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Imaging of Blood Flow Changes Following Angioplasty for Treatment of Vasospasm

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Vasospasm is a major source of morbidity and mortality after subarachnoid hemorrhage (SAH); it can result in temporary or permanent delayed ischemic deficits. Thirty percent of patients who survive the initial hemorrhage will develop symptomatic intracranial vasospasm 3 days to 2 weeks after the event [1, 2]. Medical regimens involving pharmacologic agents and hypertensive hypervolemic therapy have achieved only limited success [3–5].

Recent reports of microballoon angioplasty of the intracranial arteries narrowed by vasospasm have been encouraging [6–10]. This technique may offer an effective therapy for a subgroup of patients, who, despite aggressive medical therapy, still show signs of progressive deterioration. Improved cerebral blood flow (CBF) following angioplasty has been inferred by clinical examination and transcranial Doppler sonography [8]. Zubkov et al. [6] demonstrated improved CBF with the use of xenon-133. To date, however, there have been no direct CBF imaging studies showing the effect of angioplasty.

We report a patient in whom xenon CT CBF studies done immediately before and after angioplasty document the effect of the procedure.

Case Report

A 41-year-old man presented with a recurrent skull base myxoid chondrosarcoma that had been partly resected at two sessions, 19 and 6 years, respectively, before the current admission. Preoperative evaluation included a cerebral angiogram, which showed slight irregularity and posteromedial displacement of the cavernous segment of the right internal carotid artery (ICA) but no significant narrowing. Vertebral angiography showed elevation of the right posterior cerebral artery (PCA) and the right superior cerebellar artery (SCA) (Fig. 1A). A balloon test occlusion of the right ICA with a 15-min clinical examination followed by a xenon CT CBF study showed the patient to be at minimal risk for ICA occlusion (Fig. 1B) [11].

The patient underwent the first stage of skull base resection, with removal of a large segment of skull base on the right: The middle cranial fossa, cavernous sinus, sphenoidal sinus, and petrous apex

were resected, and a saphenous vein graft was used to bypass the cavernous segment of the ICA.

The postoperative course was complicated by a left thalamic infarct and a CSF leak that required surgical repair. Five days after skull base resection, the patient had a hypertensive and tachycardiac episode, after which he became unresponsive to verbal commands, developed tonic extension of the lower extremities, and retained only semipurposeful movements of the left upper extremity. CT showed hemorrhage into areas of previous infarction in the left thalamus. Over the next 6 days, the patient showed slight improvement initially, but his condition again deteriorated. Subsequent CT scans failed to show any significant change.

A second xenon CT CBF study was done 12 days after skull base resection because of the patient's continued poor clinical condition. This showed areas of markedly decreased flow in both PCA territories, and the noncontrast CT was negative for a corresponding area of infarction (Figs. 1C and 1D). Immediate angiography showed spasm of the distal basilar and both proximal PCAs and SCAs (Fig. 1E). An ITC (International Therapeutics Corp., So. San Francisco, CA) angioplasty balloon was introduced and dilatation of the proximal portions of both PCAs was performed (Fig. 1F). A third xenon CT CBF study performed within an hour of the angioplasty and within 2 hours of the preangioplasty CBF study demonstrated return of normal flow to both occipital lobes (Fig. 1G). A final xenon CT CBF study obtained 2 days after the angioplasty showed persistence of normal flow in the occipital lobes and a corresponding lack of infarction as defined by noncontrast CT.

Visual evoked potentials obtained 6 days after the angioplasty showed normal response in the right occipital lobe and abnormal response in the left. The left-sided changes were consistent with injury to the optic radiation, and since CT of the left occipital lobe remained normal, it was assumed that these changes were likely caused by the thalamic hemorrhage.

Discussion

Angioplasty of vasospasm from SAH has already proved to be a promising technique in the prevention of ischemic sequelae. A critical issue for consideration is the selection of patients who would benefit from this admittedly hazardous procedure. The decision to perform angioplasty has been based on (1) clinical criteria, (2) lack of infarction as determined

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Fig. 1.—41-year-old man with recurrent skull base myxoid chondrosarcoma.

A, Preoperative left vertebral angiogram shows slight deviation to the left of superior portion of basilar artery (*arrowhead*) and elevation of right superior cerebellar artery (SCA) and right posterior cerebellar artery (PCA).

