Clinical Grade, Regional Cerebral Blood Flow and Angiographical Spasm in the Monkey After Subarachnoid and Subdural Hemorrhage

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Abstract:
Subarachnoid and subdural hemorrhage has been induced in the anesthetized monkey by injection of fresh autogenous blood via a needle inserted through a subfrontal twist drill hole. Serial angiographical studies and regional cerebral blood flow (rCBF) determinations were carried out concurrently for three hours following hemorrhage. The changes observed were subsequently correlated with the clinical state of the animals upon reversal of anesthesia.

In general, angiographical vasospasm and a reduction in CBF occurred simultaneously. Occasionally, however, cerebral perfusion remained unaltered after SAH even though marked vasoconstriction of the intradural vessels was present. An excellent correlation between the severity of reduced perfusion and the degree of neurological deficit was found.

Additional Key Words
post-SAH arrhythmias
133Xenon intra-arterial injection method neurological assessment

Introduction
The pathophysiology of post-subarachnoid hemorrhage vasospasm and its influence on cerebral perfusion and function remains unsettled. While there is general agreement that global depression of cerebral blood flow occurs after significant subarachnoid hemorrhage (SAH), the pathogenesis of reduced perfusion has not been fully elucidated.

We have previously investigated vasospasm after repeated subarachnoid hemorrhages in the monkey and failed to find a correlation between vasospasm present within one hour after SAH and the morbidity and mortality of the animals.1 In a subsequent investigation,2 using the 133Xenon intraarterial injection method for measuring cerebral blood flow (CBF), we found a correlation between the degree of impairment of perfusion and the severity of neurological deficit in the monkey.

In an attempt to determine whether reduced perfusion is the result of vasospasm, a study was designed to concurrently investigate changes in angiographical arterial caliber and regional cerebral blood flow (rCBF) following induced SAH. The changes observed were subsequently compared to the clinical and neurological state of the animals following termination of each experiment.

Methods
Twenty-one adult female Rhesus monkeys weighing 2.3 to 3.2 kg were utilized. Sedation which facilitated introduction of flexometallic endotracheal tubes was achieved by intravenous sodium pentobarbital (20 to 30 mg/kg). Anesthesia was maintained with nitrous oxide and oxygen from a reservoir in a ratio of 2:1. The animals were curarized and artificially ventilated with a Harvard variable phase mechanical respirator. Esophageal temperature was kept near 37.5°C by an infrared light bulb positioned above the animal.

Blood pressure was continuously measured through a catheter positioned in the femoral artery. Arterial blood samples were frequently taken for blood gas and pH analysis. Hemoglobin and hematocrit studies required for cerebral blood flow calculation were performed at the onset, midpoint, and upon termination of each experiment. Electrical activity of the myocardium was continuously monitored and in several animals the electrical activity of the brain was recorded through eight disk electrodes on a Grass Model 6 encephalograph. Intracranial pressure was continuously monitored in several animals using a Numoto pressure...
switch implanted extradurally through a burr hole in the medial parietal area.

In each monkey neck surgery was performed using an operating microscope. After the external carotid artery was doubly ligated a fine cannula was introduced via the lingual artery into the internal carotid artery. The catheter was connected to a three way stop cock, through which heparinized saline was slowly infused (10 ml per hour).

Serial angiography was performed in all monkeys by forceful injection of 1 to 1.5 ml meglumine iothalamate (Conray 60) into the lingual catheter. Only lateral angiograms were obtained and care was taken to maintain magnification factors constant in each experiment. In the control series angiograms were performed on completion of surgery and prior to termination of the experiments. In the monkeys subjected to hemorrhage, baseline angiograms were carried out after completion of surgery and upon insertion of the needle into the chiasmatic area. Post-hemorrhage angiographical studies were performed at 20, 90 and 180 minutes.

