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Angioplasty for the treatment of symptomatic vasospasm following subarachnoid hemorrhage

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✓ Angioplasty of narrowed cerebral arteries was performed in 10 patients who became symptomatic from vasospasm following subarachnoid hemorrhage. This procedure was accomplished with a microballoon catheter via percutaneous transfemoral insertion. Patients were selected for treatment if they had delayed neurological deficits due to vasospasm which were not responsive to hypervolemic hypertensive therapy. Eight patients (80%) showed sustained improvement in neurological function following the procedure. In two patients transcranial Doppler ultrasound recordings were obtained which revealed decreased mean blood flow velocities following angioplasty. Two patients died, one from an aneurysmal rebleed, and one secondary to diffuse vasospasm. There was one case of delayed stroke 6 weeks following the procedure. The overall results of this series indicate that in selected cases percutaneous balloon angioplasty can offer marked improvement to patients with ischemic deficits due to vasospasm following subarachnoid hemorrhage.

KEY WORDS • **vasospasm** • **angioplasty** • **subarachnoid hemorrhage** • **aneurysm rupture** • **interventional neuroradiology**

DELAYED ischemic deficit (DID) from vasospasm is a frequent complication following subarachnoid hemorrhage (SAH).[13,18] Angiographic vasospasm has been reported to occur in 30% to 70% of patients between 4 and 12 days following SAH.[13] Delayed ischemic neurological deficits caused by vasospasm may develop in 20% to 30% of patients.[13] Numerous therapies have been proposed for this condition and these have been extensively reviewed by Wilkins.[31] In recent years hypervolemic hypertensive therapy has gained wide acceptance, and calcium channel blockers have been reported to improve outcome in patients with vasospasm.[4,5,24,25] Despite reported success with these treatments, vasospasm continues to be a major cause of morbidity and mortality following SAH.

The use of a balloon catheter to dilate cerebral arteries narrowed by vasospasm was first reported by Zubkov, *et al.*,[32] in 1984. The present report describes the use of this technique in 10 patients to treat DID's due to SAH which were persistent despite hypervolemic hypertensive treatment. The clinical results following the procedure, as well as changes in blood flow velocities identified by transcranial Doppler ultrasound (TCD) are reported.

Clinical Material and Methods

During a 10-month period between February, 1988, and December, 1988, 10 patients admitted to the University of Washington Affiliated Hospitals underwent percutaneous balloon angioplasty for vasospasm. Table 1 lists the clinical aspects of the patient group. There were five men and five women, with ages ranging between 38 and 61 years. All patients had SAH confirmed by lumbar puncture or computerized tomography (CT), and the presence of a cerebral aneurysm was confirmed by angiography in all but one. In the remaining patient no aneurysm was found despite repeated angiography. The distribution of aneurysms was as follows: four arose from the anterior communicating artery, three from the internal carotid artery, one from the middle cerebral artery, and one from the basilar tip. Angiography was performed on all patients prior to surgery and was repeated prior to angioplasty. Eight patients underwent surgery for obliteration of their ruptured aneurysm before angioplasty was performed, and the ruptured aneurysm was successfully clipped in seven of these patients. The other patient had an unsuccessful attempt at clipping and was awaiting balloon occlusion. One patient underwent successful intra-ar-

TABLE 1

*Clinical characteristics and course of 10 patients who underwent angioplasty**

Case No.	Age (yrs), Sex	Aneurysm Location	Admission Grade†	SAH to Surgery (days)	SAH to Angioplasty (days)	Neurological Deficit	Angioplasty		Outcome‡
							Vessels Treated	Results	
1	61, F	rt CA, ophthalmic	I	3	5	decreased LOC	rt ICA	improved transiently	dead
2	54, M	lt PCoA	I	3	4	decreased LOC, rt hemiparesis	lt MCA, rt MCA, basilar, lt & rt PCA	improved	good recovery
3	56, M	ACoA	III	0	7	decreased LOC, lt hemiplegia	lt ICA, lt MCA, lt ACA	not improved	dead
4	48, M	basilar tip	II	2	6	decreased LOC	lt vertebral, basilar, lt PCA, rt ICA	improved	good recovery
5	38, M	ACoA	I	8	9	lt hemiplegia, confusion	rt ICA, rt MCA	improved	good recovery
6	56, F	lt MCA	V	1	6	decreased LOC	lt ICA, lt MCA	improved	good recovery
7	41, F	ACoA	II	2	5	decreased LOC, lt hemiplegia	lt & rt ICA, lt vertebral, basilar, lt & rt MCA, lt & rt PCA	improved	moderate disability
8	40, F	no aneurysm	II	—	10	decreased LOC, aphasia, rt hemiparesis	lt ICA	improved	good recovery
9	52, F	lt CA, ophthalmic	II	2 (balloon occlusion)	8	decreased LOC	lt vertebral, lt ICA, rt PCA	improved	good recovery
10	41, M	ACoA	II	5	7	decreased LOC, rt hemiparesis	lt ICA, lt vertebral, lt PCA, basilar	improved	moderate disability

