

Are your **MRI contrast agents** cost-effective?

Learn more about generic **Gadolinium-Based Contrast Agents**.



FRESENIUS
KABI

caring for life

AJNR

Thrombus formation and structure and the evolution of mass effect in intracranial aneurysms treated by balloon embolization: emphasis on MR findings.

C M Strother, P Eldevik, Y Kikuchi, V Graves, C Partington and A Merlis

This information is current as of April 19, 2024.

AJNR Am J Neuroradiol 1989, 10 (4) 787-796

<http://www.ajnr.org/content/10/4/787>

Thrombus Formation and Structure and the Evolution of Mass Effect in Intracranial Aneurysms Treated by Balloon Embolization: Emphasis on MR Findings

Charles M. Strother^{1,2}
 Petter Eldevik²
 Yoichi Kikuchi¹
 Virgil Graves¹
 Curtis Partington¹
 Anthony Merlis³

This study was designed to further assess the capabilities of MR as a tool for the diagnostic evaluation of patients with giant intracranial aneurysms, to determine MR's ability to define the degree of thrombosis present within giant aneurysms before and after treatment with balloon occlusion, and to delineate the MR characteristics of both spontaneous and induced thrombus within giant aneurysms. Nine patients with unclippable intracranial aneurysms treated by parent artery occlusion with detachable balloons were evaluated with MR, angiography, and CT. Pretreatment and posttreatment MR studies were evaluated for their ability to (1) define the size, configuration, and anatomic relationships of an aneurysm; (2) detect and characterize thrombus within an aneurysm; and (3) determine if treatment successfully caused complete aneurysm thrombosis.

MR imaging does not replace angiography in either the pretreatment or the posttreatment evaluation of patients with giant intracranial aneurysms. Thrombus formation and dissolution is a complex, dynamic process. Active thrombus in incompletely thrombosed aneurysms differs from isolated organizing thrombus in completely thrombosed aneurysms. Induced and spontaneous thrombi differ in mechanisms of formation and in composition; their MR characteristics are also different. Reduction in mass effect is common after complete thrombosis of giant intracranial aneurysms.

When intracranial aneurysms are unclippable, occlusion of the parent artery in an effort to induce aneurysm thrombosis is often appropriate [1–3]. The use of detachable balloons offers advantages over traditional surgical techniques for achieving this goal [4, 5]. MR imaging is useful for both the diagnosis and detection of thrombus within intracranial aneurysms [6–8]. As yet, neither the reliability of MR for determining the effectiveness of proximal artery occlusion in inducing complete thrombosis within an aneurysm nor its utility for portrayal and characterization of the changes that occur within an aneurysm and the adjacent brain after initiation of such thrombosis has been fully defined.

Until recently, the MR signal characteristics seen in both partially and “completely” thrombosed aneurysms have been related to the pathologic changes known to occur in intracerebral hematomas [7, 8]. Prior to the report of Kwan et al. [9], adequate distinction had not been made between the differences in the structure of hematomas as compared with thrombi. In this report, we describe our observations on sequential MR studies in nine patients with giant intracranial aneurysms treated by parent artery balloon occlusion.

Materials and Methods

Between February 1985 and February 1988, nine patients with unclippable intracranial aneurysms treated by parent artery occlusion with silicon rubber detachable balloons* were evaluated with MR, angiography, and CT. Uninflated balloon dimensions were: outside diameter, 1.8 mm; length, 5.0 mm; and maximum volume, 0.5 cm³. Parent artery occlusion was chosen for management because of the treatment objective of obtaining reduction in mass effect after aneurysm thrombosis.

Received July 11, 1988; revision requested September 16, 1988; revision received December 5, 1988; accepted December 6, 1988.

Presented at the annual meeting of the American Society of Neuroradiology, Chicago, May 1988.

¹ Department of Radiology, Division of Neuroradiology, University of Wisconsin Clinical Health Sciences Center, Madison, WI 53792.

² Department of Neuroradiology, Ullevål Sykhus 0407, Oslo 4, Norway. Address reprint requests to C. M. Strother.

³ Department of Radiology, St. Mary's Hospital Medical Center, Madison, WI 53792.

AJNR 10:787–796, July/August 1989
 0195–6108/89/1004–0787

© American Society of Neuroradiology

* Interventional Therapeutics Corp., South San Francisco, CA.

Two patients presented with subarachnoid hemorrhage; the others were evaluated because of signs or symptoms of an intracranial mass lesion. Five aneurysms involved the intracavernous carotid artery and four involved the supraclinoid carotid artery. Two of the intracavernous aneurysms were posttraumatic; it could not be determined if these were false aneurysms or pseudoaneurysms.

Angiography was performed with standard film-screen techniques with 2:1 magnification. CT was performed with third-generation scanners, and studies were done both with and without IV contrast medium. Only axial CT images were obtained; slice thickness varied between 4 and 10 mm. MR was performed with a GE 1.5-T unit. Images were obtained in two ($n = 4$) or three ($n = 5$) orthogonal planes. T1-weighted images were obtained with a spin-echo sequence with short TRs and TEs 600/20, 25/4 (TR/TE/excitations). Proton-density- and T2-weighted images were also obtained with spin-echo sequences, 2000/20/4 and 2000/90, 120/4. The matrix size was 256×256 . The slice thickness ranged between 3 and 10 mm. Neither cardiac gating nor gradient-echo sequences was used.

