Effect of transluminal angioplasty on cerebral blood flow in the management of symptomatic vasospasm following aneurysmal subarachnoid hemorrhage

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In this study the authors have examined the effects of transluminal angioplasty on cerebral blood flow (CBF) in the management of intractable vasospasm following aneurysmal subarachnoid hemorrhage (SAH). Fourteen consecutively enrolled patients underwent attempted angioplasty with or without intraarterial infusion of papaverine. Twelve patients underwent pre- and postangioplasty xenon-enhanced computerized tomography (Xe-CT) scanning to measure regional CBF in 55 to 65 regions of interest (ROIs) per patient. Angioplasty was possible in 13 (93%) of 14 patients, with angiographically demonstrated improvement in all 13. Twelve (92%) of the 13 patients were neurologically improved following angioplasty; seven (58%) of the 12 patients who improved had a complete reversal of all delayed ischemic deficits. Angioplasty significantly decreased the mean number of ROIs at risk (11.4 ROIs pre- and 0.9 ROIs postangioplasty) (p < 0.00005, t-test). All patients had a reduction in the number of ROIs at risk after angioplasty; six (50%) of 12 no longer had any ROIs remaining at risk after angioplasty. Angioplasty significantly increased the mean CBF within at-risk ROIs (13 ml/100 g/minute pre- and 44 ml/100 g/minute postangioplasty) (p < 0.00005, t-test). All patients experienced an improvement in mean CBF in at-risk ROIs after angioplasty, with the mean CBF improving to above 20 ml/100 g/minute in all cases. No differences in the degree of improvement were found in patients who received intraarterial papaverine compared with those who did not. In the majority of patients with refractory vasospasm following SAH, angioplasty effectively dilated spastic arteries, reversed delayed neurological deficits, and significantly improved CBF in areas of brain at risk of infarction.

KEY WORDS • angioplasty • cerebral aneurysm • cerebral blood flow • subarachnoid hemorrhage • vasospasm • xenon study

CEREBRAL arterial vasospasm continues to be a major contributor to morbidity and mortality in patients who have suffered aneurysmal subarachnoid hemorrhage (SAH). Transluminal angioplasty has been advocated as a treatment strategy when symptomatic vasospasm persists despite maximum medical management. Although these reports demonstrate neurological improvement after angioplasty, the effects of arterial dilation on cerebral blood flow (CBF) have yet to be fully characterized in this setting. This is the first study designed to examine the effects of angioplasty on quantitative CBF measurements in a series of consecutively enrolled patients with intractable vasospasm following SAH.

Clinical Material and Methods

Patient Selection

Between June 1, 1990 and December 31, 1995, 404 patients underwent 444 operations for cerebral aneurysms at the University of Pittsburgh Medical Center. Of this group, 14 patients underwent angioplasty for the treatment of cerebral vasospasm that proved to be refractory to medical management.

All patients with aneurysmal SAH underwent surgery within 24 hours of admission to our institution unless they were hemodynamically unstable or moribund. All patients were closely monitored in a neurosurgical cerebrovascular intensive care unit. Standard medical management was used, including administration of nimodipine, ventricular intracranial pressure monitoring and treatment of hydrocephalus by cerebrospinal fluid drainage, maintenance of hyervolemia and a hematocrit level of 33 ± 2%, and optimization of the patient’s general hemodynamic and medical status. A xenon-enhanced computerized tomography (Xe-CT) CBF study was performed on postoperative Day 1 to serve as a baseline for future comparison. Patients who developed delayed neurological deficits were treated by maximizing hemodilution, hyervolemia, and cerebrospinal fluid drainage by using hypertensive therapy; they then proceeded immediately to Xe-CT scanning for assessment of anatomical structures and CBF. Patients whose CT scans revealed nonschematic causes for their delayed deficits, such as subdural hematoma, were man-
Effects of angioplasty on cerebral blood flow

![Flow diagram illustrating the management of patients with delayed neurological deficits using Xe-CT CBF data to identify patients with regional cerebral ischemia who are candidates for angioplasty.](image)

Patient Characteristics

The 14 patients studied ranged in age from 31 to 64 years (mean 47 ± 11 years); 86% were women. The Hunt and Hess grade on presentation ranged from II to V; 10 patients (71%) were assigned a grade of III or higher. The location of the aneurysm varied; six aneurysms (43%) were located in the posterior circulation. Thirteen (93%) of the 14 patients underwent successful clipping of their aneurysm prior to angioplasty. Surgery was performed on the day of the hemorrhage or the following day in 10 patients (71%). The episode of vasospasm that was refractory to medical management occurred between posthemorrhage Days 6 and 12 in all cases. Patient characteristics are summarized in Table 1.