The films showing the arterial phase most distinctly on each angiogram were selected and the caliber of the cerebral arteries at predetermined fixed locations were measured using a calibrated lens system.

Regional cerebral blood flow (rCBF) was measured by the intra-arterial technique described by Ingvar, Lassen and Hoedt-Rasmussen.4-7 133Xenon (3.0 to 3.5 mCi) dissolved in 0.25 to 0.5 ml sterile saline was injected over 2 to 3 seconds into the lingual artery. Ipsilateral extracerebral contamination with 133Xenon was minimized by ligation of the external carotid artery.

Regional clearance rates for 133Xenon (rCBF) were measured using a six-detector scintillation counter assembly constructed in our laboratory. The detector system was designed to record the radioactivity in each of five discrete cylindrical volumes of ipsilateral brain tissue, with the sixth detector recording radioactivity in the ipsilateral orbital tissues. Each scintillation counter comprised of one-fourth-inch diameter, one-half-inch thick NaI(Tl) crystal coupled to a one-half-inch diameter photomultiplier (XP 101) with a truncated plexiglass light guide. The detectors were mounted in a stainless steel collimator block with the front face of each crystal recessed three inches from the block face. An additional one-half-inch lead collimator applied to the block face was used in the present study.

Pulses from each detector were amplified and then input to single-channel analyzers which accepted pulses equivalent to the energy range 70 to 90 kev. The output from each single-channel analyzer was recorded in a high speed buffered multiplexor, in a 400-word magnetic core memory. Upon termination of each ten-minute experimental period, the information was punched out on paper tape and was processed by computer. Compartmental, stochastic (height over area) and initial-slope-index methods were used for calculation of regional cerebral blood flow (rCBF). Theoretical considerations have been previously published.5

With each rCBF measurement, PaCO2, PaO2 and pH were determined and incorporated into the computer program. Both corrected and uncorrected flow values for PaO2 were calculated. Particular care, especially in the post-hemorrhage period, was taken to keep PaCO2 levels near 40 mm Hg.

Subarachnoid hemorrhage was simulated by the method described by Weir et al.1 In summary, 4 ml of fresh autogenous blood was injected over a 20-second period through a circumferentially beveled needle positioned (under fluoroscopy) in the chiasmatic cistern. Return of clear CSF ensured correct needle placement. Subdural hemorrhage was achieved by the same method except blood was injected into the prechiasmatic subdural space.

Six monkeys were included in the control series and had repeated rCBF studies performed at 30-minute intervals for a five to six-hour period. Fifteen animals were subjected to intracranial hemorrhage. Subarachnoid hemorrhage was induced in eight, pure subdural hemorrhage in two and combined SDH:SAH in five other monkeys. Usually three to four baseline rCBF determinations at 30-minute intervals were made prior to needle insertion into the subarachnoid or subdural space. After correct needle placement, angiography was

![Lateral angiogram showing scintillation detector placement. Detectors 1, 2, 3, and 5 measure CBF from the frontal, central, parietal and temporal areas of the brain (regional hemispheric CBF). Detector 6 monitors cerebellar perfusion, while detector 4 monitors orbital tissue perfusion.](https://stroke.ahajournals.org/doi/figure/10.1161/01.STR.4.2.300)
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carried out and was followed by a final baseline rCBF. Insertion of the needle into the subarachnoid or subdural spaces did not cause alteration in rCBF or vessel caliber. Post-hemorrhage rCBF studies were started within three minutes of the hemorrhage and continued at 30-minute to 45-minute intervals for a period of three hours. In one study, rCBF and angiography was performed at 24 hours post-hemorrhage.

Following the three-hour post-hemorrhage period anesthesia was discontinued and the animals reversed with atropine and neostigmine. The animals were observed over the next one to two hours and their clinical and neurological state was evaluated. The type, degree and location of hemorrhage was determined by gross pathological examination.

