Serial measurement of regional cerebral blood flow in patients with SAH using $^{133}$Xe inhalation and emission computerized tomography

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A noninvasive three-dimensional method for measuring cerebral blood flow (CBF), xenon-$^{133}$ inhalation and emission computerized tomography, was used to investigate the CBF changes accompanying delayed neurological deterioration following subarachnoid hemorrhage (SAH). A total of 67 measurements were performed on 20 patients in Hunt and Hess' clinical Grades I to III in the first 21 days post SAH. Five patients with normal CBF tomograms on admission developed delayed neurological deficits in the 2nd week after hemorrhage, at which time repeat CBF tomograms in four patients revealed large areas of well defined regional flow decrease in the vascular territories of the anterior or middle cerebral arteries. Severe vasospasm was noted in three of these patients in whom arteriography was performed in the 2nd week post SAH. Diffuse bihemispheric CBF decreases were noted later in the course of delayed neurological deficits; however, measurements obtained soon after the onset of focal symptoms suggest that the only CBF decreases directly produced by vasospasm in Grade III patients are regional changes.

KEY WORDS: subarachnoid hemorrhage, vasospasm, cerebral blood flow, $^{133}$Xe inhalation tomography

A wealth of clinical and radiographic evidence points to cerebral ischemia due to arterial vasospasm as the major cause of delayed neurological deterioration following subarachnoid hemorrhage (SAH). Using intracarotid xenon-$^{133}$ ($^{133}$Xe) or positron-emitting isotopes, various investigators have documented regional as well as hemispheric decreases in cerebral blood flow (CBF) in SAH patients in all clinical grades. Regional flow decreases have been found to correlate with the presence of focal neurological deficits, and with computerized tomography (CT) and autopsy evidence of cerebral infarction. While measurements obtained with intracarotid techniques are usually limited because of their invasiveness to one or two studies of a single hemisphere in each patient, preventing firm conclusions from being drawn about the timing of CBF changes in relation to clinical events. As an alternative to the intracarotid methods, noninvasive techniques using inhaled or intravenously administered $^{133}$Xe and fixed arrays of detectors have been applied in this setting in an attempt to more closely monitor the CBF changes associated with ischemic events. These methods allow repeated measurement of CBF in both hemispheres, but they have been found in other settings to be plagued by errors introduced by "cross talk" between the two hemispheres, and in some cases by an inability to detect infarctions documented on CT scanning. Their role in the investigation of focal ischemia has yet to be defined.

New methods of CBF measurement using emission CT in association with inhaled or intravenously administered $^{133}$Xe or positron-emitting isotopes have recently been developed. These methods allow the measurement of regional CBF changes in three dimensions and with greater precision than is possible with the other noninvasive techniques, in part because the CT principle avoids superimposition of tissues and hence facilitates the detection and localization of low-flow areas.

The usefulness of $^{133}$Xe inhalation and emission CT in the investigation of transient cerebral ischemia and stroke has been previously reported. The present study reports the application of this technique to the serial measurement of regional CBF changes in the early period following SAH in an attempt to better identify the changes accompanying delayed neurological deterioration.
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Clinical Material and Methods

Clinical Material

The series was comprised of 20 patients admitted to the neurosurgery service of the Rigshospital in Copenhagen with a verified SAH during the period from June 1 through December 31, 1982. There were 14 women and six men, with a mean age of 44 years (range 20 to 62 years). Clinical grading of the patients was performed according to the classification initially suggested by Hunt and Hess, except that intercurrent illness and angiographic vasospasm were not considered criteria for reduction in grade. A delayed neurological deficit was defined as a focal deficit developing after admission not due to surgery, rebleeding, or hydrocephalus. Only patients in Grades I, II, and III on admission were included, as it was considered that subsequent clinical and CBF changes could be more clearly evaluated in this group. The series was not consecutive in that a total of 23 SAH patients were admitted in Grades I, II, and III during the study period. Of the three Grade I patients not studied for technical reasons, two underwent uncomplicated craniotomies for aneurysm clipping, and one was well until left moribund by a rebleed.

