the small size of the posterior cerebral artery at its origin, this vessel segment ruptured.

Two other patients died as a result of unclipped aneurysms that subsequently bled 4 and 12 days after angioplasty, respectively. For both of these patients, surgery was delayed to allow for clinical improvement. One of these two patients had a mycotic aneurysm of a cortical branch of the posterior cerebral artery. This patient underwent angioplasty of only the internal carotid artery and the MCA. No angioplasty was performed in the vertebrobasilar circulation from which the aneurysm arose. Two other patients were in poor condition (Hunt and Hess Grade V) at the time of angioplasty, continued to deteriorate, and subsequently died, despite angioplasty and maximal medical therapy.

Fifteen (32%) clinically symptomatic patients did not objectively improved within 72 hours of angioplasty, although angiographic dilation did occur. It is of note that four other patients did not have overt clinical evidence of deterioration but did have severe vasospasm demonstrated by TCD and angiography, with accompanying single photon emission computed tomography demonstrated abnormalities. These patients were thought to be at high risk for the development of symptomatic spasm and underwent angioplasty, achieving good angiographic results and resolution of the TCD-demonstrated changes. Three of these patients were neurologically intact, with GCS scores of 15 before balloon angioplasty, and one other patient (Fig. 5) was seriously ill (Hunt and Hess Grade IV).

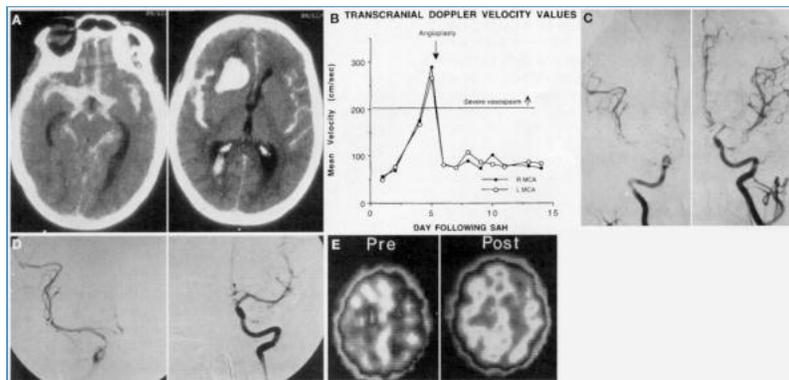


FIGURE 5. A, CT scan demonstrating severe SAH and intracerebral hemorrhage from a ruptured anterior communicating artery aneurysm. The patient was assigned Hunt and Hess Grade IV at admission. B, patient had rapidly increasing TCD velocities, which precipitated performing single photon emission computed tomography and angiography. C, patient underwent angiography, which demonstrated severe bilateral MCA vasospasm. D, angioplasty was performed, and the post-treatment angiogram demonstrates resolution of the MCA spasm. E, initial SPECT scan (left) demonstrating bilateral MCA perfusion deficits. The SPECT scan obtained after angioplasty (right), demonstrates

marked improvement. TCD velocities after the procedure revealed no recurrence of the vasospasm during the next week.^B

One patient developed a temporary deficit 6 weeks after angioplasty. This patient developed right hemiparesis 2 days after clipping of a left posterior communicating artery aneurysm. Angiography revealed that the patient had severe vasospasm and developed symptomatic spasm in the MCA, which underwent angioplasty that was performed using a high-pressure polyethylene balloon after dilation with the low-pressure silicone balloon failed. This high-pressure balloon ruptured in one small middle cerebral branch without adverse sequelae at the time of the immediate postangioplasty angiogram. The patient's new onset of hemiparesis from vasospasm, which prompted angioplasty, resolved within 2 days. The patient did, however, return 6 weeks later with mild right arm weakness, and subsequent angiography demonstrated that the involved MCA branch had occluded. During the next week, the patient regained his arm strength and became clinically neurologically intact. No other patients in this series demonstrated any permanent or temporary deficits during limited long-term follow-up (range, 26-61 mo; mean, 42 mo [22 patients whom we were able to contact]).