* CA = carotid artery; PCoA = posterior communicating artery; ACoA = anterior communicating artery; MCA = middle cerebral artery; SAH = subarachnoid hemorrhage; LOC = level of consciousness; ICA = internal carotid artery; PCA = posterior cerebral artery; ACA = anterior cerebral artery.

† Hunt and Hess classification.[15]

‡ Glasgow outcome score according to Jennett and Bond.[16]

terial balloon obliteration of her aneurysm before the onset of vasospasm and angioplasty.

Patients underwent balloon angioplasty if they met the following criteria: 1) there was new onset of a neurological deficit after SAH not attributable to other causes (such as hematoma, hydrocephalus, or swelling); 2) no evidence of infarction in a major vascular distribution could be seen on CT scans; 3) the neurological deficit was not reversed by institution of hypervolemia or hypertension; and 4) vasospasm was seen angiographically in a location that could be responsible for the ischemic deficit. In eight cases angioplasty was performed within 12 hours of the onset of neurological deterioration. In one case 48 hours elapsed and in another 72 hours elapsed between neurological deterioration and angioplasty.

Angioplasty was carried out with a silicone microballoon attached to a microcatheter of variable stiffness* (Fig. 1). The silicone balloon was developed for this purpose by Grant Hieshima, M.D., Bill Dormandy, and Julie Bell (G. Hieshima, *et al.*, personal communication, 1988). The balloon measures 0.85 mm in diameter uninflated, but expands to 3 mm in diameter and 12 mm in length when inflated with 0.15 cc of iodinated contrast material (Fig. 1). All procedures were performed via a transfemoral approach under full heparinization. The angioplasty procedures were performed under general anesthesia in three patients and neuroleptic analgesia in seven patients. Delayed follow-up arteriograms were obtained in three patients and serial TCD velocity measurements were obtained in two other patients before and after angioplasty.†

All patients were maintained in an intensive care unit with frequent monitoring of vital signs and neurological examinations after completion of the procedure. Neurological condition was assessed by both the Glasgow Coma Scale (GCS) and Hunt and Hess[15] grading. Patients were considered significantly improved following the procedure if they improved at least two GCS points or showed improvement of two grades on motor testing within 48 hours of the procedure.

Results

Eight of the 10 patients showed sustained improvement. Table 1 illustrates the results and Glasgow Outcome Scale[16] score at 1 month after angioplasty. One patient (Case 1) transiently improved, but then deteriorated, rebled from the unprotected aneurysm, and eventually died. There was no improvement in Case 3 following the procedure. This patient had a preexisting

* Silicone microballoon obtained from Interventional Therapeutics Corp., South San Francisco, California; microcatheter obtained from Target Therapeutics, San Jose, California.

† Transpect ultrasound system manufactured by Meda Sonics, Mountain View, California.

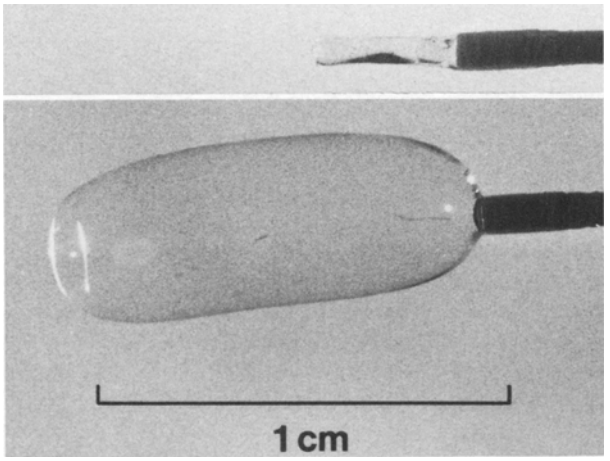

FIG. 1. Photograph of the silicone microballoon used for angioplasty. The soft silicone elastomer shell results in a low-pressure balloon that conforms to the shape of the parent artery during dilatation.