All patients underwent CT scanning on admission and arteriography shortly thereafter. Each patient had at least one bilateral carotid arteriogram, and all but two had vertebral arteriograms. Of the 20 patients, 17 had aneurysms identified angiographically. Angiographic vasospasm was evaluated and graded by a neuroradiologist. The degree of vasospasm was considered "severe" if the lumen of the internal carotid artery, anterior cerebral artery, or middle cerebral artery (MCA) was narrowed by 50% or more when compared to normal vessels on other studies of the same patient. Vasospasm was termed "mild" if only minimal changes were present, and "moderate" if it fell between these two grades.

CBF Measurement

The CBF was measured with a Tomomatic 64, which utilizes a rotating array of 64 Na-I crystals to monitor the cerebral uptake and washout of $^{133}$Xe from three brain slices simultaneously. The scanner was oriented parallel to the orbitomeatal line and centered 1, 5, and 9 cm above it. The $^{133}$Xe was inhaled for 1.5 minutes, reaching a maximum concentration of 10 mCi/liter, which yields a calculated radiation dose per study of 0.06 rads to the gonads and 0.6 rads to the lung. Counts from the head were summed for four periods (the 1.5-minute period of $^{133}$Xe inhalation, and each of three subsequent 1-minute periods), and an arterial input curve was estimated from the output of a single collimated detector positioned over the right lung. By a conventional filtered back-projection algorithm, flow values were calculated for each pixel in a 32 x 32-

* Tomomatic 64 manufactured by Medimatic, Inc., Copenhagen, Denmark.

A total of 65 CBF measurements were obtained on the 20 patients in the first 21 days after SAH. During CBF measurement, patients were in a relaxed resting state.

A hemispheric CBF may be calculated by averaging the flow values for all pixels in one hemisphere in the slice centered 5 cm above the orbitomeatal line. The hemispheric CBF values so measured in 10 normal volunteers (hospital staff, mean age 43 years) were 56 ± 5 ml/100 gm/min (mean ± standard deviation). These 10 subjects were each restudied twice at 1-week intervals, revealing the standard deviation of the difference between two hemispheric CBF measurements in the same subject to be 5 ml/100 gm/min.

Experience with normal volunteers has revealed a stable, bilaterally symmetrical pattern of CBF distribution based on resting blood flow levels in different anatomic structures (Fig. 1). In a normal subject, changes in global flow induced by changes in arterial pCO$_2$ do not alter this pattern. Just as conventional CT abnormalities can be recognized by their disruption of bilateral symmetry or by differences from the pattern of normal anatomic densities, focal CBF changes can be identified by their asymmetrical nature and by differences from the stable flow distribution of normal subjects at rest.

A total of 65 CBF measurements were obtained on the 20 patients in the first 21 days after SAH. During CBF measurement, patients were in a relaxed resting state.

Fig. 1. Case 10. Normal cerebral blood flow pattern in a 31-year-old woman who was neurologically intact 10 days following the rupture of a left internal carotid artery aneurysm. Left: The cerebellum is seen in the posterior half of the slice centered 1 cm above the orbitomeatal line, which is dominated anteriorly by an airway artifact created by $^{133}$Xe in the ethmoid and sphenoid sinuses. Right: Flow values depicted for cortex over the convexity of the brain reflect the proximity of low flow in the overlying skull and scalp and in the underlying white matter, which influence the calculations with a partial volume effect. Where layers of cerebral cortex are opposed, as in the interhemispheric and Sylvian fissures, a high flow is depicted. The basal ganglia and thalamus are visualized, together with the infolded cortical layers of the Sylvian fissures, as a broad band of high-flow gray matter crossing the slice centered 5 cm above the orbitomeatal line.