Neurological Assessment

Neurological examination was performed in all monkeys in the one to two-hour period after anesthesia was discontinued. A five-division neurological grading system was utilized for evaluation of the animals.

Grade 1: no evidence of neurological deficit; alert, active and vocal ("croaking"); accept food and water.

Grade 2: mildly obtunded; not as active or vocal, no significant neurological deficit.

Grade 3: moderately obtundated; neurological deficit, i.e., hemiparesis, cranial nerve palsy. Usually assume semi-supine position but will sit up when stimulated. Respond to all forms of stimulation (auditory, touch, pain).

Grade 4: severely obtunded; severe neurological deficit (i.e., hemiplegia, quadriplegia); little or no response to painful stimulation. Frequently exhibit generalized intermittent chronic seizures of variable duration.

Grade 5: moribund; unresponsive to all forms of stimulation; failing vital signs (falling BP, arrhythmias, shallow irregular respirations).

Results

CONTROL SERIES
Cardiovascular Responses

The arterial blood pressure in the six control monkeys averaged 113 ± 9.8 mm Hg while the mean heart rate was 206 ± 19.2 beats per minute. Cardiac rhythm abnormalities were not seen in the entire series. Hemoglobin and hematocrit determinations made at the onset, midpoint, and prior to termination of each experiment averaged 12.9 ± 0.5 gm %, 37.3 ± 1.9 vol %; 11.7 ± 0.9 gm %, 34.0 ± 2.2 vol %; and 10.6 ± 0.9 gm %, 30.8 ± 2.1 vol %, respectively. Arterial P_{CO_2}, P_{O_2} and pH values averaged 39.1 ± 3.7 mm Hg, 113.2 ± 16.9 mm Hg, and 7.32 ± 0.4, respectively.

Regional Cerebral Blood Flow (rCBF)

Reproducibility of the method employed to measure CBF was examined in a monkey by performing flow determinations in the same animal on two different occasions one week apart. CBF values determined by the compartmental, stochastic and initial-slope-index methods averaged 52 ± 3.5, 43 ± 2.8, and 43 ± 3.4 ml/100 gm per minute in the first experiment compared to values of 53 ± 1.6, 44 ± 3.6, and 48 ± 5.1 ml/100 gm per minute on the second study.

In six control monkeys, rCBF determined at intervals of 30 to 45 minutes for a period of five to six hours, no significant variation in cerebral perfusion occurred throughout this time period (fig. 2). The mean compartmental, stochastic and initial-slope-index rCBF values and standard deviations are

![Figure 2](https://stroke.ahajournals.org/)

Mean rCBF values (ml/100 gm per minute) in six control monkeys measured over a five-hour period. Hemispheric values (detectors 1, 2, 3, and 5) show little interregional variation. Mean cerebellar flow (detector 6) is approximately 25% below and mean orbital tissue perfusion (detector 4) is 50% lower than mean hemispheric values. Stochastic (height/area) values are used.
shown in figure 3. Flow values recorded from the frontal, central, parietal and temporal areas were not significantly different from each other. Mean rCBF values in the cerebellum (detector 6) was 25% lower than the combined means of flows in the cerebral hemisphere (detectors 1, 2, 3 and 5), while orbital tissue flow (detector 4) was reduced by approximately 50%.

The mean CBF measured by the contralateral single detector system was not significantly different from the mean sum of the ipsilateral multidetector system (detectors 1, 2, 3 and 5). The small variations present were attributable to extracerebral contamination and counting geometry differences.

Angiography
Lateral angiograms performed at the onset and upon termination of each experiment did not reveal significant change in vessel caliber.

Neurological Assessment
Using the five-division grading system outlined above, all control animals were classified as Grade 1, and were utilized for subsequent experiments.