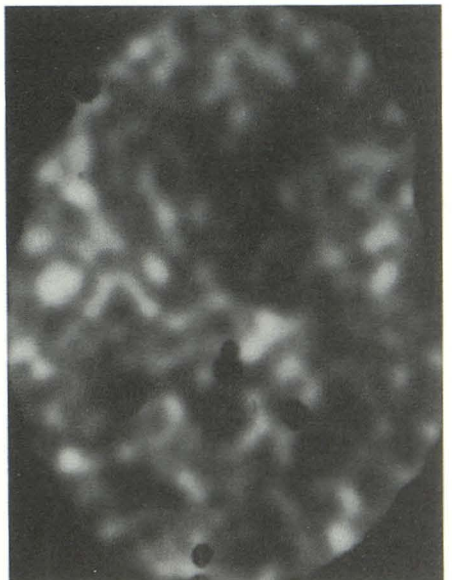
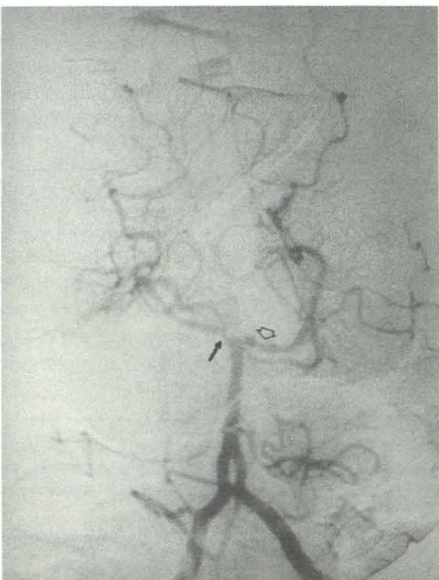
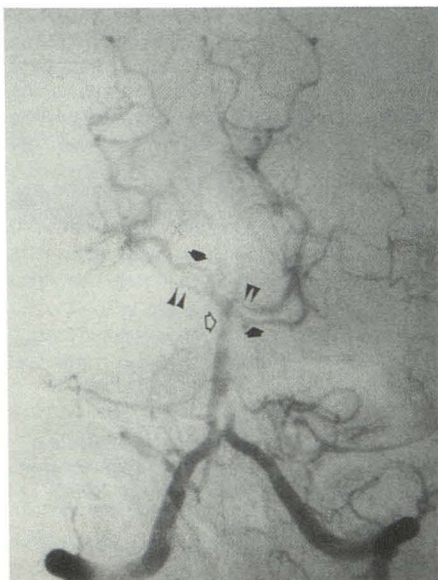
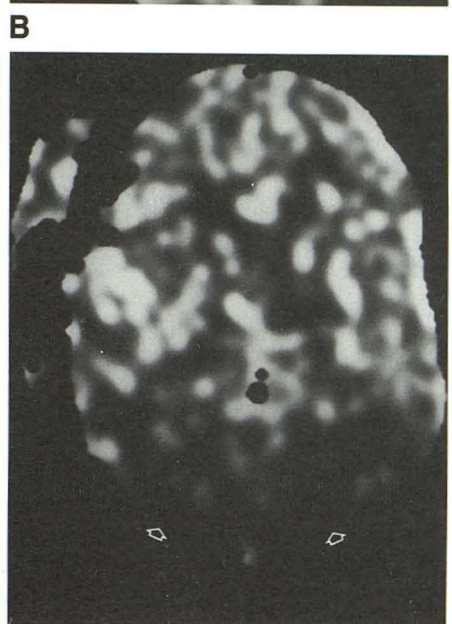
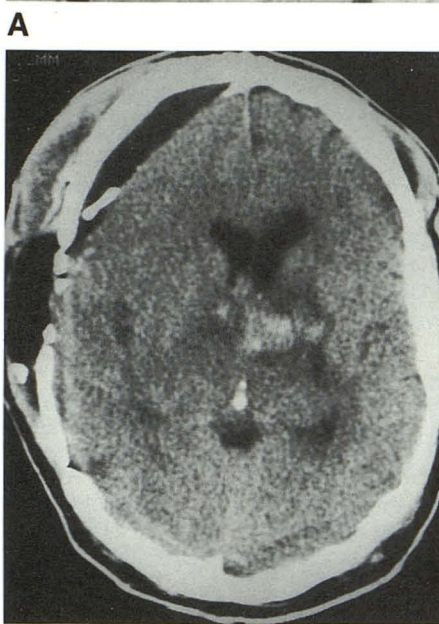
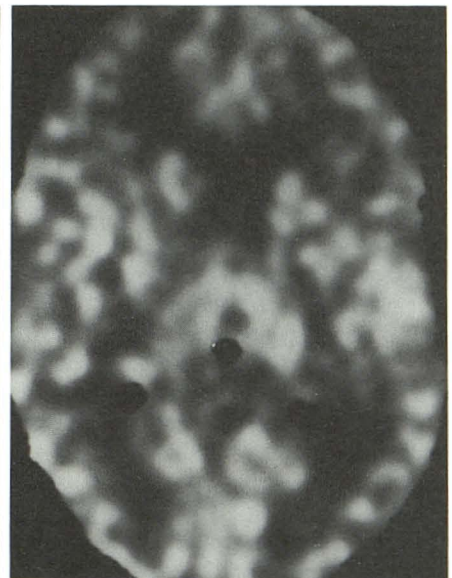
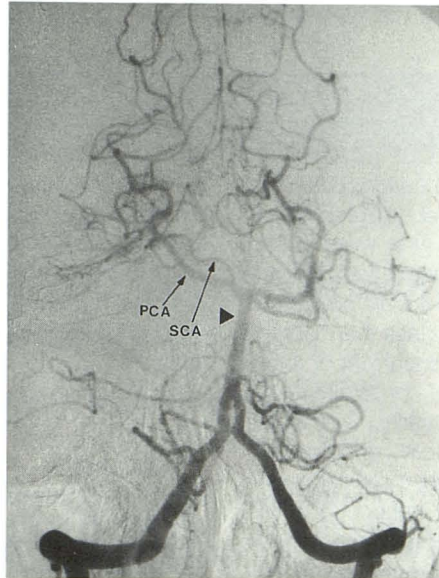
B, Xenon CT cerebral blood flow map obtained with right internal carotid artery occluded as part of balloon test occlusion shows normal blood flow. Regional cortical flow at this level ranged from 40 to 60 ml/100 ml/min.

