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CT of Subarachnoid Hemorrhage due to Ruptured Aneurysm

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Computed tomographic scans in 81 consecutive patients with subarachnoid hemorrhage due to ruptured aneurysm were analyzed for patterns of hemorrhage and lucency and correlated with the development of spasm and hydrocephalus. The circulation time was measured angiographically in representative cases of spasm. Hemorrhage corresponded in a general way to the fossa of aneurysm origin and, when there was parenchymal or ventricular hemorrhage, in more specific ways to anterior communicating, middle cerebral, and posterior inferior cerebellar artery aneurysms. Basal ganglionic hemorrhages due to aneurysm ruptures occurred in two cases and could not be distinguished by appearance from hypertensive hemorrhage. Regions of low attenuation (lucencies) were often persistent and had lateralizing value; they showed a high correlation with arterial spasm. Conversely, spasm, particularly of a distal type, showed a significant correlation with increased circulation time and the occurrence of brain lucency. In cases of multiple aneurysms arising from different vessels in which there was hemorrhage or lucency, CT scans correctly predicted the site of aneurysm in 77% of cases. Periventricular lucency was a weak predictor of progressive hydrocephalus, while an intraventricular hemorrhage was a strong predictor of moderate to severe hydrocephalus.

To determine the localizing features of hemorrhage due to ruptured aneurysms and to evaluate the significance of associated ischemic changes and hydrocephalus, CT scans and angiograms were reviewed in 81 consecutive patients with subarachnoid hemorrhage due to aneurysm.

Materials and Methods

Subarachnoid hemorrhage was established by lumbar puncture; the presence of aneurysms was demonstrated by angiography and confirmed either by surgery or autopsy. Most CT scans were performed on a first generation head scanner with water bag; some recent scans were done on a high resolution scanner. All patients were scanned in the first 5 weeks after subarachnoid hemorrhage. The circulation time was measured in those cases of spasm in which complete serial angiography was available.

For purposes of this study, the aneurysms were divided into seven groups by location (table 1). These groups were further subdivided according to whether the aneurysms were single or multiple and whether hemorrhage was present on the CT scan (table 2). Determination of which of multiple aneurysms had bled was made by a combination of radiologic and clinical means, including surgery.

Results

Hemorrhage

The earliest scan after subarachnoid hemorrhage in each case was analyzed for intracerebral, intraventricular, and subarachnoid hemorrhage, as well as for subdural hematoma. Fifty cases showed hemorrhage on the initial scan; the median time of scan was 6 days. The hemorrhages were tabulated according to

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the location of hemorrhage and the site of the bleeding aneurysm (table 3). In several cases, a single aneurysm bled into more than one site.

Spasm, Circulation Time, and Lucency

Spasm was classified according to its location with respect to the circle of Willis. Spasm proximal to or within the circle of Willis was termed "proximal," while spasm distal to the circle of Willis (with or without accompanying proximal spasm) was termed "distal." Fifty-nine cases had spasm on at least one angiogram. Forty-two had proximal spasm, and 17 had distal spasm.

The circulation time, in seconds, from the internal carotid artery siphon to a parietal vein opacification was measured in each of 22 cases of spasm. The results were tabulated according to the type of spasm (proximal or distal) on angiography and the occurrence of lucency (absence or presence) in the affected territory of the corresponding CT scan (table 4).

Definite regions of abnormal lucency were noted on the initial CT scans of 21 patients. These cases were tabulated with regard to the location of the aneurysm and the distribution of the lucency (table 5). Lucency immediately adjacent to a hematoma was considered to represent ischemic necrosis and was not tabulated. Cases with lucency were also analyzed for associated angiographic spasm, functional deficit (determined clinically and related to CT lucency), and persistence of lucency on follow-up scan (table 6). Several patients' scans had more than one region of lucency.

Multiple Aneurysms

Nineteen patients had multiple aneurysms. Each patient, however, had only one ruptured aneurysm. Surgical confirmation of the ruptured aneurysm, with evidence suggestive of the source of hemorrhage, was obtained in all cases. Cases were tabulated according to the site of hemorrhage, predicted and confirmed (table 7).

TABLE 1: **Location of Ruptured Aneurysms**

Location	No.
Anterior communicating artery (ACoA)	27
Anterior cerebral–pericallosal (ACA)	2
Posterior communicating artery (PCoA)	20
Distal internal carotid artery (ICA):	
Anterior choroidal	1
Ophthalmic	1
Supraclinoid internal carotid	1
Internal carotid bifurcation	6
Proximal anterior cerebral	1
Middle cerebral artery (MCA)	11
Basilar artery (BasA):	
Midbasilar	1
Basilar tip	5
Proximal posterior cerebral	1
Vertebral artery (VertA):	
Hypoglossal	1
Posterior inferior cerebellar	3

TABLE 3: **Distribution of Hemorrhage**

Type of Hemorrhage	ACoA	ACA	PCoA	ICA	MCA	BasA	VertA
Subdural hematoma	0	0	0	0	2	0	0
Cisternal hemorrhage:							
Pericallosal	5	2	0	0	0	0	0
Interhemispheric	15	2	5	2	3	0	2
Sylvian	9	0	5	4	4	0	1
Interpeduncular	4	0	1	0	0	1	0
Perimesencephalic	3	0	2	1	2	2	0
Intracerebral hemorrhage:							
Septal	8	0	0	0	0	0	0
Inferomedial frontal	6	0	0	2	0	0	0
Deep frontal	0	0	2	0	0	0	0
Callosal	0	2	0	0	0	0	0
Basal ganglionic	0	0	0	1	1	0	0
Lateral frontal	0	0	1	1	2	0	0
Temporal	0	0	1	0	6	0	0
Midbrain	0	0	0	0	0	2	0
Intraventricular hemorrhage:							
Lateral ventricles	6	0	1	2	1	0	0
Third ventricle	5	0	0	2	1	0	3
Fourth ventricle	4	0	0	2	1	0	3
Third and fourth ventricle only	1	0	0	0	0	0	3

Note.—Abbreviations explained in table 1.