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Cardiovascular and Intracranial Pressure Responses
The mean arterial BP prior to SAH in eight monkeys studied was 114 ± 12.3 mm Hg, while the post-SAH values averaged 117 ± 17.7 mm Hg. Injection of 4 ml autogenous blood into the basal cisterns produced systemic hypertension beginning at 5 to 10 seconds and reaching a maximal value (173 ± 14.4 mm Hg) within 30 to 45 seconds after the onset of injection. Within two to eight minutes following SAH, systemic BP returned to baseline levels.

Pre-hemorrhage heart rate (HR) averaged 209 ± 17.1 compared to 181 ± 30 beats per minute in the post-SAH period. Bradycardia was usually observed within 30 seconds after onset of injection and the minimal value averaged 121 ± 25 beats per minute. Postinjection values beyond the first five to ten minutes were not significantly different from pre-SAH values.

Cardiac arrhythmias which were first observed 25 to 45 seconds from the onset of SAH were seldom present after five minutes. The most frequent changes observed included sinus arrhythmia, nodal beats, nodal rhythm and premature ventricular contractions. Other EKG abnormalities, i.e., increased T and P wave amplitude, S-T elevation, and U waves also were common. A comprehensive discussion of EKG changes after induced SAH was published previously.

**FIGURE 3**
Mean rCBF and standard deviations (ml/100 gm per minute) determined by the compartmental (top values), stochastic (middle values) and initial-slope-index (bottom values) methods over a five-hour period in six control monkeys.
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Intracranial pressure changes during and after SAH were monitored in three animals. Pre-hemorrhage values averaged 17 ± 5.5 mm Hg compared to 34 ± 11 mm Hg in the post-SAH period. After a mean peak pressure of 140 ± 8.2 mm Hg observed at 20 to 30 seconds, ICP returned to near baseline values within five minutes. Following this initial rapid decrease, ICP values gradually increased during the subsequent three-hour period, perhaps secondary to ensuing cerebral edema.

Mean pre-hemorrhage PaCO_2_, Pao_2_and pH values were 38.9 ± 3.3 mm Hg, 110 ± 13.5 mm Hg, and 7.37 ± 0.1, respectively. Post-SAH values averaged 39.0 ± 3.0 mm Hg, 110 ± 9.5 mm Hg, and 7.37 ± 0.1, respectively. Hemoglobin and hematocrit values at onset of the experiments averaged 12.5 ± 1.5 gm %, 37.0 ± 5.2 vol %; prior to SAH 11.5 ± 1.7 gm %, 33.4 ± 5.2 vol %, and upon termination 10.5 ± 1.8 gm % and 30.3 ± 4.9 vol %.

Regional Cerebral Blood Flow (rCBF)

Of the eight monkeys subjected to SAH, six (75%) showed a significant reduction in rCBF (fig. 4). The decrease was present in all brain regions viewed by the five-ipsilateral detectors. Orbital tissue perfusion (detector 4) was not significantly affected by SAH. The reduction in rCBF was immediate (first rCBF at three minutes post-SAH) and flow values remained significantly reduced for the three-hour duration. Little variation in rCBF occurred after reduced perfusion was established. Contralateral hemispheric perfusion measured by the single detector system also was significantly decreased. Mean pre-SAH and post-SAH rCBF values with standard deviations calculated by the compartmental, stochastic and initial-slope-index methods are represented in figure 5. One animal subjected to rCBF studies 24 hours after SAH continued to show a significant global reduction in cerebral perfusion.

Two monkeys (25%) with profuse SAH did not develop a decrease in rCBF in the three-hour period following injection (fig. 6).

Angiography

Baseline lateral angiograms were performed immediately following lingual artery catheterization and after needle insertion into the SAH. Needle placement did not produce alteration in vessel diameter (t = 0.412-1.128). Post-hemorrhage angiography was carried out immediately after measurement of the first post-SAH rCBF (i.e., at 20 minutes post-SAH), 1.5 hours and three hours post-hemorrhage. In one study angiography was carried out 24 hours later.