DISCUSSION

We report the effects of balloon angioplasty in 50 patients with severe vasospasm after SAH. Forty-six had delayed ischemic deficit resulting from vasospasm and 61% of these patients demonstrated objective neurological improvement 72 hours after angioplasty. Within this group, four patients without overt clinical deterioration but with severe vasospasm involving the dominant hemisphere documented by increasing TCD velocities and decreased perfusion on brain SPECT scans were treated, resulting in marked sustained decrease in TCD velocities at 72 hours.

The pathogenesis of vasospasm after SAH is not well understood (1,7,8,21,22,28). Structural changes in cerebral arteries after SAH may be related to collagen deposition in the arterial wall (27). Some additional evidence exists that cerebral vessels exposed to blood products are less distensible in vitro than are normal vessels (5). Prolonged narrowing may result in decreased elasticity, resulting in persistence of the stenosis and producing a less distensible vessel. The effect of mechanically dilating arteries in spasm has not been well studied. Angioplasty may be effective by disrupting the extracellular matrix, which maintains the narrowed state (6,11,29).

Angioplasty is not without risk. Vessel rupture can occur, and, therefore, angioplasty should not be performed distal to the genu of the MCA, beyond the A1 segment of the anterior cerebral artery, or beyond the P1 segment of the posterior cerebral artery. Once the balloon passes distal to these segments, the balloon diameter exceeds the diameter of the vessel. Therefore, we advocate primarily dilating the supraclinoid carotid and the proximal MCAs in addition to the distal vertebral and basilar arteries. Only on rare occasions is it necessary to attempt to dilate the proximal portion of the anterior and posterior cerebral arteries. In addition, the A1 segment can be difficult to safely access with an angioplasty balloon. Papaverine infusion has been reported to be useful in this situation (16,17).

It has been our experience that progressive, successive dilation of vasospasm is safer than the standard approach typically used for atherosclerotic disease, for which immediate full balloon inflation is the standard. Although no scientific studies have been performed to determine whether one long inflation is better than multiple small inflations in mechanically dilating spastic cerebral vessels, our rationale for the use of cautious and progressive balloon inflations is based on safety and not necessarily on effectiveness in achieving luminal dilation. Gentle and graded balloon inflations allow the interventionalist to develop a subjective feel for a spastic vessel, which likely decreases the risk of catastrophic vessel rupture or occlusion.

In our experience, the longer the vessel has been in spasm, the more difficult it is to perform angioplasty. If the onset of vasospasm is recent, then balloon dilation is relatively simple; the vessel usually dilates after the third or fourth balloon inflation. In contrast, if vasospasm has been present for a number of days, the spastic vessel is more fibrotic and difficult to perform angioplasty on. If the patient has been in vasospasm for some time, which is usually the case, then many more balloon inflations are necessary. Furthermore, the neurological deficit will likely have been present for a few days and is less likely to recover, even if angioplasty is technically successful. Therefore, our approach has been aggressive angioplasty in any patient with vasospasm for whom medical management has failed, with a goal to treat within 12 hours of symptom onset.

Although many patients had TCD or angiographic evidence of vasospasm, generally, we treated only patients for whom medical therapy had failed. However, in four cases with refractory vasospasm, angioplasty was performed in vessel segments demonstrating severe angiographic spasm with antecedent TCD evidence of markedly increased velocities (>200 cm/s) and a dramatic rate of rise of TCD velocity (>50 cm/s/d), both of which are markers of severe vasospasm (12,25). These are exceptional instances in which we consider that the likelihood of ischemic insult is high enough to warrant prophylactic balloon angioplasty.

The effect of balloon angioplasty seems to be sustained. Only two patients had subsequent symptomatic vasospasm requiring balloon angioplasty, but the distribution of the spasm was different, involving new segments. Only one segment required subsequent redilatation. Although our long-term follow-up results are limited and suffer from probable selection bias, there have been no new reported neurological deficits related to angioplasty of those discharged.