* Tomomatic 64 manufactured by Medimatic, Inc., Copenhagen, Denmark.
TABLE 1

CBF measurements and clinical course in seven patients with delayed onset of symptoms

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>Location of Aneurysm</th>
<th>CBF (ml/100 gm/min)</th>
<th>CO₂ (%)</th>
<th>Day</th>
<th>Clinical Course</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F, 44</td>
<td>ACoA</td>
<td>lt 61, rt 59</td>
<td>4.8</td>
<td>2</td>
<td>Grade I; normal flow pattern</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 62, rt 48</td>
<td>5.0</td>
<td>5</td>
<td>uncomplicated rt craniotomy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 62, rt 58</td>
<td>5.0</td>
<td>9</td>
<td>onset of confusion, somnolent, lt hemiparesis; flow defect lt ACA, rt ACA, rt MCA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 62, rt 59</td>
<td>4.6</td>
<td>12</td>
<td>somnolent, lt hemiparesis; flow defect unchanged</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 52, rt 37</td>
<td>4.6</td>
<td>13</td>
<td>rt carotid arteriogram; severe spasm rt ICA, MCA, ACA</td>
</tr>
<tr>
<td>2</td>
<td>F, 51</td>
<td>none identified</td>
<td>lt 66, rt 64</td>
<td>5.1</td>
<td>3</td>
<td>confused &amp; somnolent; normal flow pattern</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 33, rt 41</td>
<td>4.4</td>
<td>9</td>
<td>onset stupor, rt hemiparesis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 33, rt 41</td>
<td>4.4</td>
<td>10</td>
<td>stuporous, lt 3rd nerve palsy; flow defect lt MCA</td>
</tr>
<tr>
<td>3</td>
<td>F, 39</td>
<td>lt MCA</td>
<td>lt 66, rt 61</td>
<td>4.2</td>
<td>4</td>
<td>Grade I; subdural Sylvian flow asymmetry</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 52, rt 58</td>
<td>4.7</td>
<td>5</td>
<td>uncomplicated lt craniotomy, awake postop</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 55, rt 61</td>
<td>4.0</td>
<td>6</td>
<td>disoriented</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 59, rt 58</td>
<td>4.2</td>
<td>10</td>
<td>onset of rt hemiparesis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 52, rt 58</td>
<td>4.7</td>
<td>11</td>
<td>flow defect lt MCA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 55, rt 61</td>
<td>4.0</td>
<td>12</td>
<td>aphasic, hemiparesis resolved; flow defect unchanged. Bilat carotid arteriogram: severe spasm lt ICA, rt MCA; moderate spasm lt MCA, lt ACA, rt ICA, rt ACA</td>
</tr>
<tr>
<td>4</td>
<td>F, 60</td>
<td>none identified</td>
<td>lt 62, rt 56</td>
<td>5.4</td>
<td>4</td>
<td>Grade I; nonfocal hemispheric flow asymmetry. Bilat carotid arteriogram: severe spasm rt MCA, rt ACA, lt ACA; moderate spasm rt ICA, lt ICA, lt MCA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 48, rt 44</td>
<td>4.0</td>
<td>11</td>
<td>onset of somnolence, lt hemiparesis (leg weaker than arm)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 57, rt 55</td>
<td>5.2</td>
<td>13</td>
<td>lt hemiparesis unchanged; flow defect rt ACA</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 78, rt 75</td>
<td>5.2</td>
<td>14</td>
<td>awake, lt hemiparesis improved (ambulatory); flow defect resolved</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 66, rt 71</td>
<td>5.0</td>
<td>15</td>
<td>mild aphasia; lt MCA flow defect resolved; flow asymmetry in region of brain retraction</td>
</tr>
<tr>
<td>5</td>
<td>F, 49</td>
<td>basilar</td>
<td>lt 51, rt 49</td>
<td>4.0</td>
<td>5</td>
<td>intermittently confused; normal flow pattern</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 54, rt 48</td>
<td>4.6</td>
<td>9</td>
<td>uncomplicated rt craniotomy; intermittent confusion persistent postop</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 50, rt 49</td>
<td>3.6</td>
<td>12</td>
<td>rt MCA flow defect</td>
</tr>
<tr>
<td>6</td>
<td>F, 59</td>
<td>rt MCA</td>
<td>lt 51, rt 49</td>
<td>4.0</td>
<td>11</td>
<td>intermittently confused; normal flow pattern</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 54, rt 48</td>
<td>4.6</td>
<td>12</td>
<td>rt MCA flow defect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 50, rt 49</td>
<td>3.6</td>
<td>13</td>
<td>flow defect resolved</td>
</tr>
<tr>
<td>7</td>
<td>F, 62</td>
<td>rt ICA</td>
<td>lt 53, rt 53</td>
<td>4.2</td>
<td>4</td>
<td>confused; normal flow pattern</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 75, rt 68</td>
<td>5.4</td>
<td>9</td>
<td>onset of somnolence, lt hemiparesis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 53, rt 53</td>
<td>4.2</td>
<td>10</td>
<td>(pton stupor, rt hemiparesis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>lt 75, rt 68</td>
<td>5.4</td>
<td>11</td>
<td>awake, confused, hemiparesis resolved</td>
</tr>
</tbody>
</table>