FIGURE 4

Decrease in rCBF (ml/100 gm per minute) in six of eight monkeys subjected to SAH. Hemispheric and cerebellar perfusion decreased after SAH, whereas orbital tissue flow remained constant. Stochastic (H/A) values were used.
Mean pre-SAH and post-SAH compartmental, stochastic (H/A) and initial-slope-index rCBF values (ml/100 gm per minute) in six monkeys which displayed a significant decrease in cerebral perfusion.

Significant generalized vasospasm of the intradural vessels was present in all monkeys subjected to SAH (fig. 7). The mean percentage reduction in all vessels measured at the predetermined post-SAH times was 30 ± 9.3%.

Table 1 compares the mean percentage decrease of the intradural arterial tree in monkeys which showed a significant rCBF decrease compared to the animals which did not. Monkeys displaying a reduction in rCBF exhibited a mean vessel caliber.
Lateral angiograms of a monkey which exhibited significant reduction in rCBF after SAH. Films were taken (1) prior to SAH (A), (2) 20 minutes post-SAHI (B), and (3) three hours post-SAHI (C). Diffuse intradural vasospasm is present in the post-SAHI films (B) and (C).

decrease of $35 \pm 7.3\%$ throughout the three-hour period. In the angiographical study performed at 24 hours, a $30\%$ reduction in vessel caliber was present. Vasospasm remained intense for the period of investigation in monkeys which displayed reduced cerebral perfusion. Monkeys not exhibiting a decrease in rCBF had a mean vessel caliber decrease of $26 \pm 9.8\%$. Although spasm was intense initially, there was a tendency for some vessel relaxation, although the decrease was not statistically significant (fig. 8).
Neurological Assessment

The animals with significant reduction in cerebral blood flow following SAH displayed severe neurological abnormalities. All were severely obtunded, hemiplegic or quadriplegic and responded poorly to all forms of stimuli (Grade 4). Occasionally intermittent generalized clonic seizures ensued. The animal developed failing vital signs during the period of examination and died (Grade 5).

Monkeys exhibiting vasospasm but no reduction in cerebral perfusion after SAH appeared mildly obtunded but did not display a neurological deficit. They were classified as Grade 2 clinically.

Subdural Hemorrhage Series

Seven monkeys, grouped into three categories by virtue of the amount of blood present in the subdural space on postmortem examination, comprised the
subdural hemorrhage series. Two animals displaying only subdural hemorrhage were classified as Group A; three showed primarily SDH with a minute amount of blood in the subarachnoid space—Group B; and two animals presented with a 20% to 30% SAH component—Group C.

Cardiovascular and Intracranial Pressure Responses

There were no significant differences in cardiovascular and intracranial pressure responses between the three groups of monkeys studied. The mean arterial BP prior to induction of SDH was 108 ± 19.0 mm Hg, while the post-SDH values averaged 118 ± 24.0 mm Hg. Subdural hemorrhage produced a systemic hypertension beginning at about ten seconds and reached a mean peak value (169 ± 25.0 mm Hg) within 60 seconds from onset of injection. Post-
hemorrhage values generally reverted to baseline values in five to ten minutes.

The mean baseline heart rate (HR) of 197 ± 30.5 beats per minute was not significantly different from mean post-hemorrhage value (178 ± 39.4 beats per minute). Decrease in HR was observed at 5 to 15 seconds after onset of injection and the mean minimal value (114 ± 46.0 beats per minute) was reached at 20 to 30 seconds. Post-hemorrhage HR beyond 25 minutes was not significantly different from baseline values.

Arrhythmias were frequently observed in this series and usually began 20 to 30 seconds after SDH. The EKG changes were similar to ones observed after SAH.

Pre-hemorrhage intracranial pressure averaged 20 ± 8.0 mm Hg compared to 45 ± 20.0 mm Hg in the three-hour period after SDH. The mean peak ICP observed at 30 to 50 seconds post-injection was 150 ± 24.0 mm Hg. Unlike the SAH series where ICP usually reverted to baseline levels within five minutes, the pressure gradually decreased but did not reach baseline levels in the entire three-hour period.