TABLE 4: **Circulation Time and Type of Spasm**

	Proximal Spasm		Distal Spasm	
	Without Lucency	With Lucency	Without Lucency	With Lucency
No. cases	11	2	2	7
Range of circulation time (sec)	5.0–8.5	6.0–8.5	7.5–9.0	6.0–13.5
Average circulation time (sec)	6.86	7.25	8.25	8.36
Combined average circulation time (sec)	6.96		8.33	

TABLE 2: **Numbers and Types of Aneurysms, Including Visualized Hemorrhage and Time of Scan**

Type of Aneurysm and Time	No. Aneurysms (No. with Hemorrhage on CT)						
	ACoA	ACA	PCoA	ICA	MCA	BasA	VertA
Single aneurysm	22 (15)	1 (1)	15 (6)	5 (3)	9 (9)	7 (3)	3 (3)
Multiple aneurysms	5 (2)	1 (1)	5 (2)	5 (4)	2 (1)	0	1 (1)
Total	27 (17)	2 (2)	20 (8)	10 (7)	11 (10)	7 (3)	4 (4)
Median time (days) of scan after ictus	5	1	7	6	4	9	1

Note.—Abbreviations explained in table 1.

TABLE 5: **Distribution of Lucency**

Location	ACoA	ACA	PCoA	ICA	MCA	BasA	VertA
Medial frontal	9	2	0	1	0	0	0
Lateral frontal	1	0	0	2	1	0	0
Deep frontal	0	0	3	1	0	0	0
Temporal	0	0	2	0	1	0	0
Parietal	0	0	0	0	1	1	0

Note.—Abbreviations explained in table 1.

TABLE 6: **Correlation of Lucencies with Spasm and Site of Aneurysm**

	ACoA	ACA	PCoA	ICA	MCA	BasA	Total
Patients with CT lucency	11	2	3	2	2	1	21
With spasm	11	2	3	2	1	1	20
With distal spasm	5	2	3	0	1	0	11
With matching functional deficit	9	2	3	2	2	0	18
With follow-up scan	5	2	0	0	2	0	9
With persistent lucency	4	2	0	0	2	0	8

Note.—No patients with a ruptured vertebral artery aneurysm had CT lucency. Abbreviations explained in table 1.

Hydrocephalus

The lateral ventricles were evaluated for mild, moderate, or severe dilatation. Forty-seven patients had hydrocephalus of some degree on the initial scan. These cases were tabulated with regard to the site of the aneurysm and an association with periventricular lucency (table 8). Follow-up scans were analyzed for progression of hydrocephalus, and comparison was made between those with and those without periventricular lucency (table 9).

Intraventricular Hemorrhage

Fourteen patients had intraventricular hemorrhage. These were analyzed with regard to lateral and other ventricular involvement (table 3). *All* had hydrocephalus of some degree. A distribution with regard to site of aneurysm and degree of intraventricular hemorrhage was obtained (table 10). Incidence and degree of hydrocephalus were compared in cases with and without intraventricular hemorrhage (table 11).

Discussion

The distribution of aneurysm locations was roughly in agreement with that obtained in the Cooperative Study [1]. The proportion of cases with multiple aneurysms was 19/81 (23%) (table 2).

Incidence of Hemorrhage

The lowest rates of detection (11/27, about 40%) of hemorrhages with CT scanning occurred with cases of aneurysms originating near the posterior communicating

TABLE 7: **Multiple Aneurysms**

Confirmed Location of Ruptured Aneurysm/Individual Cases	Hemorrhage on CT	Lucency on CT	Site Predicted by CT
ACoA:			
1	Yes	No	Correct
2	No	Yes	Correct
3	No	No	Nonspecific
4	Yes	No	Incorrect
5	Yes	No	Correct
ACA:			
1	Yes	No	Correct
2	Yes	No	Correct
PCoA:			
1	No	No	Nonspecific
2	Yes	No	Nonspecific*
3	No	No	Nonspecific
4	Yes	No	Nonspecific
5	No	No	Nonspecific
ICA:			
1	No	No	Nonspecific
2	Yes	No	Correct
3	Yes	Yes	Correct
4	Yes	No	Correct
5	Yes	No	Nonspecific
MCA:			
1	No	Yes	Correct
2	Yes	Yes	Correct
VertA†	Yes	No	Correct

Note.—Abbreviations explained in table 1.
* Aneurysms of same vessel.
† Posterior inferior cerebellar artery.

TABLE 8: **Hydrocephalus—Distribution and Type**

	ACoA	ACA	PCoA	ICA	MCA	BasA	VertA
Total no. of cases with ruptured aneurysm at this site	27	2	20	10	11	7	4
Hydrocephalus of either type	20	1	10	4	6	3	3
Hydrocephalus with periventricular lucency	11	1	3	2	1	3	2

Note.—Abbreviations explained in table 1.

TABLE 9: **Progression of Hydrocephalus**

	Hydrocephalus with Periventricular Lucency	Hydrocephalus without Periventricular Lucency
No. patients	23	24
No. with follow-up scan	13	13
No. with progression	5 (38%)	3 (23%)

TABLE 10: **Hydrocephalus with Intraventricular Hemorrhage**

Degree of Hydrocephalus	ACoA	ACA	PCoA	ICA	MCA	BasA	VertA
Mild	2	0	1	1	1	0	0
Moderate	3	0	0	1	0	0	3
Severe	1	0	0	1	0	0	0
Total	6	0	1	3	1	0	3

Note.—Abbreviations explained in table 1.

artery or at the basilar tip (table 2). Median time of scan for these sites was later, 7 and 9 days, respectively (table 2), than for the other groups. The lower incidence of visualized hemorrhage with delayed scanning was in agreement with a similar trend noted by Scotti et al. [2]. CT scans of aneurysms at other sites showed rates of detection for hemorrhage in excess of 60%.

Localizing Value of Hemorrhage

Cisternal hemorrhage tended to be nonspecific with the following exceptions (table 3). Hemorrhage primarily into the anterior part of the pericallosal cistern was suggestive of a source on the anterior cerebral artery (including the anterior communicating artery). However, with hemorrhage into the interhemispheric fissure, the pattern was nonspecific. Hemorrhage into the sylvian fissure was suggestive of an internal carotid, posterior communicating, or middle cerebral artery aneurysm only if there was no pericallosal or interhemispheric hemorrhage to suggest an anterior cerebral circulation aneurysm.