Transluminal Angioplasty and Intraarterial Papaverine

Fifty-two vessels or vessel segments were subjected to angioplasty in 15 angiography sessions; one of the 14 patients required a repeated angioplasty for vasospasm in a different vascular territory 3 days after the first angioplasty. One patient underwent angiography with the intent of performing angioplasty but did not actually undergo the procedure because we were unable to position the balloon appropriately. Ten vessels in eight patients underwent intraarterial papaverine infusion (papaverine hydrochloride; Eli Lilly and Co., Indianapolis, IN) in addition to angioplasty. Vessels treated by angioplasty and intraarterial papaverine infusion are tabulated in Table 2.

For angioplasty, a standard transfemoral angiographic approach was used to place a guide catheter in the cerebral circulation. Angioplasty was performed using a micro-balloon inserted coaxially (Interventional Therapeutics Corp., Fremont, CA). The degree of balloon inflation was calculated so that it was proportional to the normal size of the vessel being dilated; whenever possible, attempts were made to return stenotic vessels to normal or near-normal caliber. When distal spasm was noted beyond the reach of conventional microballoon catheters, a 0.3% solution of papaverine dissolved in normal saline (300 mg/100 ml) was infused via microcatheters (Target Therapeutics, Fremont, CA) into the affected vessels at a rate of 3 to 7 ml/minute. Total doses of papaverine for each occasion ranged from 300 to 600 mg. Digital angiograms were obtained before and after each intervention (Fig. 2).

Grading of Angiograms

The pre- and postprocedure angiograms were graded according to the method of Kassell, et al. (0, no vasospasm; +, mild vasospasm with < 50% stenosis; ++, moderate vasospasm with 50% stenosis; ++++, severe vasospasm with > 50% stenosis). In addition, vasospasm was labeled as “diffuse” if more than 50% of the length of the vessel segment was involved or “focal” if less than 50%...
of the length of the vessel was involved. All angiograms were obtained immediately before and after the intervention. When both angioplasty and intraarterial papaverine were used, angiograms were graded between each procedure to determine the effect of each intervention on vessel caliber (Fig. 3).

### Xenon-Enhanced CT CBF Studies

All patients underwent preangioplasty Xe-CT CBF studies. Of the 13 patients in whom angioplasty was performed, 12 (92%) underwent postangioplasty CBF studies (one patient died prior to repeated CBF study). In one patient, pre- and postangioplasty CBF studies were technically inadequate because of patient-motion artifact; the CBF data were not interpretable and were therefore excluded from the CBF analysis. One patient underwent angioplasty on two occasions and, therefore, underwent two sets of pre- and postangioplasty CBF studies. In total, therefore, there were 12 sets of pre- and postangioplasty studies analyzed in 11 patients (Fig. 4).

Quantitative CBF studies were performed using standard CT scanners to which an independent system for xenon delivery and CBF calculation were added (Xe/CT System; Diversified Diagnostic Products, Houston, TX). Technical aspects of the Xe-CT studies have been described in previous publications. While patients inhaled a 33% Xe/67% O₂ mixture (XeScan; Praxair Pharmaceutical Gases, Danbury, CT) over a 4.5-minute period, CT images were obtained at three levels through the brain. During the studies, arterial blood pressure and PCO₂ were measured. Systolic blood pressure did not change by more than 20 mm Hg for the pre- and postangioplasty studies in each case. The mean pressure was 180 mm Hg for the pre- and postangioplasty studies in each case.

### TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Location of Aneurysm</th>
<th>Hunt &amp; Hess Grade</th>
<th>Day of SAH</th>
<th>Pre-ANEURYSM Aneurysm Clipped</th>
<th>Post-SAH Day of Vasospasm</th>
<th>Pretreatment Neurological Exam Findings</th>
<th>Posttreatment Neurological Exam Findings</th>
<th>Overall Improvement</th>
<th>Complications of Angioplasty</th>
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* ACA = anterior cerebral artery; ACoA = anterior communicating artery; exp = expressive; FP = frontoparietal; ICA = internal carotid artery; loc = localizing; no commands = patient not following commands; PCA = posterior cerebral artery; PCoA = posterior communicating artery; SCA = superior cerebellar artery; VA = vertebral artery; VF = visual field.
† Represents a return to best neurological examination prior to vasospasm episode.
‡ Aneurysm trapped.
§ Aneurysm not successfully clipped because of severe brain edema at operation.
Initial angiogram was negative.
Effects of angioplasty on cerebral blood flow

<table>
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<tr>
<th>Case No.</th>
<th>Location of Aneurysm</th>
<th>Vessels Treated W/ Angioplasty</th>
<th>Vessels Treated W/ Papaverine</th>
<th>Degree of Vasospasm on Angiogram</th>
<th>Angiographic Effects of Papaverine</th>
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*ACA = anterior cerebral artery; ACoA = anterior communicating artery; A₁, A₂, A₃ = A₁ and A₂ and A₃ segments; d = diffuse; f = focal; ICA = internal carotid artery; M₁, M₂, M₃ = M₁ and M₂ segments; NA = not applicable; nL = normal; NS = no CBF studies; PCA = posterior cerebral artery; PCoA = posterior communicating artery; P₁ = P₁ segment; post = posttreatment; pre = pretreatment; SCA = superior cerebellar artery; VA = vertebral artery; * = mild vasospasm with less than 50% stenosis; ++ = moderate vasospasm with 50% stenosis; +++ = severe vasospasm with more than 50% stenosis.

† Measurements of CBF are given as milliliters per 100 g per minute.
‡ Intracarotid papaverine administered before angioplasty.

Interpretation of CBF Values

Xenon-enhanced CT CBF data were analyzed by means of a computerized data analysis program that calculated the mean CBF within a series of 2-cm-wide circular regions of interest (ROIs) distributed through the cortical and subcortical areas. Three axial CT scan slices were...
studied for most patients (> 90%), yielding between 55 and 65 ROIs per patient. Determination of the mean CBF in all vascular territories could then be conducted. Because pre- and postangioplasty Xe-CT scans measured CBF according to a standardized protocol, direct comparisons could be made between each of the 55 to 65 ROIs both before and after intervention (Fig. 5). A total of approximately 1200 ROIs were analyzed in this manner and approximately 600 pre- and postangioplasty ROI comparisons were made in 11 patients.

Regions of interest with CBF values in the ischemic range (defined in this study as ≤ 20 ml/100 g/minute) were identified as regions at risk of infarction. The total number of ROIs at risk of infarction and the mean CBF within these ROIs were calculated pre- and postangioplasty. The ROIs within areas that corresponded to artifacts, catheters, or blood clots were excluded from the analysis.

**Clinical Assessment**

Detailed neurological examinations were performed on admission, daily, and before and after any other clinical change or intervention in all patients. Neurological deficits immediately pre- and postangioplasty were carefully documented and included in the analysis for all patients. Neurological findings are summarized in Table 1. All complications of angiography, angioplasty, intraarterial papaverine infusion, and Xe-CT scanning were carefully noted and included in the analysis.

**Results**

**Neurological Status**

Of the 13 patients who underwent angioplasty, 12 (92%) exhibited neurological improvement after the procedure in either level of consciousness, motor function, or speech. One patient underwent repeated angioplasty 3 days after the first procedure for a different delayed neurological deficit in a different vascular territory, but did not improve neurologically. One patient underwent attempted but unsuccessful angioplasty; this patient’s neurological status remained unchanged following the procedure.