CONCLUSION

Clinical benefits have been observed using calcium channel blockers and hypertensive and hypervolemic therapy for the treatment of delayed ischemia secondary to vasospasm. Despite this, some patients continue to deteriorate (2-4,18,26). In patients with vasospasm who clinically deteriorate despite maximal medical therapy, the judicious and aggressive use of timely (within 12-18 h of symptom onset) percutaneous balloon angioplasty in experienced hands can improve short-term outcomes. Patients in comas with widespread vasospasm may also benefit from dilation of anterior and posterior circulations. The effects of angioplasty seem to be sustained; however, more complete clinical and pathological long-term follow-up is necessary. Although the results of our series are encouraging, randomized controlled trials and cooperative investigations are warranted to determine the true efficacy of angioplasty in the treatment of vasospasm.

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COMMENTS

Eskridge et al. describe their experience with balloon angioplasty for the treatment of medically intractable vasospasm. The senior investigator (JME) in this group is an extremely experienced interventional neuroradiologist who reports honestly and frankly his experience with balloon angioplasty. I think it is important to note that this procedure is not without risk and that vessel rupture can occur. We can all learn from the senior investigator's initial experience about the limitations of angioplasty. We have learned from his efforts that it is not wise to perform angioplasty beyond first-order vessels (i.e., A-1, P-1, and M-1). Angioplasty of unclipped aneurysms should be performed cautiously or, perhaps, not at all. Furthermore, we can learn from their aggressive approach in terms of prophylactic angioplasty with markedly elevated transcranial Doppler velocities.

I think our experience has benefited markedly from the reports of early investigators in not having to make the same mistakes. What we have learned, however, is that, as with the stroke model, the time to intervention should be shortened if at all possible. Our experience with 80 patients has indicated that if patients can undergo angioplasty within a 2-hour period after medical therapy has failed, there is 90% angiographically demonstrated improvement with 70% sustained clinical improvement. After 2 hours and up to 18 hours, we still had an 88% angiographic improvement, but only 40% sustained clinical improvement, perhaps indicating that end organ failure had already occurred and that despite a markedly improved angiogram, cerebral infarction had already occurred. With the advent of angioplasty, we now have an additional tool to treat one of the major causes of morbidity in this patient population.

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Eskridge et al. lead in the development of balloon angioplasty for the treatment of cerebral vasospasm. In this article, they report their experience with a consecutive series of 50 patients who were chosen for balloon angioplasty after ruptured intracranial aneurysms. The mortality rate of only 4% indicates that in the hands of these skilled clinicians, balloon angioplasty can be performed on a very sick population of patients with appropriate risk. In this series, 60% of the patients showed clinical improvement after being subjected to the angioplasty procedure. These results are encouraging and indicate that balloon angioplasty may be helpful in the management of cerebral vasospasm.

At my institution, our experience with balloon angioplasty has not been as positive as in this report. I am sure that the reason behind this discrepancy is that we have selected only gravely ill patients who have continued to rapidly deteriorate despite maximal hypertensive hypervolemic therapy. Although this point is not completely clear in the article, it seems that the authors have a very low threshold for instituting balloon angioplasty treatment in patients who begin to develop ischemic symptoms despite medical therapy. A positive benefit of the procedure will most likely be observed with early intervention. However, my own experience with 121 patients who have shown signs of cerebral ischemia after aneurysmal subarachnoid hemorrhage indicates that only 25% of these patients will either die or be left with a significant disability related to cerebral ischemia when maximal triple-H therapy is used. Long-term results were not available from the series presented by Eskridge et al., so that direct comparison is difficult. Although balloon angioplasty may have been of benefit to some of the patients in this series, it is likely that

a large number of the 60% of patients who achieved improvement in their clinical conditions within 72 hours after the procedure might also have improved with medical therapy alone.

Despite the favorable results of this study, the question still remains regarding when the risk of balloon angioplasty are worth taking in a patient who is in the early stages of delayed cerebral ischemia. The authors even include four patients in their series who were not symptomatic, and it is difficult to justify balloon angioplasty in asymptomatic patients with our present understanding of the procedure.