TABLE 11: **Hydrocephalus with and Without Intraventricular Hemorrhage**

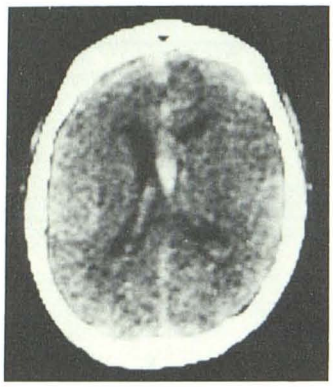
	With Intraventricular Hemorrhage	Without Intraventricular Hemorrhage
Total no. patients	14	66
Hydrocephalus	14 (100%)	33 (50%)
Moderate to severe hydrocephalus	9 (64%)	21 (32%)

In agreement with Liliequist et al. [3], we found that the pattern of parenchymal hemorrhage was more specific than that of cisternal hemorrhage for localization. Septal hemorrhage, first reported by Hayward and O'Reilly [4], occurred in eight of 27 cases of ruptured anterior communicating artery aneurysm (table 3); one was confirmed by autopsy (fig. 1). Like Weisberg [5], we found septal hemorrhage to be specific for anterior communicating artery aneurysms.

Inferomedial frontal hemorrhage occurred in six cases of ruptured anterior communicating artery aneurysm, but also in two cases of internal carotid aneurysm (one ophthalmic and one internal carotid bifurcation; table 3), both with anteriorly directed fundi. Therefore, inferomedial frontal hemorrhage was usually, but not always, due to an anterior communicating artery aneurysm.

Hemorrhage into the anterior corpus callosum was unusual but was seen in both cases of pericallosal aneurysm [5] (table 3). Parenchymal callosal hemorrhage was difficult to distinguish from pericallosal cisternal hemorrhage, a less specific type. Coronal CT scanning could improve discrimination of this difference.

Deep frontal (including basal ganglionic), lateral frontal, and temporal hemorrhages were due, in various proportions,



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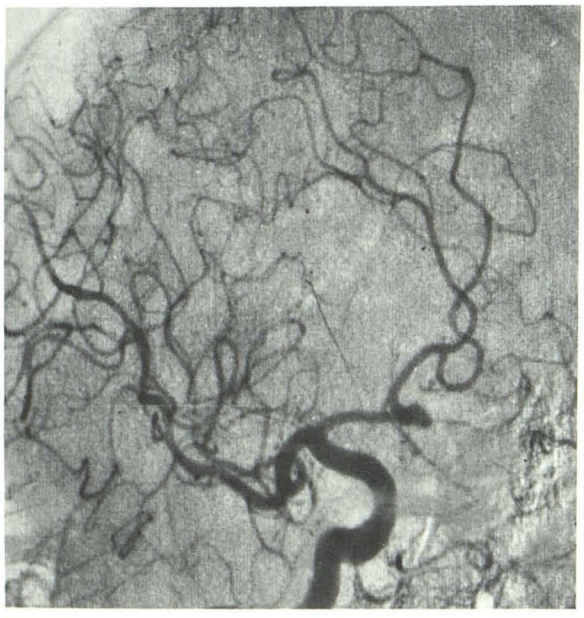

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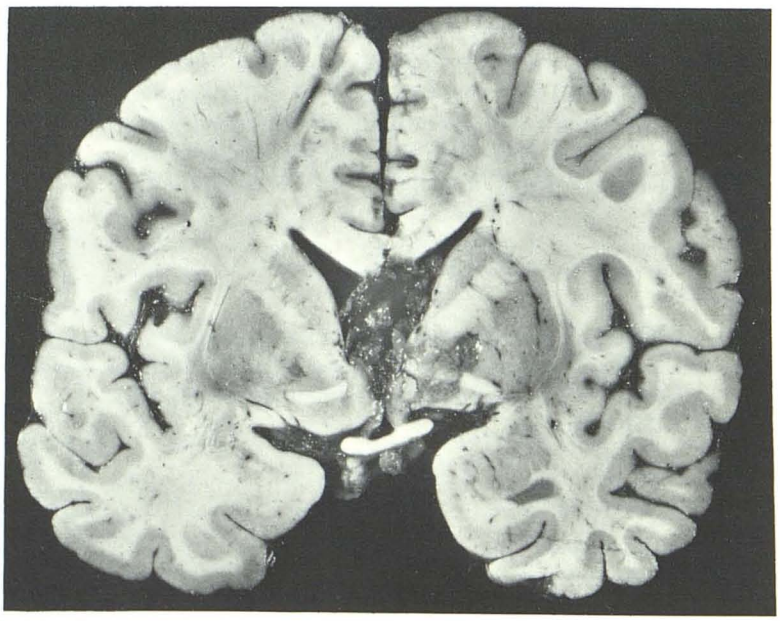
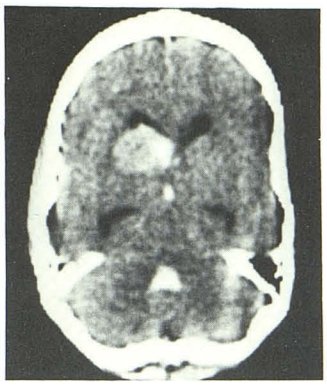


C

Fig. 1.—Septal hemorrhage due to ruptured anterior communicating artery aneurysm. **A**, Axial CT. **B**, Oblique angiogram. **C**, Autopsy confirmed large discrete hemorrhage dissecting septum pellucidum. Direction of fundus in this and several other anterior communicating artery aneurysms was not superior and did not correlate with presence of septal hematoma.



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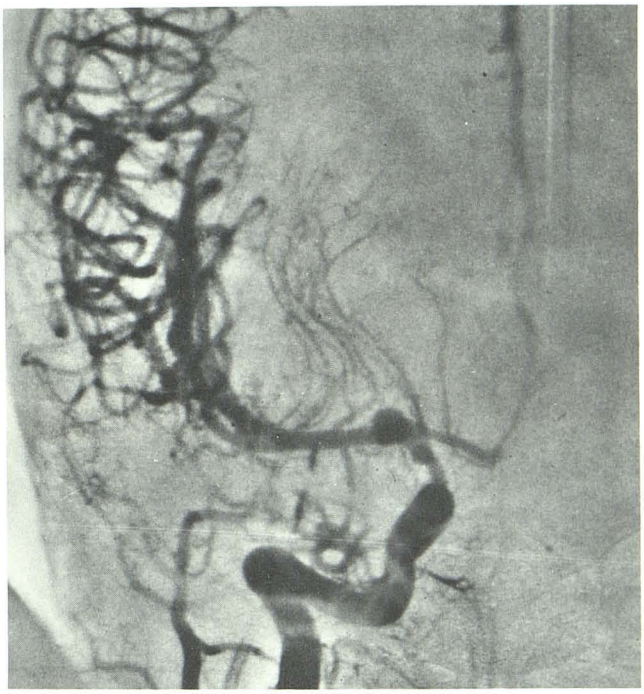