After angioplasty, seven (58%) of the 12 patients who had improved neurologically returned to either their best neurological function prior to the vasospastic episode or to what would be expected in a normal examination. Ten patients had improved levels of consciousness and 10 patients had improved motor function after angioplasty. Two patients had aphasia as part of their delayed ischemic neurological deficit; both had normal speech after angioplasty. No patient neurologically declined immediately following the procedure. Details of neurological progress before and after angioplasty are outlined in Table 1.

**Angiographic Changes**

Angioplasty was technically possible in 13 of 14 patients on 14 occasions. In one patient angioplasty could not be performed because catheter positioning in the vessel to be dilated was unstable and believed to be unsafe. In all cases in which angioplasty could be performed, at least some degree of angiographic improvement was obtained. In three cases, complete resolution of vasospasm was demonstrated on the postprocedure angiogram; in the remainder of cases, at least some degree of vasospasm persisted. Intraarterial papaverine infusion was used in eight of 14 patients. Papaverine was found to have no angiographic effect in four (50%) of the eight patients. In the four patients in whom an angiographic effect was
Effects of angioplasty on cerebral blood flow

seen, it was minor compared to the effects of angioplasty. The angiographic results are presented in Table 2.

Cerebral Blood Flow Changes

Angioplasty significantly decreased the mean number of ROIs identified as “at risk” with a CBF less than or equal to 20 ml/100 g/minute (p < 0.00005; t-test: paired two sample assuming unequal variances, two tailed). All patients with ROIs identified as at risk prior to angioplasty had a reduction in the number of at-risk ROIs postangioplasty. The total number of ROIs at risk preangioplasty for the group was 137; the total number of ROIs remaining at risk postangioplasty was 11. The mean number of ROIs at risk preangioplasty was 11.4 ± 4.3 (standard deviation [SD]); the mean number of ROIs remaining at risk was 0.9 ± 1.6 (SD). Six (50%) of the 12 patients had improvements in CBF such that they no longer had any ROIs remaining at risk after angioplasty. The individual and group patient data are summarized in Table 2 and Figs. 6 and 7.

Angioplasty significantly increased the mean CBF within at-risk ROIs (p < 0.00005; t-test: paired sample for means, two tailed). All patients had an improvement in mean CBF in ROIs at risk preangioplasty. In all patients, the mean CBF in ROIs at risk preangioplasty was above the at-risk threshold of 20 ml/100 g/minute after interven-
tion. The mean CBF of ROIs at risk preangioplasty was 13 ± 2.1 ml/100 g/minute (SD); postangioplasty these ROIs had a mean CBF of 44 ± 13.1 ml/100 g/minute (SD). The individual and group patient data are summarized in Table 2 and Figs. 8 and 9.

There was no statistically significant difference in the number of ROIs remaining at risk posttreatment in the group of patients receiving angioplasty alone (mean 0.5 ROIs) versus the group receiving angioplasty plus intraarterial papaverine infusion (mean 1.3 ROIs) (p = 0.3; t-test: two sample assuming unequal variances, two tailed). Similarly, there was no significant difference in the degree of increase in posttreatment mean CBF of ROIs that were previously at risk in the group of patients who received angioplasty alone (mean 34.5 ml/100 g/minute) compared to the group that received angioplasty plus intraarterial papaverine infusion (mean 28.5 ml/100 g/minute) (p = 0.5; t-test: two sample assuming unequal variances, two tailed).

Complications Related to Angioplasty

There were two complications related to angioplasty in these patients (Table 1). The patient in Case 2 presented with a Hunt and Hess Grade II SAH from a left posterior communicating artery aneurysm that was clipped on the day after hemorrhage. On posthemorrhage Day 12, she redeveloped a right hemiparesis and expressive aphasia, which became refractory to hypertensive therapy. Her
CT scan revealed hypodensity in the left middle cerebral artery territory; Xe-CT imaging showed perfusion in the 10- to 20-ml/100 g/minute range within the hypodense territory. The patient underwent successful angioplasty; her speech and right arm strength returned to normal, but she developed a large left frontoparietal hemorrhage within 2 hours after the procedure and subsequently died.