Pre-SDH PaO₂, PaCO₂, and pH values were 40.1 ± 3.9 mm Hg, 109 ± 13.4 mm Hg, and 7.35 ± 0.04, compared to post-SDH values of 39.6 ± 4.5 mm Hg, 110 ± 11.1 mm Hg, and 7.35 ± 0.06, respectively.

Regional Cerebral Blood Flow (rCBF)

**Group A (SDH):** Monkeys exhibiting subdural hemorrhage without a subarachnoid component did not have a reduction in CBF over the three-hour period after SDH. Mean pre-hemorrhage stochastic values were 77 ± 20.1 compared to 85 ± 24.8 ml/100 gm per minute in the post-SDH period.

**Group B (SDH with a small SAH component):** These animals showed a small nonsignificant reduction to CBF in the post-hemorrhage period. Mean pre-hemorrhage and post-hemorrhage values were 82 ± 26.1 and 70 ± 24.2 ml/100 gm per minute, respectively (t = 1.367).

**Group C (SDH with a 20% to 30% SAH component):** In animals which displayed a 20% to 30% subarachnoid component a significant reduction in cerebral perfusion occurred (t = 2.796, P < 0.02).

**Angiography**

**Group A Monkeys**

Reduced vessel caliber was observed only in arteries in the immediate vicinity of the basal subdural hemorrhage (intracavernous internal carotid and the internal carotid artery at the origin of the ophthalmic artery). There was no evidence of caliber reduction in the intradural portion of the arterial tree.
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FIGURE 8
Lateral angiograms taken (1) prior to SAH (A), (2) 20 minutes post-SAH (B), and (3) three hours post-SAH (C). Although no decrease in rCBF occurred after SAH, diffuse intradural vasospasm was exhibited in the post-SAH films (B) and (C).

Group B Monkeys
Decrease in arterial vessel caliber was present primarily in the intracavernous internal carotid artery, internal carotid at the ophthalmic origin and, to a lesser extent, the proximal segment of the middle cerebral and intradural internal carotid arteries. No significant reduction in the proximal and distal pericallosal artery was evident.
Group C Monkeys

The intradural arteries measured in animals which displayed a 20% to 30% subarachnoid component showed a significant reduction in vessel caliber of 20% to 25%. Unlike Groups A and B monkeys, no significant reduction in extradural vessel caliber occurred.

Neurological Assessment

Monkeys subjected to subdural hemorrhage with or without a small subarachnoid component (Groups A and B) appeared mildly obtunded but did not exhibit significant neurological deficit and were classified as Grade 2.

Monkeys with a 20% to 30% subarachnoid...
component were moderately obtunded and displayed significant neurological abnormalities including hemiparesis, depressed response to stimuli, and cranial nerve palsy. They were classified as Grade 3 (table 2).

**ANGIOGRAPHICAL-CBF-NEUROLOGICAL CORRELATION**

Subarachnoid hemorrhage induced by injection of autogenous blood into the basal subarachnoid space produced generalized sustained constriction of the intradural arterial tree. Although significant arterial vasospasm was observed in all animals subjected to SAH, reduction in cerebral perfusion occurred in 75%. These animals exhibited severe post-hemorrhage neurological abnormalities and a correlation between vasospasm, reduced cerebral perfusion, and neurological state was demonstrated. However, in 25% of cases, intense vasoconstriction did not
produce a reduction in cerebral perfusion nor result in neurological deficit. Reasons for the discrepancy between vasospasm and cerebral perfusion (or neurological function) are not explainable in the present study. Perhaps the resistance vessels (distal arterioles and capillaries) of a size beyond angiographical resolution were not compromised and cerebral perfusion and function remained unaltered. Presence of excellent correlation between cerebral blood flow (rCBF) and the neurological state of the animals suggested that perfusion studies are more sensitive than the degree of vessel caliber constriction with respect to cerebral function and survival after subarachnoid hemorrhage.