* Cerebral blood flow (CBF): mean of all pixels in left or right hemisphere in the slice centered 5 cm above the orbitomeatal line. CO₂: percent CO₂ in end-expiratory air. Days are post subarachnoid hemorrhage (SAH). Day of SAH is designated as Day 1. ACoA = anterior communicating artery; ACA = anterior cerebral artery; MCA = middle cerebral artery; ICA = internal carotid artery. See Fig. 2 for regional CBF patterns in these seven patients.

state with closed eyes. Expired pCO₂ was measured with an infrared analyzer three times during each study, and auscultatory blood pressure was measured at the end of each study. Each flow map was visually evaluated for side-to-side asymmetry and differences from the normal pattern of CBF distribution, and compared with previous flow maps from the same patient. The CBF was calculated for both hemispheres in the slice centered 5 cm above the orbitomeatal line. Flow values were not corrected for changes in pCO₂.

Results

Delayed Regional CBF Changes

Five patients with normal CBF tomograms on admission developed delayed neurological deficits in the 2nd week after hemorrhage (Table 1), at which time repeat CBF tomograms revealed large but well defined regional flow decreases in the vascular territories of the anterior or middle cerebral arteries in the four so studied (Cases 1 to 4, Fig. 2). The fifth patient (Case 7) developed a less obvious relative CBF decrease in the right Sylvian region on Day 4 after SAH. She became confused and developed a left hemiparesis on Day 9, but it was not possible to obtain later CBF studies. Arteriograms were performed during the 2nd week after hemorrhage in Cases 1, 3, and 4, revealing severe vasospasm in all three. Substantial functional recovery in two patients was accompanied by normalization of the CBF image (Cases 3 and 4). No neurological improvement was noted in the other two patients with repeat CBF
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Fig. 2. Regional cerebral blood flow patterns in seven patients with delayed onset of symptoms. For actual flows and clinical course see Table 1.
tomograms: one died and was proven at autopsy to have a cerebral infarction (Case 2), and the other survived with a dense hemiplegia and a large hypodense area on CT scanning (Case 1). Follow-up CBF studies in the latter case revealed persistence of the low-flow area.

One patient (Case 5) experienced the delayed onset of somnolence and confusion, and a regional CBF decrease was noted in the territory of the left MCA on Day 9 after SAH (Fig. 2). She rebled massively the following day and died without developing focal neurological findings. An arteriogram on Day 6 after SAH had revealed mild vasospasm. Another patient (Case 6), who was intermittently confused and somnolent throughout a long hospital course, was noted to have a regional CBF decrease in the distribution of the right MCA after clipping of a right MCA aneurysm (Fig. 2). The patient did not develop a focal neurological deficit, a CT scan revealed no low-density area, and the flow defect partially resolved over the course of the next 2 weeks. This was the only patient to develop such a flow decrease without an associated change in neurological status.

Regional CBF Changes Directly Related to Surgery

Of the 11 Grade I and II patients who did not develop delayed neurological deficits, eight had completely uncomplicated craniotomies for aneurysm clipping. Postoperatively, a flow asymmetry of varying degrees could be noted in these eight patients when the cortical region at the craniotomy site was compared to the contralateral side (Fig. 3). These flow decreases, which tended to persist in those patients having repeated postoperative studies, were smaller and more superficially located than the regional CBF decreases described above. No other deviation from the normal pattern of flow distribution was noted in these 11 Grade I and II patients. Similar flow changes were noted postoperatively in Cases 3 and 6 after larger regional flow decreases resolved, and postoperatively in the two Grade III patients who did not develop delayed regional flow decreases.