The patient in Case 7 underwent angiography with the intention of undergoing angioplasty but angioplasty could not be performed. The catheter positioning within the left M1 segment was too unstable to attempt balloon inflation. The procedure was, therefore, aborted. We have included this patient as a complication because she underwent the risks of both angiography and the introduction of a microcatheter into the cerebral circulation but did not derive any benefit from this intervention.

Discussion

Interpretation of Data

The results of this study strongly indicate that transluminal angioplasty with or without intraarterial papaverine infusion benefits patients who develop delayed neurological deficits secondary to vasospasm caused by aneurysmal SAH. In the majority of cases, enlargement of vessel caliber can be achieved, causing a restoration of rCBF sufficient to improve neurological function. This study is unique in that it provides quantitative CBF information in the vast majority (92%) of consecutively enrolled patients who underwent treatment. For the first time, statistically significant quantitative improvements in blood flow in regions of brain considered to be at risk of infarction were demonstrated after intervention. All patients had decreases in the number of regions of brain at risk of infarction after angioplasty. Furthermore, in all patients, CBF in areas determined to be at risk improved to the degree that the mean rCBF in these areas was above the ischemic risk threshold after angioplasty. These improvements in tissue perfusion were long lasting; no patient required repeated treatment for recurrent vasospasm in the same vascular territory. There were two complications in the series of 14 patients. One patient underwent angiography but angioplasty was not technically possible. The other patient developed an intracerebral hemorrhage subsequent to angioplasty, which was likely caused by reperfusion of an infarct, a complication also encountered previously by Higashida, et al.10

Protocol Considerations

The 14 consecutively enrolled patients described in this study were derived from a population of patients who were managed according to an ongoing CBF-based protocol for the management of delayed ischemic neurological deficits following SAH. Delayed neurological deficits following SAH have many potential causes, including hydrocephalus, hemorrhage, metabolic factors, edema, ischemia, and perhaps other inexplicable causes. Although ischemia secondary to vasospasm is often assumed to be the cause of delayed neurological deficits when other obvious structural and metabolic causes have been ruled out, it has been our goal to test the validity of that assumption directly by means of Xe-CT CBF testing. With the exception of the second angioplasty session required in Case 9, the patients in this report only advanced to angioplasty if ischemia was identified on CBF testing. This careful selection of patients ensured that patients with other potential (but perhaps not as easily explainable) causes of delayed deficits were not subjected to an invasive therapy that would potentially be of no benefit. Indeed, although severe vasospasm was identified during the second angioplasty session in Case 9, successful arterial dilation did not improve this patient’s condition, a finding that may be related to the observation that this patient did not have documentable ischemia on CBF testing prior to the intervention. The finding of vasospasm at angiography is not sufficient to confirm that vasospasm is the cause of a patient’s delayed deficit because angiographic vasospasm frequently occurs without clinical significance.15,19,21,25 Cerebral blood flow testing with Xe-CT scanning quantitatively investigates whether angiographic spasm is causing ischemia in the territories in question.

Although transcranial Doppler (TCD) ultrasonography has shown promise in identifying patients with vasospasm...
and has become widely used,1,3,7,9,22 there are several shortcomings of this technology with respect to the diagnosis of symptomatic vasospasm. Insonation of blood velocity in the major cerebral vessels only provides indirect information about proximal vessel caliber. The TCD does not provide any information about more distal vessel segments beyond the window of insonation. Furthermore, even if the presence of proximal arterial narrowing is correctly identified, TCD does not directly address the more important question of the effects of this vasospasm on the rCBF in the associated vascular territories. In short, although TCD may aid in the determination of radiographic vasospasm, it does not necessarily assist in the identification of ischemia. Therefore, TCD does not identify patients whose delayed neurological deficits are caused by their vasospasm.3 Indeed, our initial enthusiasm for using TCD in diagnosing symptomatic vasospasm22 was tempered when we discovered that the increased TCD velocities that we believed represented vasospasm causing potential ischemia actually turned out to correlate with increased rCBF in the vascular territory of the vessels studied.3 Thus, although TCD may aid in the determination of radiographic vasospasm, it does not necessarily assist in the identification of ischemia. Therefore, TCD does not identify patients whose delayed neurological deficits are caused by their vasospasm.3