C and D, Noncontrast CT scan (C) obtained 12 days after original surgery shows a postoperative subdural collection on the right and a left thalamic hemorrhage. Corresponding xenon CT cerebral blood flow map (D) shows critically low flow in both PCA territories (*arrowheads*). Regional flow measured 5.2 ml/100 ml/min in the right occipital lobe, and 2.6 ml/100 ml/min in the left occipital lobe.

E, Preangioplasty left vertebral angiogram shows spasm of distal basilar artery (*open arrow*), both proximal PCAs (*double arrowheads*), and both proximal SCAs (*closed arrows*).

F, Left vertebral angiogram obtained after angioplasty of proximal portions of both PCAs shows dilatation of both proximal PCAs. A focal area of narrowing persists at origin of right PCA (*closed arrow*) and proximal left PCA (*open arrow*).

G, Xenon CT cerebral blood flow study immediately after angioplasty shows improved blood flow in both occipital lobes. Regional blood flow measured 17.3 ml/100 ml/min in the right occipital lobe, and 23.6 ml/100 ml/min in the left.



E

F

G

by CT or MR; and (3) angiographic evidence of vasospasm [6, 7, 9, 10]. The authors of one report used transcranial Doppler sonography to estimate changes in CBF [8]. Zubkov et al. [6] reported the use of xenon-133 to measure CBF.

Using xenon CT CBF studies, Yonas et al. [12] identified those patients with vasospasm from SAH who were at increased risk for ischemic deficits. They observed that cortical regions with flow values less than 15 ml/100 ml/min predicted severe neurologic deficits. Regions with flow less than 12 ml/100 ml/min were always accompanied by concurrent or later infarction. Patients who had altered levels of consciousness, mild to moderate motor and/or sensory deficits, and flow values above 18 ml/100 ml/min did not typically progress to CT-defined infarction; they usually experienced clinical resolution of the deficit [12]. At our institution, xenon CT CBF information guides appropriate medical therapy to optimize CBF in this critical group of patients.

Blood flow threshold for irreversible ischemia, as determined by multiple techniques, is at or below 15 ml/100 ml/min [12-14]. A newer and less well-known concept is the time threshold for irreversible ischemia. This holds that above a certain minimal blood flow, tissue viability will be maintained indefinitely. Below this critical blood flow threshold, there is a correlative time threshold, the two of which together determine when ischemia is irreversible. For example, Bell et al. [13] demonstrated a time threshold for irreversible ischemia in a primate model. In their study, if blood flow was under 40% of normal, then ischemia became irreversible after 30 min.