B

Fig. 2.—Hemorrhage into caudate head due to ruptured internal carotid bifurcation aneurysm. **A**, Axial CT. **B**, Frontal angiogram. Pattern could have been confused with hypertensive hemorrhage into basal ganglia. There was also smaller, more proximal, unruptured aneurysm, intraventricular hemorrhage, and hydrocephalus.

to middle cerebral, posterior communicating, and other internal carotid artery aneurysms (table 3). Distinction among these sources was usually not possible except in cases of clear-cut temporal lobe hemorrhages which were almost always due to middle cerebral artery aneurysms (table 3). Temporal lobe hemorrhages due to posterior communicating artery aneurysms were more medial than those due to aneurysms of the middle cerebral artery. At times, it was difficult to distinguish temporal lobe from sylvian fissure hemorrhages; they often occurred in combination. Occasionally, however, coronal CT scans permitted this discrimination where axial CT scans did not. Coronal CT scanning should, therefore, be of value in differentiating temporal lobe and sylvian fissure hemorrhages; that is; it should help in distinguishing middle cerebral from internal carotid arterial aneurysmal ruptures.

In his series of 50 cases of aneurysmal subarachnoid hemorrhage, Weisberg [5] found no cases of "thalamic-ganglionic" hemorrhage that could be confused with hypertensive hemorrhage. In our series, there was one case of hemorrhage into the head of the caudate nucleus (fig. 2) due to an internal carotid aneurysm and another case of recurrent hemorrhage into the region of the putamen (fig. 3) due to a middle cerebral artery aneurysm (table 3). On purely morphologic grounds, therefore, the distinction between aneurysmal and hypertensive hemorrhage into the basal ganglia may not be possible.

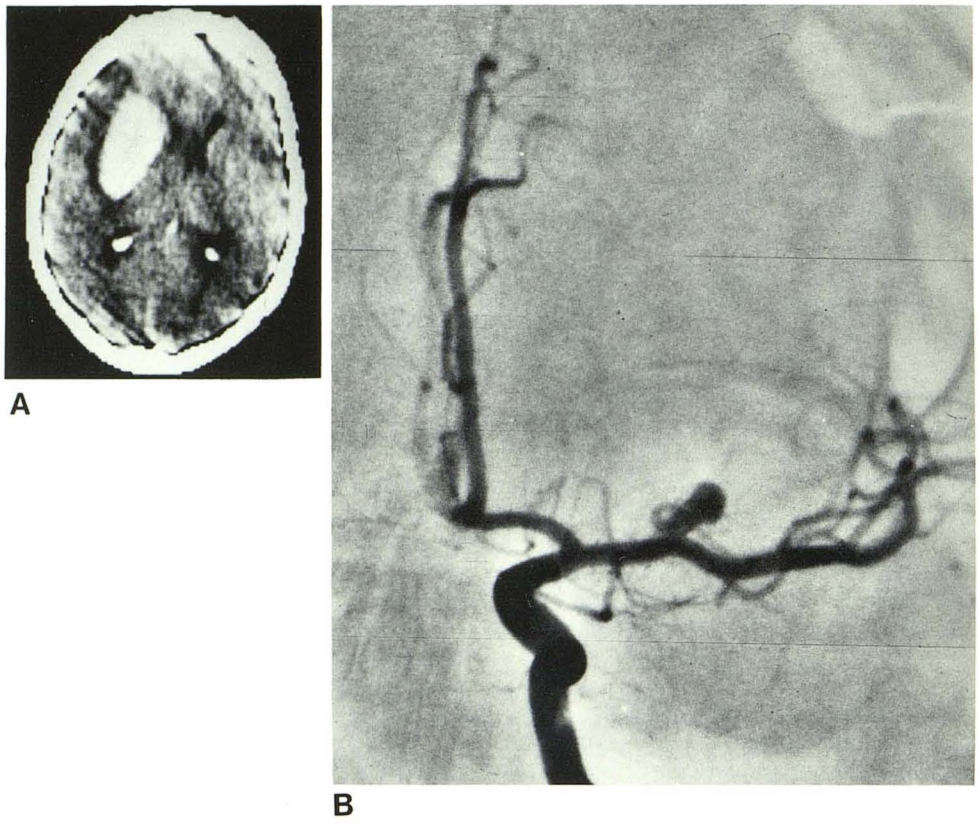
Perimesencephalic or interpeduncular cisternal hemorrhages were often due to basilar tip aneurysms (table 3). While this type of hemorrhage was not specific for basilar tip aneurysms, it was suggestive in isolation. In association with midbrain hemorrhage, it was more specific.

Hemorrhage into the fourth and third ventricles, with little or no hemorrhage into the lateral ventricles (fig. 4), was usually due to a vertebral aneurysm arising near the origin of the posterior inferior cerebellar artery (table 3). While this pattern was seen in one case of anterior communicating artery aneurysm, the third ventricular hemorrhage in that case was more dense than the fourth; with the three intraventricular hemorrhages due to posterior inferior cerebellar artery aneurysm, the relative densities were reversed. Since there was cisternal and intraventricular hemorrhage, we assume that blood entered the ventricular system via the outlet foramina of the fourth ventricle.

Lucencies and Spasm

Radiolucencies were associated with functional deficit in 85% of cases. In eight of nine cases with follow-up scans (table 6), lucencies persisted (fig. 5). In this group of patients, therefore, lucencies seemed to represent infarcts; this was confirmed by autopsy in one case. Lucencies did not seem to represent transient ischemic changes in these cases.