Hemispheric CBF Changes

On admission, 15 patients were in Grades I and II, and five patients in Grade III. Hemispheric CBF values for neither group were significantly different from those of the normal subjects (Table 2). Of the 15 patients initially in Grades I and II, four deteriorated neurologically to Grade III. A total of 36 CBF measurements were performed in patients in Grades I and II, and a total of 29 CBF measurements were performed in Grade III patients. The CBF values calculated from both hemispheres in these two groups averaged 60 ± 8 and 53 ± 7 ml/100 gm/min, respectively. Due to the inclusion of both hemispheres and studies of patients with only mild degrees of somnolence and confusion, this latter figure does not accurately reflect the lower hemispheric CBF values seen in Grade III patients. In the four patients in whom CBF measurements were obtained at the time of delayed neurological deterioration (Cases 1 to 4), the average hemispheric CBF ipsilateral to the regional CBF decrease was 42 ± 8 ml/100 gm/min.

Two patients remained in Grade III throughout prolonged periods of hospitalization but did not develop delayed neurological deficits or regional CBF decreases of the type depicted in Fig. 2. The lowest hemispheric CBF values seen in these two patients were 44 and 43 ml/100 gm/min, respectively. Both CBF and mental status improved in one of these patients after a ventriculoatrial shunting procedure.

Discussion

Noninvasive methods of CBF measurement using inhaled or intravenously administered 133Xe require that the arterial input of the isotope, which is required for all flow calculations, be indirectly determined from the pulmonary or expired air 133Xe concentration. The potential inaccuracy of this estimation is the major source of error in the quantitative measurement of CBF with the present technique. Presumably on the basis of pulmonary dysfunction, it may underlie the surprisingly high CBF values measured in some individuals, although criteria for their identification have not been developed. Because errors in estimating the true arterial input function affect flow calculations for all pixels equally, they do not affect relative flow differences measured between bilaterally symmetrical brain regions. The major strength of the present technique is that these relative flow differences can be more accurately localized and more reliably measured than with stationary detector systems, in part because tomography circumvents errors introduced by the superimposition of tissues.

In the present series, small areas of decreased regional CBF were noted postoperatively at the craniotomy site. Similarly located postoperative flow changes have been reported previously and presumably reflect the sequelae of brain retraction. A subclinical but permanent decrease in the number of functioning neurons at that site would explain the persistent changes noted on later CBF measurements.

The most striking finding in the present series was the delayed appearance of CBF decreases confined to

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Cases</th>
<th>CBF (ml/100 gm/min)</th>
<th>CO₂ (%)</th>
<th>Day After SAH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grades I &amp; II</td>
<td>15</td>
<td>63 ± 9</td>
<td>4.7 ± 0.6</td>
<td>5 ± 3</td>
</tr>
<tr>
<td>Grades III</td>
<td>5</td>
<td>58 ± 7</td>
<td>4.5 ± 0.4</td>
<td>5 ± 3</td>
</tr>
<tr>
<td>normal individuals</td>
<td>10</td>
<td>56 ± 5</td>
<td>4.8 ± 0.3</td>
<td>—</td>
</tr>
</tbody>
</table>

* Cerebral blood flow (CBF): mean of all pixels in both hemispheres in the slice centered 5 cm above the orbitomeatal line. CO₂: percent CO₂ in end-expiratory air. The day of the subarachnoid hemorrhage (SAH) is designated Day 1.
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brain regions perfused by major arterial trunks. Arterial vasospasm best explains both the delayed onset and the location of the regional CBF decreases noted in Cases 1 to 6. Arteriography was not performed specifically to evaluate these CBF decreases, but severe vasospasm was noted in the three patients undergoing arteriography in the 2nd week after hemorrhage (Cases 1, 3, and 4). Both patients studied at the onset of focal neurological deficits (Cases 1 and 3) had such a regional CBF decrease, while the CBF values outside this area were unchanged from presymptomatic levels. The regional CBF decreases observed may well explain the delayed neurological deficits that developed. In three patients (Cases 1, 3, and 5), CBF values outside the area of decreased regional CBF were unchanged from presymptomatic levels at the time of the onset of somnolence and confusion. The present study does not disclose the etiology of these nonfocal symptoms, but these three cases reveal that they are not necessarily caused by diffuse cerebral ischemia.