The window of opportunity exists when reperfusion can be established within a critical time from onset of ischemia. Restoration of blood flow to infarcted brain carries an increased risk of hemorrhage and cerebral edema, with no known benefit to justify the risk. In the case of intracranial angioplasty for vasospasm, the added risks inherent to the procedure (e.g., arterial rupture, intimal injury, and thrombus formation) mandate well-defined patient selection criteria.

Our case demonstrates rescue of ischemic tissue by angioplasty. Using previous criteria, we would have considered this tissue to be irreversibly damaged [12]. The previous data were obtained in the preangioplasty era, and therefore reflect the suboptimal results of using only aggressive medical therapy. In this instance, there was not enough time at low blood flow for infarction to occur.

Microballoon angioplasty for the treatment of intracranial vasospasm is a promising technique still in its infancy. Several unresolved issues need to be addressed in future studies: When is the optimal time to perform the procedure? Which

subgroup of patients would benefit? What are the absolute contraindications? How far distally in the intracranial arteries is it necessary to dilate? [10]. We believe blood flow data may answer many of these critical questions and that CBF studies should be a part of future examinations. These data can also provide information regarding the effect of proximal angioplasty in relation to perfusion of the microvasculature [9]. Flow data obtained before and after angioplasty will help define relative risk-benefit ratios for each subgroup of patients.

REFERENCES

1. Allcock JM, Drake CG. Ruptured intracranial aneurysms—the role of arterial spasm. *J Neurosurg* **1965**;22:21–29
2. Heros RC, Zervas NT, Varsos VG. Cerebral vasospasm after subarachnoid hemorrhage: an update. *Ann Neurol* **1983**;14:599–608
3. Wilkins RH. Attempts at prevention or treatment of intracranial arterial spasm: an update. *Neurosurgery* **1986**;18:808–825
4. Awad IA, Carter LP, Spetzler RF, et al. Clinical vasospasm after subarachnoid hemorrhage: response to hypervolemic hemodilution and arterial hypertension. *Stroke* **1987**;18:365–372
5. Seiler RW, Reulen HJ, Huber P, et al. Outcome of aneurysmal subarachnoid hemorrhage in a hospital population: a prospective study including early operation, intravenous nimodipine, and transcranial Doppler ultrasound. *Neurosurgery* **1988**;23:598–604
6. Zubkov YN, Nikiforov BM, Shustin VA. Balloon catheter technique for dilatation of constricted cerebral arteries after aneurysmal SAH. *Acta Neurochir (Wien)* **1984**;70:665–679
7. Higashida RT, Halbach VV, Cohen LD, et al. Transluminal angioplasty for treatment of intracranial arterial vasospasm. *J Neurosurg* **1989**;71:648–653
8. Newell DW, Eskridge JM, Mayberg MR, et al. Angioplasty for the treatment of symptomatic vasospasm following subarachnoid hemorrhage. *J Neurosurg* **1989**;71:654–660
9. Higashida RT, Halbach VV, Dormandy B. New microballoon device for transluminal angioplasty of intracranial arterial vasospasm. *AJNR* **1990**;11:233–238
10. Brothers MF, Holgate RC. Intracranial angioplasty for treatment of vasospasm after subarachnoid hemorrhage: technique and modifications to improve branch access. *AJNR* **1990**;11:239–247
11. Erba SM, Horton JA, Latchaw RE, et al. Balloon test occlusion of the internal carotid artery with stable xenon/CT cerebral blood flow imaging. *AJNR* **1988**;9:533–538
12. Yonas H, Sekhar L, Johnson DW, et al. Determination of irreversible ischemia by xenon-enhanced computed tomographic monitoring of cerebral blood flow in patients with symptomatic vasospasm. *Neurosurgery* **1989**;24:368–372
13. Bell BA, Symon L, Branston NM. CBF and time thresholds for the formation of ischemic cerebral edema and effect of reperfusion in baboons. *J Neurosurg* **1985**;62:31–41
14. Powers WJ, Grubb RL, Baker RP, et al. Regional cerebral blood flow and metabolism in reversible ischemia due to vasospasm. *J Neurosurg* **1985**;62:539–546