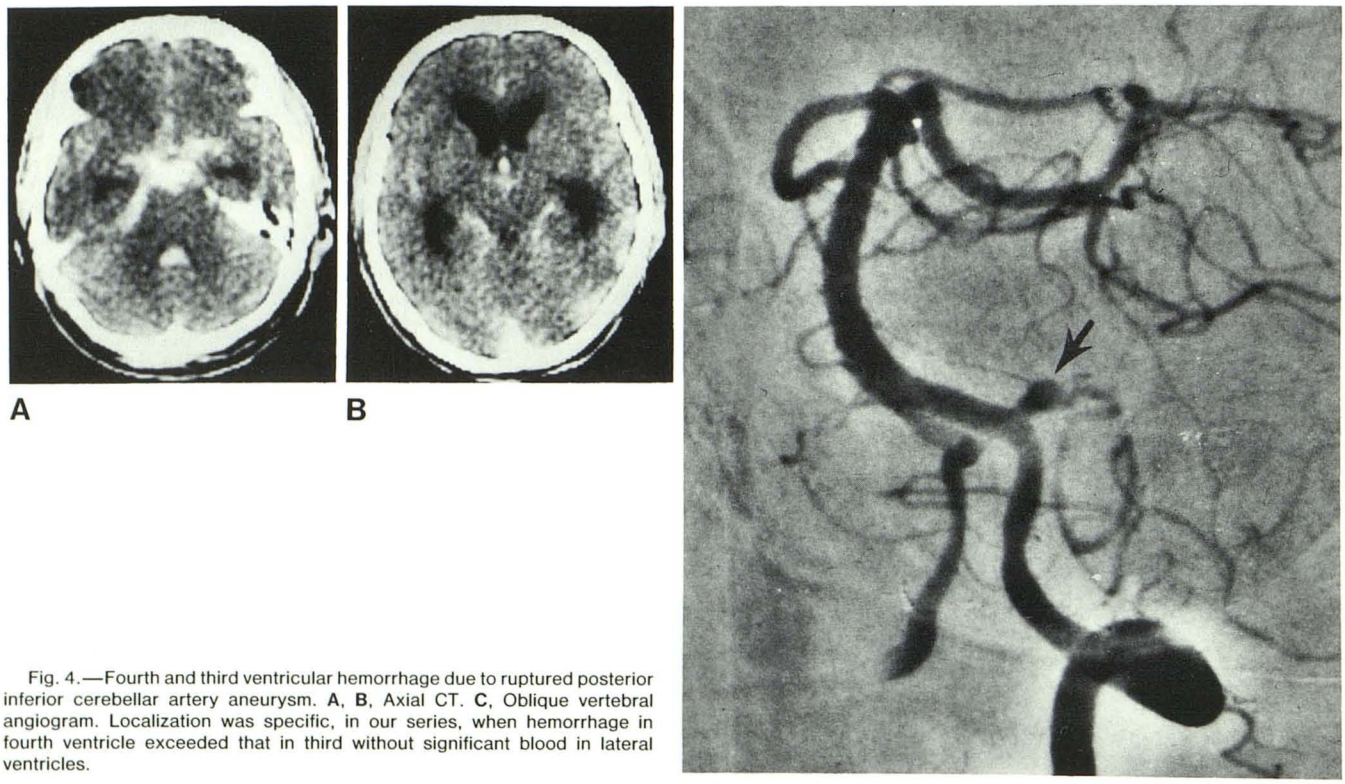
The association of clinically determined cerebral deficit with spasm was observed by Fisher et al. [6]. Liliequist et al. [3], however, did not observe a correlation between the occurrence of angiographic spasm and CT lucency, although they noted the occurrence of lucency on CT scan in two cases of clinically determined cerebral deficit. Weisberg [5] observed a high degree of association between lucency on CT scan and spasm on angiography. In five cases of CT lucency, he noted angiographic spasm in all cases. Bryan et al. [7] also noted angiographic spasm in all nine cases of CT lucency. We found angiographic spasm in 20 (95%) of 21 cases of CT lucency. In the case without associated angiographic spasm, the first angiogram was performed a month after the CT scan, so that spasm may have occurred



A

B

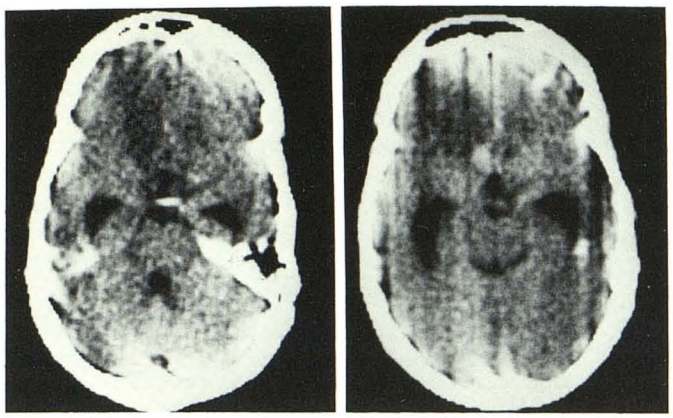
Fig. 3.—Putaminal hemorrhage due to ruptured middle cerebral artery aneurysm. **A**, Axial CT. **B**, Frontal angiogram. Unusual proximal location. Aneurysmal etiology of hematoma could not be distinguished from hypertensive hemorrhage on CT alone.