Role of Intraarterial Papaverine Infusion

Some patients in this series received intraarterial papaverine infusion in addition to angioplasty as an attempt to treat vasospasm that was distal to the reach of conventional catheters or to dilate segments that were difficult to subject to angioplasty. Our impression is that intraarterial papaverine infusion is of marginal benefit in these patients. This impression is substantiated by four observations. 1) Angiograms performed after infusion of papaverine revealed that there was little, if any, improvement in vasospasm compared with the results of angioplasty. 2) There was no significant difference in the degree of CBF improvement or the number of ROIs considered to be at risk of infarction in patients who received angioplasty coupled with intraarterial papaverine infusion compared with patients who underwent angioplasty alone. 3) Our experience with intraarterial papaverine infusion without concomitant angioplasty reveals inconsistent improvements in clinical outcome and CBF: clinical improvement was observed on only 22% of occasions in a group of 10 similarly selected patients; CBF augmentation was observed on only 46% of occasions, with the changes being smaller than those described in this report (unpublished data). These results are very similar to those of Kassell, et al.,14 who demonstrated reversal of vasospasm in 57% of 12 patients and clinical improvement in only 25% of patients. 4) Intraarterial papaverine infusion tends to be a short-lived intervention; 50% of patients who underwent this infusion alone required repeated treatments because of recurrence of vasospasm (unpublished data). No patient in this series required repeated treatment for recurrent vasospasm in the same distribution after angioplasty. This supports the notion that the long-lasting effects of angioplasty exceeded the variable and short-lived effects of intraarterial papaverine infusion.

Selection of At-Risk ROIs

The 2-cm ROIs that we selected represent mixed cortical blood flows. In healthy volunteers, the mean CBF measured by Xe-CT is 51 ± 10 ml/100 g/minute; pure gray matter averages 84 ± 14 ml/100 g/minute and pure white matter measures 20 ± 5 ml/100 g/minute.23 Mixed cortical flow values less than 20 ml/100 g/minute are, therefore, clearly abnormal. Cerebral blood flow less than
20 ml/100 g/minute has been found to correlate closely with neurological deficits. Furthermore, in studies of symptomatic vasospasm following SAH, infarction did not occur in patients with mixed cortical CBF values greater than 18 ml/100 g/minute, whereas mixed cortical values less than 15 ml/100 g/minute were predictive of infarction.6,25

These data support the concept that mixed cortical ROIs with rCBFs less than or equal to 20 ml/100 g/minute may be considered areas at risk of infarction. The identification of such at-risk areas allowed for straightforward comparison of CBF in discrete regions of brain before and after angioplasty. In this manner, quantification of the degree of efficacy of angioplasty in improving rCBF in areas where it is most needed could be easily conducted.

Review of the Literature

Previous investigators have demonstrated the angiographic and clinical results of transluminal angioplasty in similar groups of patients. Zubkov, et al.6,26 described the first use of angioplasty for vasospasm following SAH in 1984. Since that original report Higashida, et al., reported first 13 cases26 and later 28 cases1 of patients with vasospasm treated by angioplasty, citing clinical improvements in 69% and 61% of patients, respectively. Newell and colleagues8 performed angioplasty in 10 patients, noting neurological improvement in 80%. Coyne and associates1 reported neurological improvement in only 31% of 13 patients treated with angioplasty for vasospasm following SAH. In the context of these reports, our neurological improvement rate of 92% compares favorably.

The addition of papaverine in some cases may have contributed to our slightly more favorable results, although this is not the preferred explanation, for reasons discussed previously. Another possible explanation for our favorable results is that our group of patients may be more accurately selected as having vasospasm as the cause of their delayed deficits. None of the prior reports used quantitative Xe-CT CBF measurements as part of the selection criteria to define those patients with ischemia who will advance to angioplasty. If angioplasty is used less selectively in patients who have other, perhaps inexplicable, causes for their neurological decline, it is expected that fewer of them will improve with dilation of their vessels. Coyne and associates1 also made the observation that patients who underwent angioplasty at a later time were less likely to have a reversal of their deficit. The rapid and aggressive approach we used also may have contributed to an improvement in outcomes.