Discussion

Although recent studies2, 0, 10 have demonstrated a global reduction in cerebral blood flow following subarachnoid hemorrhage, the cause of decreased perfusion is not fully resolved. Kagstrom et al.13 showed a 20% reduction in hemispheric CBF in seven patients examined during the first few weeks after SAH. A three-month follow-up study revealed a correlation between hemispheric flows and the severity of neurological sequelae. James12 found reduced cerebral perfusion in 36 patients suffering from recent SAH. The reduction in CBF correlated with the impairment of consciousness and the radiological appearance of spasm of large cerebral arteries. However, reduced CBF was frequently bilateral, whereas vasospasm was greater on the side of the arterial lesion. Zingesser et al.12 were unable to obtain a correlation between rCBF and angiographical vasospasm in 19 patients with SAH. They found a reduction in CBF even in the absence of arterial constriction, and vasospasm, when present, was not necessarily associated with reduced cerebral perfusion in the vascular territory distal to the spasm. Ferguson et al.14 studied 22 patients with SAH and found an excellent correlation between the neurological state and CBF. However, in comparing patients with vasospasm (eight cases) to those without vasospasm, no significant difference in CBF was detectable. They concluded that the significance of arterial vasospasm was debatable, although severe spasm was usually associated with a marked decrease in CBF. In a study of 17 patients with SAH, Symon et al.15 found a correlation between CBF and the clinical condition of the patients. Although they concluded that a good correlation between angiographical abnormalities (spasm, partial occlusion as a complication of surgery, areas of apparent slowed flow) and rCBF was present, only in two of four cases was there a correlation between vasospasm and cerebral blood flow. Heilbrun et al.16 performed preoperative and postoperative 133Xe rCBF studies in 14 patients suffering from SAH. All patients demonstrated a global reduction in CBF. Of five patients studied in the preoperative period, three demonstrated focal ischemia without evidence of arterial spasm. Seven were studied in the postoperative period. Four patients demonstrated spasm and reduced rCBF in the distribution of the spastic arteries. However, in comparing patients with severe and mild spasm, no difference in CBF was found. These authors concluded that because there was a global reduction in cerebral perfusion after SAH, this reduced CBF could not be attributable directly to arterial spasm since spasm was absent in five of six preoperative and four of eight postoperative studies. In addition, vasospasm when present was always localized.

Hashi et al.17 concurrently studies CBF and angiographical changes in baboons after induced SAH. Seven of ten baboons displayed angiographical vasospasm, but there was no significant difference in CBF in animals with or without spasm in the acute phase after SAH. At 24 to 48 hours post-SAHA vasospasm was still present, but a significant reduction in cerebral perfusion occurred. These authors concluded that reduced CBF was the result of perivascular edema and vasospasm. Yamaguchi and Waltz18 studied CBF in cats after puncture of the middle cerebral artery and found no consistent relationship between CBF and the calibers of surface cortical arteries.
The present study demonstrated that cerebral blood flow is more constantly related to the clinical grade than is the degree of arterial vasoconstriction after subarachnoid hemorrhage. Since the clinical grade of patients suffering from SAH appears to be a key factor in determining the prognosis, this relationship appears important. Whether or not cerebral perfusion studies actually give more information than a detailed neurological examination of the patient cannot be answered at the present time. However, we feel that CBF studies can provide additional information regarding pathophysiological factors responsible for reduced perfusion after SAH. Furthermore, such studies may be useful in evaluating the effectiveness of therapeutic agents designed to restore cerebral perfusion and the clinical grade of patients. Further clinical studies also will be necessary to show whether or not angiographical spasm in the presence of normal flows and a good clinical grade is still a contraindication to early surgery.

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