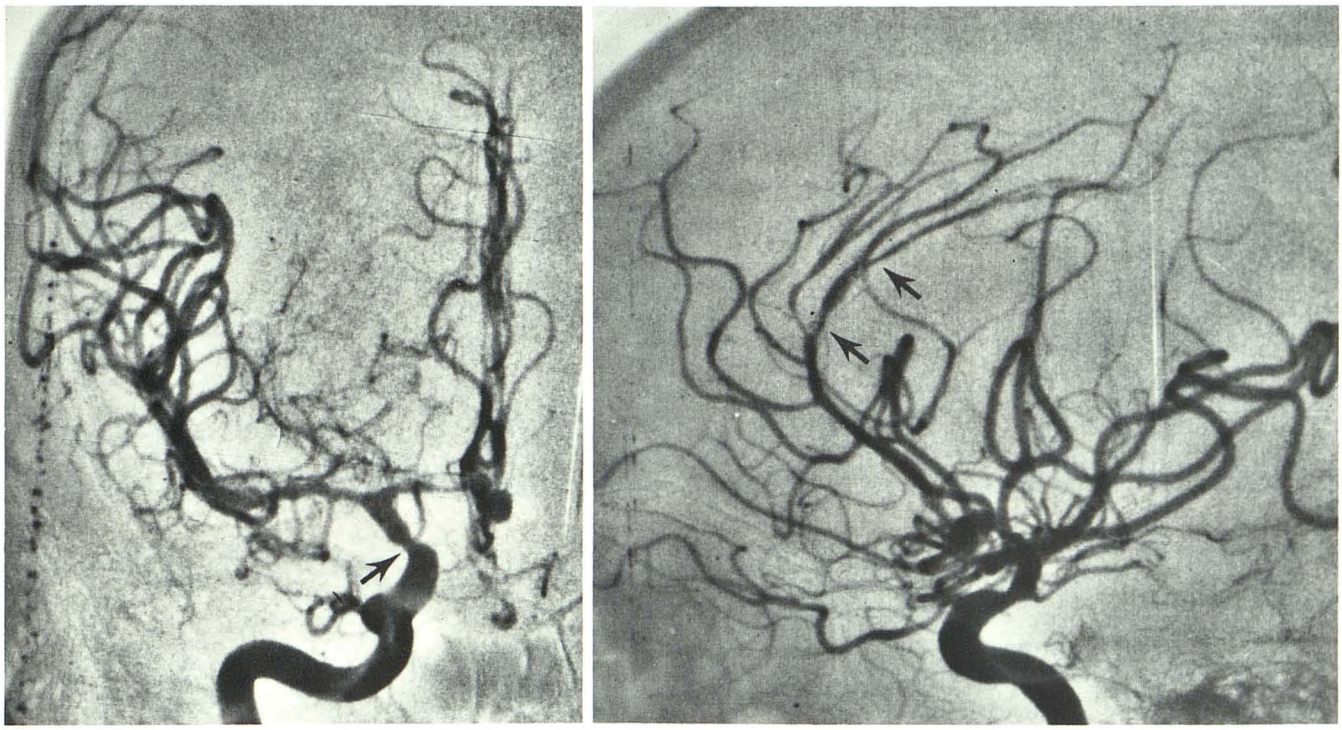
A B

C

Fig. 4.—Fourth and third ventricular hemorrhage due to ruptured posterior inferior cerebellar artery aneurysm. **A**, **B**, Axial CT. **C**, Oblique vertebral angiogram. Localization was specific, in our series, when hemorrhage in fourth ventricle exceeded that in third without significant blood in lateral ventricles.



A D



B C

Fig. 5.—Anterior communicating artery aneurysm with inferomedial frontal lucency. **A**, Axial CT. **B** and **C**, Angiograms: Proximal (**B**, *arrow*) and distal (**C**, *arrows*) spasm, both of which resolved on subsequent study. Hemiparesis was persistent. **D**, Subsequent CT revealed rebleed and persistent lucency; persistence was typical for lucencies in this series.

and resolved before angiography; in 80% of our cases with lucency, CT and angiography were performed within 4 days of each other. Saito et al. [8] described delayed development of lucency on *serial* CT scans after infarction due to spasm; we noted a similar delay in the appearance of CT lucency after vasospasm in those cases having serial CT scans and angiograms. Because of variability in the time of scanning (table 2), we did not attempt to relate extent of hemorrhage to degree of spasm, although a positive correlation was noted by Davis et al. [9].

Of 21 patients with CT lucency, 11 (52%) had distal spasm. Conversely, of 17 patients with *distal* spasm, 11 (65%) showed associated CT lucency (compared to less than 30% of all cases of *proximal* spasm showing CT lucency). Of the six cases of distal spasm *without* CT lucency, four were middle cerebral artery aneurysms with large temporal lobe hematomas; these could have masked the effects of spasm on the CT scan. In our series, distal spasm was usually associated with a discernible lucency on the CT scan, and the lucency seemed to represent infarction in almost all cases.

Saito et al. [8] emphasized the high incidence of infarction in association with spasm involving the distal branches of the middle cerebral artery; they also noted several cases of lucency with spasm of the distal anterior cerebral artery. In our series, distal spasm and lucency involved the anterior cerebral artery as frequently as the middle cerebral artery (table 5). Only one posterior circulation aneurysm rupture was associated with cortical lucency.

Circulation Time and Spasm

The circulation time was measured in 13 cases of proximal spasm and in nine cases of distal spasm according to the

method of Grietz [10]. The average circulation time in cases of distal spasm was 1.4 sec longer than in cases of proximal spasm (table 4). This difference correlated with the increased incidence of CT lucency in cases with distal spasm (figs. 6 and 7). Both sets of data show that, in our series, distal spasm represented a more severe complication than proximal spasm.

In 22 cases of spasm, both proximal and distal, the circulation time significantly exceeded normal (4.4 sec, using 50% sodium or meglumine diatrizoate [11]). This finding of increased circulation time in cases of angiographic spasm confirmed previous observations regarding circulation time as one of the most sensitive indicators of hemodynamic derangement in subarachnoid hemorrhage [12] and supplied further evidence of perfusion deficit as a cause of ischemic change in vasospasm.

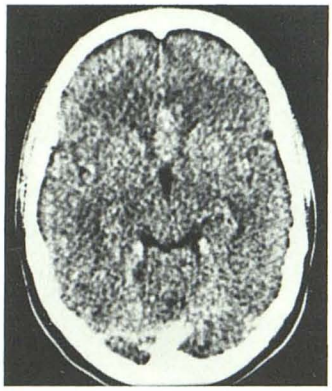
Localizing Value of Lucencies

Medial frontal lucency occurred in 12 patients and was specific for anterior cerebral artery aneurysms, either anterior communicating or pericallosal, with the exception of one case which was due to an internal carotid bifurcation aneurysm; the latter case, however, also had temporoparietal lucency (table 5). Lateral frontal, deep frontal, temporal, and parietal lucencies occurred in nine cases. Seven of these involved middle cerebral, posterior communicating, or other distal internal carotid artery aneurysms. The other two were anterior communicating and basilar tip aneurysms (table 5).

In summary, medial frontal lucencies were usually due to anterior cerebral aneurysms, while the rest (other frontal, temporal, and parietal) were due to middle cerebral, posterior communicating, other distal internal carotid, or, rarely, anterior communicating or basilar tip aneurysms.

Multiple lucencies were produced, in several cases, by anterior communicating or internal carotid bifurcation aneurysms. The lucencies were bilateral medial frontal with the former and bilateral deep or lateral frontal with the latter aneurysms.

The site of the major lucency correlated with the site of the aneurysm in all cases except those involving anterior communicating or basilar tip aneurysms. CT lucency was, therefore, of lateralizing value to the angiographer.