right carotid artery occlusion and this prevented angioplasty of the right anterior circulation. Four patients showed improvement in neurological function within minutes to hours following the procedure and the remaining patients who improved did so more gradually. Figure 2 illustrates the effect of angioplasty on the neurological condition of each patient as assessed by the GCS and the Hunt and Hess criteria. The two scales were used in combination because some patients demonstrated focal motor deficits with little change in consciousness while other patients manifested only decreases in consciousness as a consequence of vasospasm. The GCS records best motor response which could be unaffected by hemiplegia. The Hunt and Hess grade changes with focal motor deficits but is less sensitive than the GCS to the level of consciousness.

Selected Case Reports

Case 4. This 48-year-old man suffered an SAH from a basilar tip aneurysm (Fig. 3 *upper left*). Upon admission he was neurologically intact with a stiff neck (Hunt and Hess Grade II). Surgery was performed on the 2nd day following SAH and the aneurysm was successfully clipped. The patient was unchanged neurologically following surgery, and postoperative angiography demonstrated that the aneurysm was well clipped and there was no evidence of vasospasm. Five days after SAH the patient aspirated and subsequently developed pneumonia; on the next day he became unresponsive and suffered a respiratory arrest. He was immediately intubated and supportive measures were instituted. Despite hypervolemic therapy and dopamine there was no improvement of neurological function. Five hours after deterioration, the patient underwent angiography which demonstrated severe vasospasm in both distal vertebral arteries and the entire basilar artery (Fig. 3 *upper right*).

Angioplasty was immediately performed on the distal left vertebral artery, the basilar artery, the left internal

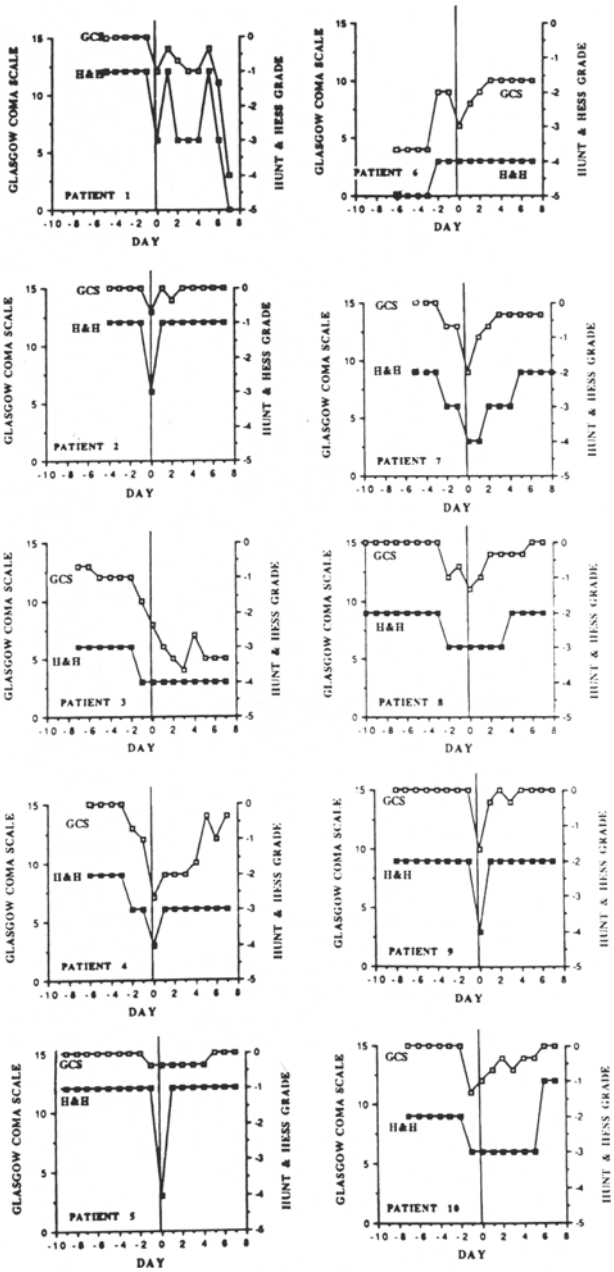

FIG. 2. Graphs illustrating the clinical condition of each patient assessed by Glasgow Coma Scale (GCS) and Hunt and Hess (H & H) grade in relation to the day of angioplasty (Day 0). The first entry on each graph represents the day of hemorrhage.