Few investigators have examined the effect of angioplasty on CBF. Investigators at our institution previously reported the case of a patient who developed vasospasm after a skull base tumor resection and improved neurologically after angioplasty of the stenotic vessels.21 This patient was demonstrated by Xe-CT scans to have a marked improvement in CBF after the procedure, from 3 to 5 ml/100 g/minute before angioplasty to 17 to 24 ml/100 g/minute after the procedure. Lewis, et al.,17 used brain single-photon emission CT (SPECT) scanning to evaluate CBF before and after angioplasty in 10 patients with SAH-induced vasospasm. They noted improved rCBF in 90% of patients, an observation similar to our own. These investigators were able to calculate an 8.8% increase in CBF in the anterior circulation by comparing cerebellar flows. Unfortunately, SPECT scanning cannot be used to determine CBF quantitatively. Reliance on corticocerebellar ratios assumes that cerebellar flows are constant, which may not always be the case, especially when vertebrobasilar vasospasm is present, as it was in 40% of their patients. The present study is the first systematic documentation of quantitative CBF changes in a series of patients undergoing angioplasty for intractable vasospasm.

Implications for Management of Vasospasm

The results of this study confirm the hypothesis that transluminal angioplasty can increase vessel caliber in vasospastic vessels in a manner that is sufficient to restore blood flow to regions of brain at risk for infarction. Xenon-enhanced CT scanning in these patients is proving to be a simple tool for use in identifying patients with ischemia as the cause of their delayed deficit after SAH. Serial Xe-CT scans obtained after intervention with hypertensive therapy and angioplasty immediately demonstrate the efficacy of these interventions in a reproducible, quantitative manner. Xenon-enhanced CT scanning may prove to be especially valuable in this setting, given the widespread availability of CT scanners and the short duration of the test (4.5 minutes of xenon inhalation time). Because most Xe-CT scans are obtained at times when patients would normally require conventional CT scans, such as a change in neurological status, additional transportation or equipment transfer was not necessary (as would be the case with SPECT scanning).

The most distinct value of Xe-CT scanning in these patients is its ability to pinpoint the ischemic cause of a delayed clinical deterioration, especially in patients who are comatose or severely neurologically injured initially, those who develop global neurological deficits, or those whose examinations are difficult to follow in the intensive care unit environment. The ease of obtaining repeated Xe-CT studies at 20-minute intervals enables the neurosurgeon to manipulate physiological variables (such as PaCO2 and blood pressure) to understand their effects on CBF and cerebrovascular physiology. As summarized in Fig. 1, cerebral autoregulation was studied in many patients in this series by performing Xe-CT CBF studies at higher and lower blood pressures. In this manner, the efficacy of hypertensive therapy was directly tested. Patients who did not derive beneficial effects on CBF from pressor medications or those whose CBF was paradoxically higher when the patient was not on pressor medications were identified and managed by discontinuing hypertensive therapy, thus avoiding its potentially harmful effects. Similarly, only those patients with ischemia or hypertension-dependent marginal ischemia were subjected to angioplasty. We believe that the high degree of success in this series of patients relates to careful screening of patients and judicious use of hypertensive therapy and angioplasty only after diagnostic evaluation of the cerebrovascular physiology and its relationship to CBF.

Although angioplasty has been traditionally reserved for those patients with delayed ischemic neurological deficits who do not respond to maximum medical therapy
Effects of angioplasty on cerebral blood flow

with pressor medications, we have moved toward a more aggressive approach by advancing to angioplasty in patients who improve clinically with hypertensive therapy but who, nevertheless, demonstrate tenuous areas of hypertension-dependent marginal perfusion on Xe-CT CBF studies. We believe that such patients have lost their ability for cerebrovascular autoregulation and are not likely to respond to hypertensive therapy eventually. We postulate that these patients would therefore benefit from earlier intervention with angioplasty to improve perfusion to marginal tissue before irreversible ischemic changes ensue.

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