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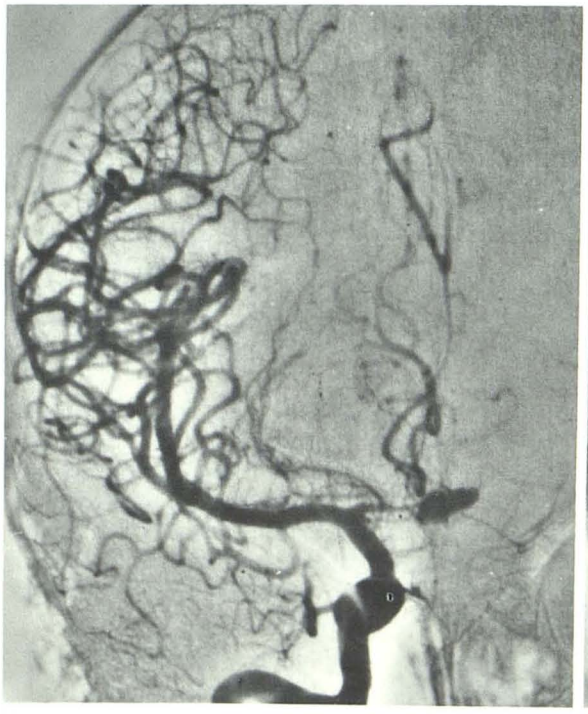

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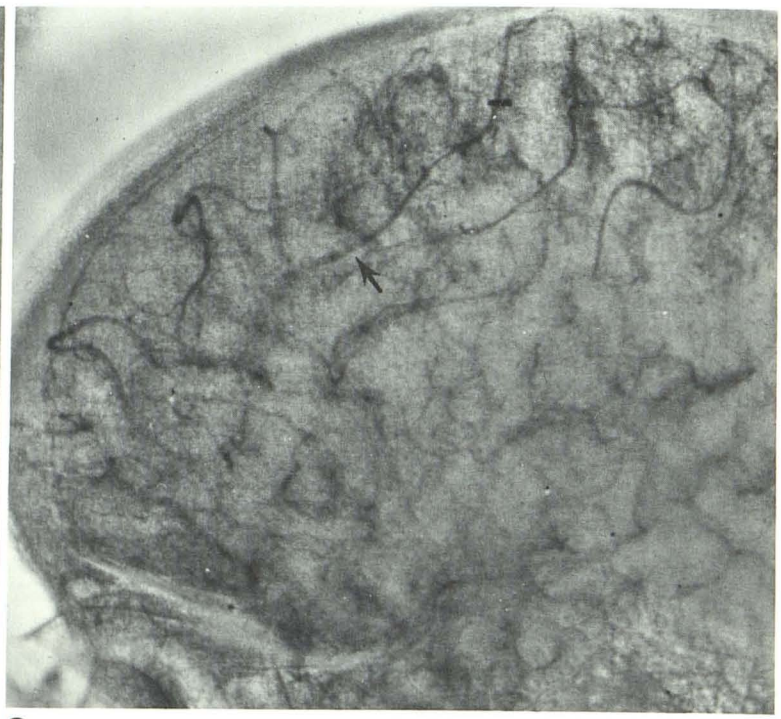

C

Fig. 6.—Anterior communicating artery aneurysm with faint bifrontal lucencies and hemorrhage into cistern of lamina terminalis. **A**, Axial CT. **B** and **C**, Angiograms. Severe distal spasm of anterior cerebral artery. Patient was young, circulation time was short (5.0 sec), and lucencies resolved on subsequent CT. **C**, Later film. Delayed filling of anterior cerebral artery branches (*arrows*) while middle cerebral vessels have entered capillary phase.