In some patients (Cases 1, 2, and 4) measurements made later in the course of delayed neurological deficits revealed a diffuse decrease of CBF in both hemispheres, in addition to a more pronounced flow decrease in a well defined region. The vasospasm observed in the present series, while often diffuse in extent was not uniform in severity, and thus seems unlikely to explain such a symmetrical CBF reduction. Previous investigators have noted that such global flow decreases correlate poorly with the presence or distribution of arteriographic vasospasm.3,12 These global flow decreases are therefore more likely related to the secondary effects of focal brain ischemia, as has been reported in patients with acute ischemic infarction due to atherosclerotic cerebrovascular disease.1,4,20,22,24 These results suggest that the only CBF decreases directly produced by vasospasm are those seen in major arterial distributions.

Under normal circumstances, the arteries traversing the basal subarachnoid cisterns contribute little to cerebrovascular resistance, so that arterial pressure measured in the cortical branches of the MCA closely approximates that measured in the internal carotid artery in the neck.2 In the presence of vasospasm, however, the difference between mean arterial blood pressure, measured extracranially, and intracranial pressure no longer represents the true perfusion pressure of the cerebral cortex. In this setting, different brain regions must have different perfusion pressures, determined by the length and diameter of spastic segments in the vessels supplying them and the efficiency of local collaterals.

In the absence of collateral flow, any acute decrease in the caliber of a cerebral artery produces some decrease in perfusion pressure distal to it. If a cerebral artery is narrowed in the subarachnoid space, the maintenance of a normal regional CBF requires that intraparenchymal arterioles distal to the spastic vessel dilate to decrease their resistance. When this arteriolar vasodilation becomes maximal, further decreases in regional perfusion pressure will produce decreases in regional CBF. Because the brain is not marginally supplied with blood at rest, mild CBF decreases may be well tolerated, but since the arteriolar vasodilatory reserve of the region has been exhausted, further decreases in regional perfusion pressure need only be slight to produce ischemic neuronal dysfunction. This range of measurable but asymptomatic CBF decreases may be relatively narrow, perhaps explaining why the present series reported only one case (Case 6) in which the delayed appearance of a regional CBF decrease was not associated with a change in neurological status.

Arteriographic criteria for identifying hemodynamically significant vasospasm, based primarily on a 50% or greater decrease of the diameter of an artery, have revealed an association between severe vasospasm and delayed neurological deterioration in large series.9,26 but these criteria have little predictive value in the individual case. To date, CBF measurements have not been able to predict preoperatively which patients are destined to develop delayed ischemia. A single CBF measurement, if normal, is of no help in detecting the presence of vasospasm or evaluating its effect on re-

![Fig. 3. Case 10. Cerebral blood flow patterns 8 days (left) and 3 months (right) postoperatively in a 31-year-old woman who was neurologically intact following the rupture of a left internal carotid artery aneurysm (see Fig. 1). This patient underwent an uncomplicated left frontotemporal craniotomy for aneurysm clipping 11 days after subarachnoid hemorrhage.](image-url)
gional perfusion pressures. On the basis of studies with intracarotid $^{133}$Xe, it has been suggested that patients with regional CBF decreases during graded hypotension are unable to safely tolerate intracranial aneurysm surgery. The presence of areas of significantly compromised perfusion pressure, manifest as areas with regional CBF decreases, could be noninvasively revealed with the present technique by repeating CBF measurements while cautiously lowering mean arterial blood pressure.

Because the complex regional changes in arterial resistance produced by vasospasm cannot be measured arteriographically, many questions remain about the impact of surgery on the development of delayed ischemia due to vasospasm, and about the efficacy of regimens currently proposed to improve tissue perfusion in its presence. Noninvasive tomographic measurements of regional CBF have the potential to answer these questions.

References


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