The experience reported in this article now lays the foundation for a more convincing prospective trial. It may be that a small number of institutions that are skilled in the management of patients with aneurysmal subarachnoid hemorrhage and balloon angioplasty procedures should randomize symptomatic patients to either balloon angioplasty or medical therapy alone. Such an approach may be the only way for us to learn which groups of patients will truly benefit from this promising therapeutic intervention.

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This report by Eskridge et al. describes their experience with balloon angioplasty for vasospasm associated with subarachnoid hemorrhage. The extent of their experience is unequalled, and therefore this is a valuable contribution to the literature. We think that some caution should be exercised before embracing this therapy. Of the patients in this series, nearly one-third were in good clinical grade (Glasgow Coma Scale scores 13-15) before undergoing angioplasty and four were asymptomatic. Asymptomatic patients were treated on the basis of transcranial Doppler recordings and single photon emission computed tomographic results; we do not think these parameters are necessarily sufficient to make a diagnosis of vasospasm that requires treatment. Angioplasty carried a 4% risk of immediate death, and the overall results presented in [Table 2](#) of the article are similar to the natural history of symptomatic vasospasm (1). One must ask whether it is justified to expose the patients with good grades and the asymptomatic patients to the risks of angioplasty. This can be determined by a prospective, randomized, controlled trial demonstrating whether angioplasty improves outcomes in this group of patients. It is not possible to make that conclusion from the retrospective uncontrolled series presented here. Nevertheless, angioplasty clearly has a dramatic dilating effect on vessel diameter, and some patients clinically improved in a very rapid fashion. It is therefore reasonable to assume that angioplasty does favorably influence outcome in some patients.

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1. Weir B: *Aneurysms Affecting the Nervous System*. Baltimore, Williams & Wilkins, 1987. [\[Context Link\]](#)

Eskridge et al. report 50 consecutive patients with angiographic vasospasm treated with balloon angioplasty. Sixty-one percent of the patients with delayed ischemic deficits and angiographically demonstrated related vasospasm improved significantly within 72 hours of angioplasty. Poor results were associated with sicker patients (Glasgow Coma Scale scores <12) and delayed treatment. Angioplasty resulted in fatal arterial rupture in two cases and significant neurological deterioration in one, for a combined major morbidity and mortality rate of 6%. Four patients were treated on the basis of transcranial Doppler and angiographic evidence of vasospasm, without any evidence of clinical deterioration. The authors recommend performing angioplasty on all angiographically narrowed vessels when treatment is undertaken.

Several points in this important article deserve emphasis. The four-point treatment algorithm outlined in this article is excellent. The key is early intervention when maximum medical therapy fails.

Another essential point made by the authors is the need to avoid ballooning distal vessels for fear of rupture. It is important to remember that A-1, P-1, and even M-1 vessel segments may be smaller than 3 mm (i.e., less than the diameter of the inflated balloon). Therefore, we rarely fully inflate the angioplasty balloon. The 3-mm silicone balloon (Target Therapeutics/Boston Scientific) used by the authors is a compliant balloon and may result in overdistention of normal segments adjacent to spastic vessels, which is another reason for extreme care and

underinflation of this type of angioplasty device.

Clouston et al. (1) suggest that intra-arterial papaverine, when used appropriately, can be a very effective adjunct to or even a substitute for angioplasty. This is particularly pertinent in view of the two fatal arterial ruptures caused by angioplasty in this series. Our experience with papaverine alone has not been very rewarding. Angiographic improvement is usually observed but often is transient and not usually associated with sustained clinical improvement. We, therefore, proceed directly to angioplasty in most cases. The risk of vessel rupture can be lessened by a procedure we learned from Alex Berenstein (personal communication). A small hole is made in the distal end of the balloon with a 30-gauge needle to create a leaky balloon that can auto-decompress inside the artery during angioplasty, presumably decreasing the risk of rupture.