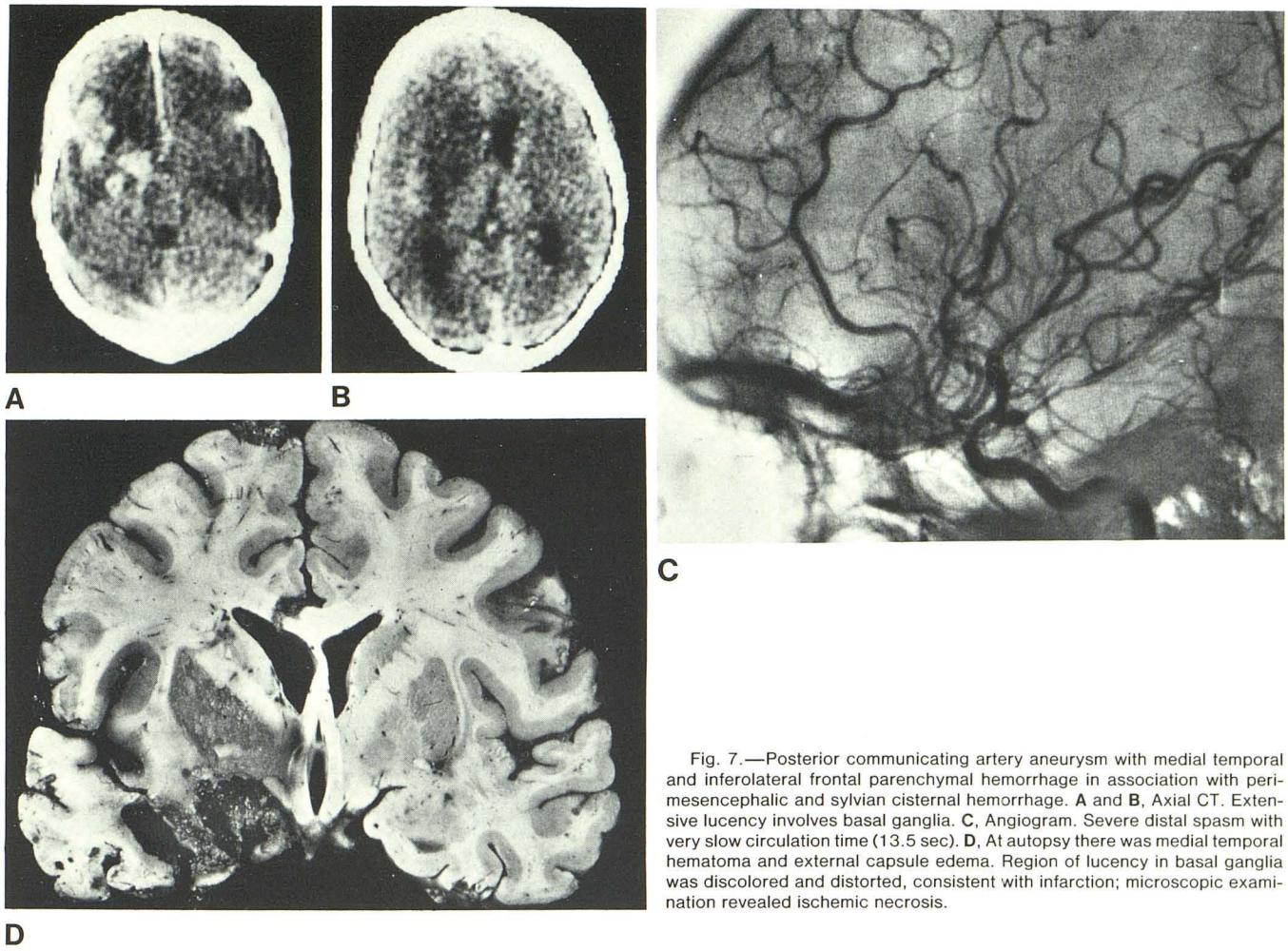

Fig. 7.—Posterior communicating artery aneurysm with medial temporal and inferolateral frontal parenchymal hemorrhage in association with perimesencephalic and sylvian cisternal hemorrhage. **A** and **B**, Axial CT. Extensive lucency involves basal ganglia. **C**, Angiogram. Severe distal spasm with very slow circulation time (13.5 sec). **D**, At autopsy there was medial temporal hematoma and external capsule edema. Region of lucency in basal ganglia was discolored and distorted, consistent with infarction; microscopic examination revealed ischemic necrosis.

Multiple Aneurysms

Of 19 patients with more than one aneurysm, one had multiple aneurysms arising from the same vessel. Of the other 18 cases, 10 had scans which permitted the correct choice of the bleeding aneurysm among the multiple aneurysms demonstrated by the angiography (table 7). Lucency determined localization in two cases, and, in a third case, fourth and third ventricular hemorrhage distinguished a source in the posterior fossa (fig. 4). The other eight scans were nonspecific or, in one case of sylvian hemorrhage, misleading. Five cases (three involving posterior communicating aneurysms) were nonspecific due to a lack of any hemorrhage or lucency on CT scan. Therefore, considering only those cases of multiple aneurysms with aneurysms arising from different vessels and with CT evidence of hemorrhage or lucency, CT was able to distinguish the *bleeding* aneurysm in 10 (77%) of 13 cases.

Hydrocephalus

Correlating the incidence of hydrocephalus with the site of the aneurysms revealed a disproportionate number of anterior communicating artery aneurysms. Periventricular lucency, however, showed a predilection for posterior circulation aneurysms (table 8).

Analysis of the progression of hydrocephalus revealed an association with periventricular lucency. All cases with hydrocephalus on the initial CT scans and with follow-up scans were tabulated for the presence of periventricular lucency on the initial scan and for the progression of hydrocephalus on follow-up scans. Of 13 cases of hydrocephalus *with* periventricular lucency on the initial scan, five (38%) showed progression on follow-up scan, while of 13 cases of hydrocephalus *without* periventricular lucency, three (23%) showed progression (table 9). These cases suggest a greater tendency for hydrocephalus with periventricular lucency to progress.

Intraventricular Hemorrhage

Intraventricular hemorrhage occurred with aneurysms at each site except the pericallosal artery and the basilar tip. There was, however, a slight preponderance of anterior communicating and, especially, posterior inferior cerebellar

artery aneurysms (table 3). There was also a tendency for anterior communicating artery aneurysms to bleed into the lateral ventricles and for posterior inferior cerebellar artery aneurysms to bleed only into the fourth and third ventricles (table 3).

Intraventricular hemorrhage was a strong predictor of hydrocephalus. In all 41 cases of intraventricular hemorrhage, there was hydrocephalus of at least a mild degree, and in nine of these cases, hydrocephalus was moderate or severe (table 10). These incidences were double those which occurred in patients without intraventricular hemorrhage on their initial CT scan (table 11). Therefore, while hydrocephalus was common after subarachnoid hemorrhage, moderate or severe hydrocephalus was more likely after intraventricular hemorrhage.

Conclusions

In this series of CT scans of 81 consecutive patients with subarachnoid hemorrhage due to ruptured aneurysm, computed tomography within 5 weeks of the ictus detected hemorrhages in slightly more than 60% of cases. Cisternal hemorrhages confined to anterior, middle, or posterior fossae showed a rough correlation with the site of the aneurysm.

Parenchymal hemorrhages showed a closer correlation with the site of aneurysm. Septal hemorrhage was specific for an anterior communicating artery aneurysm. Inferomedial frontal hemorrhage was usually due to an anterior communicating artery aneurysm (six cases), but was also seen in two cases of internal carotid aneurysm. Temporal lobe hemorrhage was usually due to a middle cerebral artery aneurysm (six cases). *Medial* temporal hemorrhage was seen in one case of a posterior communicating artery aneurysm. Deep and lateral frontal hemorrhages were specific for middle fossa aneurysms generally. Basal ganglionic hemorrhage, previously held to be characteristic of hypertensive bleed, was found in association with an internal carotid and a middle cerebral artery aneurysm. A specific pattern of third and fourth ventricular hemorrhage was found in association with all three vertebral (posterior inferior cerebellar) artery aneurysms in this series.

Regions of low attenuation (lucencies) were usually persistent on follow-up scan and were associated with functional deficit; they behaved like infarcts. Medial frontal lucency was usually due to an anterior cerebral aneurysm. Other locations of lucency were less specific. Lucencies had lateralizing value in all cases except those due to anterior communicating and basilar tip aneurysms. Bilateral lucencies were produced by anterior communicating and internal carotid bifurcation aneurysms. Lucency on computed tomography showed a high degree of correlation (95%) with angiographic spasm.

Arterial spasm correlated with lucency in many cases. "Distal" spasm was defined and found to have a significantly higher degree of correlation (65%) with lucency on CT than proximal spasm (30%). Cases of distal spasm were found to have significantly longer circulation times than those with proximal spasm. Distal spasm, therefore, represented a more significant complication in our series than the more common proximal spasm.

With multiple aneurysms arising from different vessels, CT suggested the site of the bleeding aneurysm in 77% of the cases in which there was hemorrhage or lucency on computed tomography.

Hydrocephalus was disproportionately associated with anterior communicating artery aneurysms. Periventricular lucency was a weak predictor of progression of hydrocephalus. Intraventricular hemorrhage, more common with vertebral and anterior communicating artery aneurysms, was a strong predictor of moderate to severe hydrocephalus.

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