carotid artery, and the left posterior cerebral artery. Repeat angiography demonstrated marked improvement in the luminal caliber of the vertebral basilar system as well as the left posterior cerebral artery (Fig. 3 *lower left*). By the next morning the patient was following commands, but remained intubated due to the pneumonia. By the 4th day after angioplasty he was extubated and was normal neurologically except for

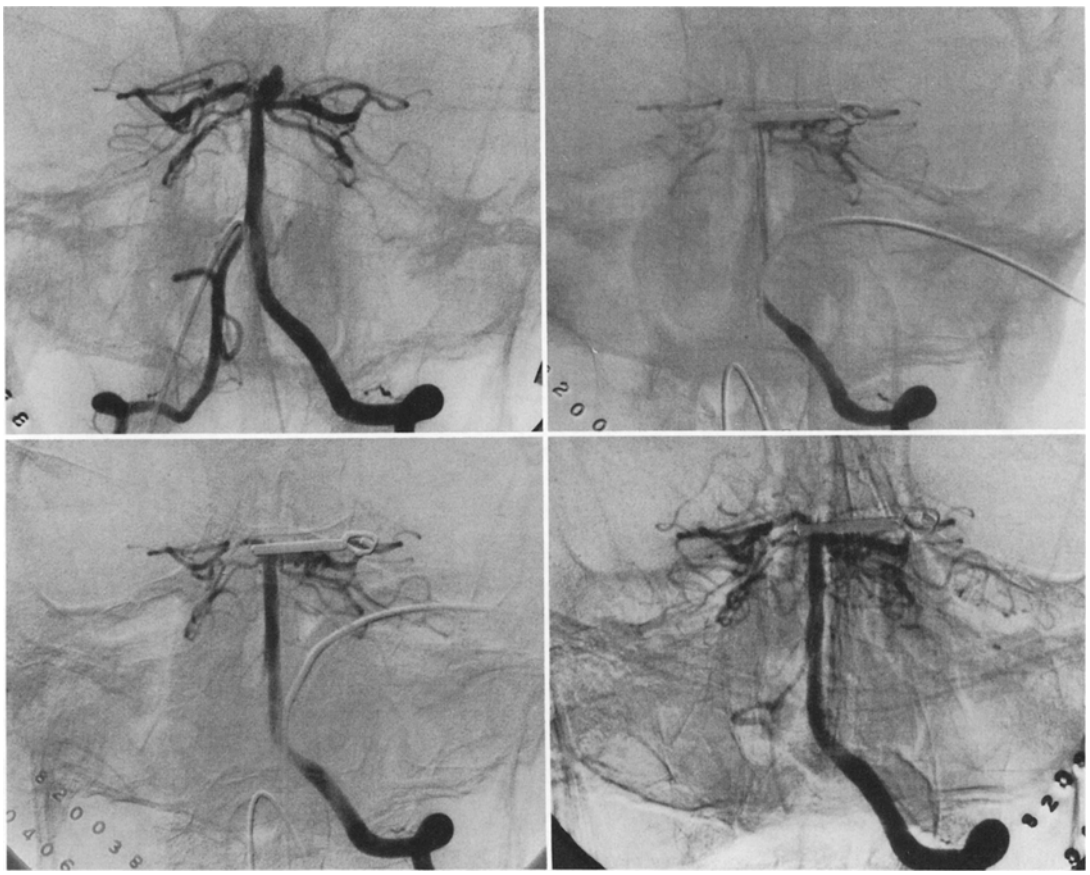

FIG. 3. Angiograms in Case 4. *Upper Left:* Left vertebral arteriogram demonstrating the basilar tip aneurysm as well as a normal caliber of the vertebral basilar lumen. *Upper Right:* Arteriogram obtained 5 hours after clinical deterioration showing severe spasm of the distal left vertebral artery, basilar artery, and both posterior cerebral arteries. The aneurysm has been clipped. *Lower Left:* Arteriogram obtained immediately after angioplasty demonstrating improvement in the luminal caliber of the vertebral basilar system and the proximal left posterior cerebral artery. *Lower Right:* Arteriogram obtained 1 week after angioplasty demonstrating continued patency of the vertebral basilar system.

mild confusion which resolved over the next 2 weeks. Follow-up angiography at 1 week demonstrated continued patency of the vertebral basilar system (Fig. 3 *lower right*).