In view of the potential for morbidity with angioplasty (i.e., arterial rupture), the authors' recommendation for angioplasty in the face of angiographic, TCD, and single photon emission computed tomographic evidence of vasospasm without clinical deficit is questionable. The authors mention two patients with unclipped aneurysms that subsequently bled after angioplasty. Another important caveat is the need to protect ruptured aneurysms before treating vasospasm, whenever possible. Guglielmi detachable coils (Target Therapeutics/Boston Scientific) are now available in most major centers where angioplasty is performed and should be considered before performing angioplasty, especially if surgery is not a viable option.

Angioplasty for vasospasm may be lifesaving if performed early after medical therapy has failed. Angioplasty may also be dangerous and must be performed with extreme care.

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1. Clouston JE, Numaguchi Y, Zoarski GH, Aldrich EF, Simard JM, Zitnay KM: Intraarterial papaverine infusion for cerebral vasospasm after subarachnoid hemorrhage. *AJNR Am J Neuroradiol* 16:27-38, 1995. [Serials Solutions](#) | [Bibliographic Links](#) | [Context Link](#)

Key words: Aneurysm; Angioplasty; Subarachnoid hemorrhage; Vasospasm

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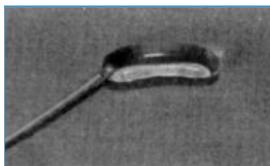


Figure 1

Circulation	
Anterior	
Internal carotid artery	45
M1 segment of MCA	61
M2 segment of MCA	12
A1 segment of ACA	5
A2 segment of ACA	1
Posterior	
Basilar artery	16
Vertebral artery	14
P1 segment of PCA	14
PCoM _A	1
P2 segment of PCA	1

* MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; PCoM_A, posterior communicating artery.

Table 1

Results	No. of Patients
Improved objectively within 72 h (61%)	28
≥2 GCS scores	12
≥2 motor grades	7
4/5 improvement to normal	5
≥1 GCS score and clearing of sensorium	3
Clearance of homonymous hemianopsia	1
Patients remained unchanged (13%)	15
Patients deteriorated within 72 h (6%)	3
Died	2
Decreased by 2 GCS scores	1

* Four other patients had refractory vasospasm detected by TCD without obvious clinical deterioration who were treated with balloon angioplasty with marked TCD and angiographic evidence of improvement at 72 hours. GCS, Glasgow Coma Scale; TCD, transcranial Doppler.

Table 2

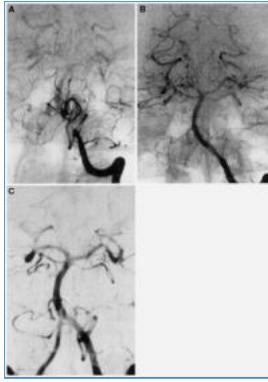


Figure 2

No. of Patients	Better (n = 28)	Unchanged (n = 15)	Worse (n = 3)
15-11	15 (54%)	6 (40%)	1 (3%)
12-7	7 (25%)	5 (33%)	1 (3%)
≤7	6 (21%)	4 (27%)	1 (3%)

Table 3

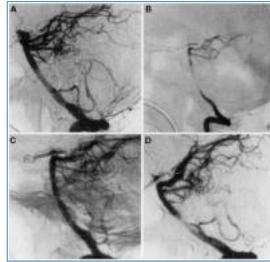


Figure 3

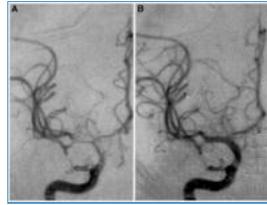


Figure 4

No. of Patients	Better (n = 28)	Unchanged (n = 15)	Worse (n = 3)
15-11	9 (32%)	4 (27%)	1 (3%)
12-7	16 (57%)	6 (40%)	1 (3%)
≤7	3 (11%)	5 (33%)	1 (3%)

Table 4

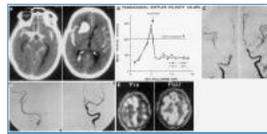


Figure 5

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