Case 5. This 38-year-old man presented with an SAH due to an anterior communicating artery aneurysm. He was in Hunt and Hess clinical Grade I. He was transferred to our institution on Day 7 following the hemorrhage, and the aneurysm was successfully clipped on Day 8. The following day he developed left hemiparesis with 0/5 strength in the left lower extremity and 1/5 strength in the left upper extremity, which did not improve with hypervolemia and hypotension. Angiography performed within 3 hours of the onset of symptoms revealed severe spasm of the right supraclinoid carotid artery, right middle cerebral artery, and right anterior cerebral artery (Fig. 4 *left*). The right supraclinoid carotid and right middle cerebral arteries were successfully dilated (Fig. 4 *right*). There was marked improvement of flow in the middle and anterior cerebral distributions, although the A_1 segment of the anterior cerebral artery was not dilated due to acute angulation of the vessel origin. The patient demonstrated immediate clinical improvement following the procedure by regaining the ability to move his left lower extremity. The following morning the patient was normal neurologically, with Grade 5/5 power in the left arm and leg. At his 2-month follow-up examination his strength was normal without pronator drift.

Transcranial Doppler Ultrasonography Findings

Transcranial Doppler ultrasonography was used to assess the degree of arterial narrowing in two patients (Cases 7 and 9) before and after the procedure. This procedure, which was introduced by Aaslid, *et al.*,[1,2] uses ultrasound to detect changes in blood flow velocities induced by changes in vessel caliber caused by vasospasm. Normal mean velocity in the middle cerebral artery is 62 cm/sec.[2] Mean velocities of 120 cm/sec indicate mild vasospasm seen on angiography[1] and

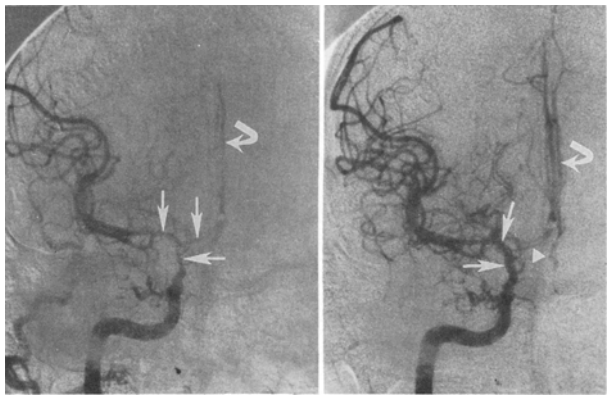

FIG. 4. Angiograms in Case 5. *Left:* Right carotid arteriogram demonstrating severe spasm of the right supraclinoid carotid artery, right proximal middle cerebral artery (MCA), and right proximal anterior cerebral artery (ACA, *straight arrows*). There is also poor filling of the ACA distribution *(curved arrow)*. *Right:* Following angioplasty there is improvement of the caliber of the right supraclinoid carotid and the right proximal MCA *(straight arrows)*. The proximal ACA *(arrowhead)* could not be dilated, but there is more flow in the ACA distribution *(curved arrow)*.

mean velocities greater than 200 cm/sec are associated with a high incidence of ischemic deficits and infarction.[23]

Figure 5 illustrates the sequential blood flow velocity recordings of four spastic arteries which were dilated using balloon angioplasty. Three of the four arteries studied did not show evidence of recurrence of high-velocity flow following the procedure.

Long-Term Outcome and Complications

Six patients had a good recovery at 1 month according to criteria established by Jennett and Bond.[16] Two patients were left with a moderate disability and two patients died. An acute complication was encountered in one patient (Case 1) who rebled from her unprotected aneurysm 1 week following the procedure. This was the only patient in the series who had an unprotected ruptured aneurysm at the time of angioplasty. A delayed complication occurred in Case 8 when a stroke developed in the left middle cerebral artery distribution 6 weeks following the procedure. An angiogram showed a branch occlusion of the middle cerebral artery with no source of emboli revealed on workup. The branch that occluded had been mechanically dilated with a more rigid balloon and guidewire than used in other cases, which results in greater vascular trauma than the silicone balloon we usually employ. We no longer use the more rigid balloon and guidewire system for vasospasm angioplasty. One patient (Case 3), in whom complete angioplasty was unsuccessful due to a prior right carotid occlusion, died 13 days following angioplasty from persistent coma and multiple medical problems.

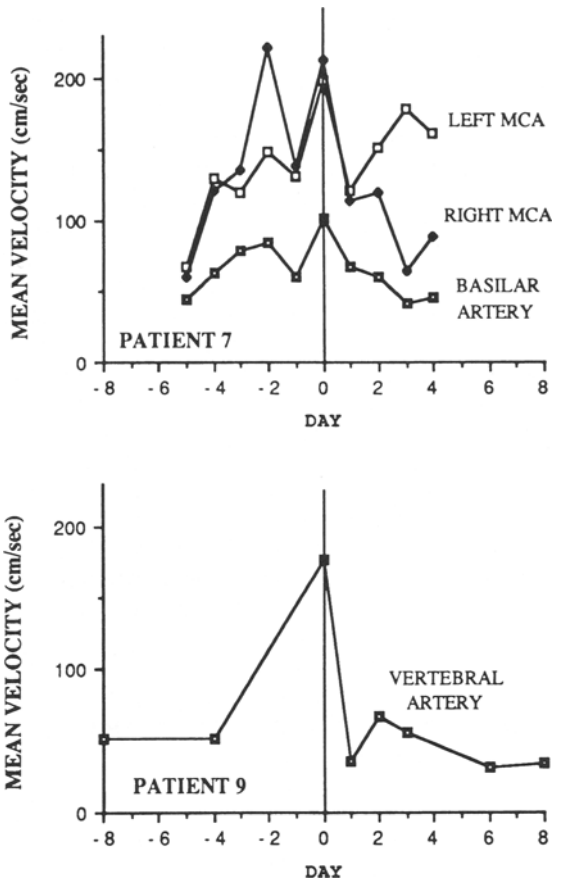

FIG. 5. Graph showing blood flow velocities measured by serial transcranial Doppler ultrasound over time in two patients (Cases 7 and 9). Day 0 indicates day of angioplasty. MCA = middle cerebral artery.

Discussion

The pathogenesis of vasospasm following SAH is not well understood, but the clinical syndrome of ischemic deficit due to vasospasm has been well described.[12] It has been observed in patients following SAH that the amount of blood deposited in the basal cisterns and subarachnoid space on CT scan is highly correlated to the degree and extent of subsequent vasospasm demonstrated by angiogram and TCD.[11,23]

Considerable controversy persists regarding the significance of morphological changes that occur in cerebral arteries following SAH. Both human autopsy[9,14,26] and intraoperative cerebral artery specimens[27,28] have shown ultrastructural changes in the vessel wall involving the intima, media, and periadventitial axons. In animal models of SAH a similar spectrum of morphological changes has been noted corresponding to the development of angiographic narrowing and the time course of vasospasm in humans.[3,8,10,19,29,30] Between 3 hours[21] and 3 days[29] after SAH, intercellular fluid accumulation has been apparent in the subintimal region of the artery, corresponding to a breakdown in endo-

thelial integrity at this time.[22] During the period of maximum vasospasm (5 to 14 days), a number of structural changes in the vessel walls have been described, including changes in endothelial morphology, smooth-muscle cell vacuolization and necrosis, proliferating myointimal cells, adventitial inflammation, and degeneration of perivascular axons.[3,8,10,19,20,29,30]

Structural changes in cerebral arteries after SAH may be related to the deposition of collagen in the arterial wall. Smith, *et al.*,[27] demonstrated an increased level of Type IV collagen in human artery specimens obtained at aneurysm surgery. In a primate model, Bevan, *et al.*,[7] have shown that cerebral arteries exposed to blood are less distensible *in vitro* than normal vessels. In this manner, prolonged arterial narrowing after SAH may represent a decrease in elasticity which results in the persistence of a constricted state. These structural changes in cerebral arteries after SAH may act alone or in combination to produce a stiff nondistensible vessel. Angioplasty may be effective by disrupting the extracellular matrix which maintains the artery in its narrowed state.

The effect of mechanically dilating the arteries in this condition is not well studied. The evidence we have from the follow-up angiography in three cases and TCD blood flow velocity readings in two cases indicates that the effect is sustained. In the other cases which demonstrated resolution of neurological deficits, the improvement was maintained, suggesting that the vascular narrowing did not recur to a clinically significant extent.

Although clinical benefits have been observed using hypervolemic hypertensive therapy and also using calcium channel blockers for the treatment of DID from vasospasm,[4–6,17,24] patients continue to deteriorate. The preliminary results in this report indicate that in a subgroup of patients who demonstrate progressive clinical deterioration from vasospasm despite therapy, percutaneous balloon angioplasty can be of benefit.

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