All patients had at least one pretreatment CT scan and five had posttreatment scans within 48 hr of treatment. Six were studied before treatment with MR. Because not all patients were examined with MR at the same follow-up interval, they were divided into those studied within 48 hr after treatment ($n = 6$), at 5–10 days after treatment ($n = 4$), and at 4–6 weeks after treatment ($n = 4$). All but one were examined at least twice after treatment. Two had three follow-up examinations (48 hr, 5–10 days, and 4–6 weeks), three had follow-up examinations at 48 hr and 5–10 days, four had follow-up examinations at 48 hr and 4–6 weeks, and two had follow-up examinations at either 48 hr or 5–10 days and at more than 11 months. Posttreatment MR studies were analyzed both for general changes occurring in these groups of patients and for sequential changes occurring in individuals who had multiple follow-up examinations.

Each MR examination was evaluated and compared with the other imaging studies as to (1) its utility in defining the size, configuration, and anatomic relationships of an aneurysm; (2) its ability to detect and characterize thrombus within an aneurysm, and to determine if treatment was successful in causing complete thrombosis; and (3) its usefulness in determining the extent, evolution, and cause of changes in the adjacent brain (e.g., increase in mass effect) that occurred after induction of aneurysm thrombosis.

Results

Pretreatment Findings

General observations.—Three aneurysms showed no evidence of thrombus before treatment; in this group there was good agreement between measurements of aneurysm size on angiography, contrast-enhanced CT, and MR. Angiographic measurements underestimated the size of five of the six aneurysms that contained thrombus (the exception was an aneurysm with a central thrombus and patent circumferential peripheral lumen); in this group, there was good agreement between the MR and CT measurements. Angiography and MR were equally useful in defining the size and configuration of aneurysm lumens; because of their multiplanar capabilities, both were superior to CT. Only angiography allowed precise definition of the relationships of an aneurysm to both its parent vessel and other immediately adjacent vascular structures. MR was superior, however, to the other techniques in demonstrating the relationships of an aneurysm to adjacent neural and cisternal structures.

All nine patients were studied with CT before treatment. On the basis of these studies as well as plain-film analysis, three aneurysms were seen to have mural calcification; this was not clearly identified with MR in any case.

Before treatment, six of the nine aneurysms in this series were shown by at least one of the techniques to contain intraluminal thrombus. MR was the only method that identified all aneurysms containing thrombus. It was also the only technique that demonstrated the internal features of the thrombi. It was impossible with any technique to recognize the site of the thrombus attachment or to define any differences between the wall of an aneurysm adjacent to a thrombus and the wall remote from a thrombus.

MR observations.—Six of the nine aneurysms were studied with MR before treatment. Only one of these contained no thrombus; it appeared as an area of homogeneous signal void on images from all pulse sequences. The signal intensities within the other five partly thrombosed aneurysms were complex. The MR appearance of the lumens of these partially thrombosed aneurysms was determined by the blood-flow characteristics. Areas of homogeneous signal void corresponding to regions of rapid blood flow, and areas of inhomogeneous signal changing from hypointense on odd-echo images to hyperintense on even-echo images corresponding to zones of flow stasis or turbulence, were seen in all instances (Fig. 1). Evidence of abnormal flow characteristics was most evident adjacent to the intraluminal thrombus. The complex configuration and signal characteristics of the pretreatment thrombus in these aneurysms make the precise evaluation of flow characteristics most difficult because of partial-volume effects.

In every instance the MR appearances of pretreatment thrombi were similar. On T1- and proton-density-weighted images, large portions of these thrombi were laminated, having zones of irregularly alternating hypointense, isointense, and hyperintense tissue. The laminations were of variable thickness but tended to be thin, usually measuring less than 2 mm. On T2-weighted images, the bulk of all but one of these thrombi became markedly hypointense. The exception showed persistence of hyperintense signal in most of the thrombi on the T2-weighted images. Often, both within and around these spontaneous thrombi were irregular but thin zones of tissue that were slightly hyperintense on T1-, proton-density-, and T2-weighted images (Figs. 1 and 2).

The patent lumen of all aneurysms could be sharply defined with MR. It was not possible, however, to always distinguish the aneurysm lumen from the aneurysm wall, nor to identify segments of an aneurysm wall that varied in thickness. When they were adjacent, it was usually impossible to distinguish a boundary between the lumen of an aneurysm from the cortical bone of the skull base. Calcification within the wall of aneurysms also was not identified accurately with MR.

Posttreatment Findings

General observations.—After treatment, evidence of new thrombus formation was seen with both MR and CT in all nine aneurysms. Only once did angiography immediately after occlusion of the parent artery reveal persistent opacification

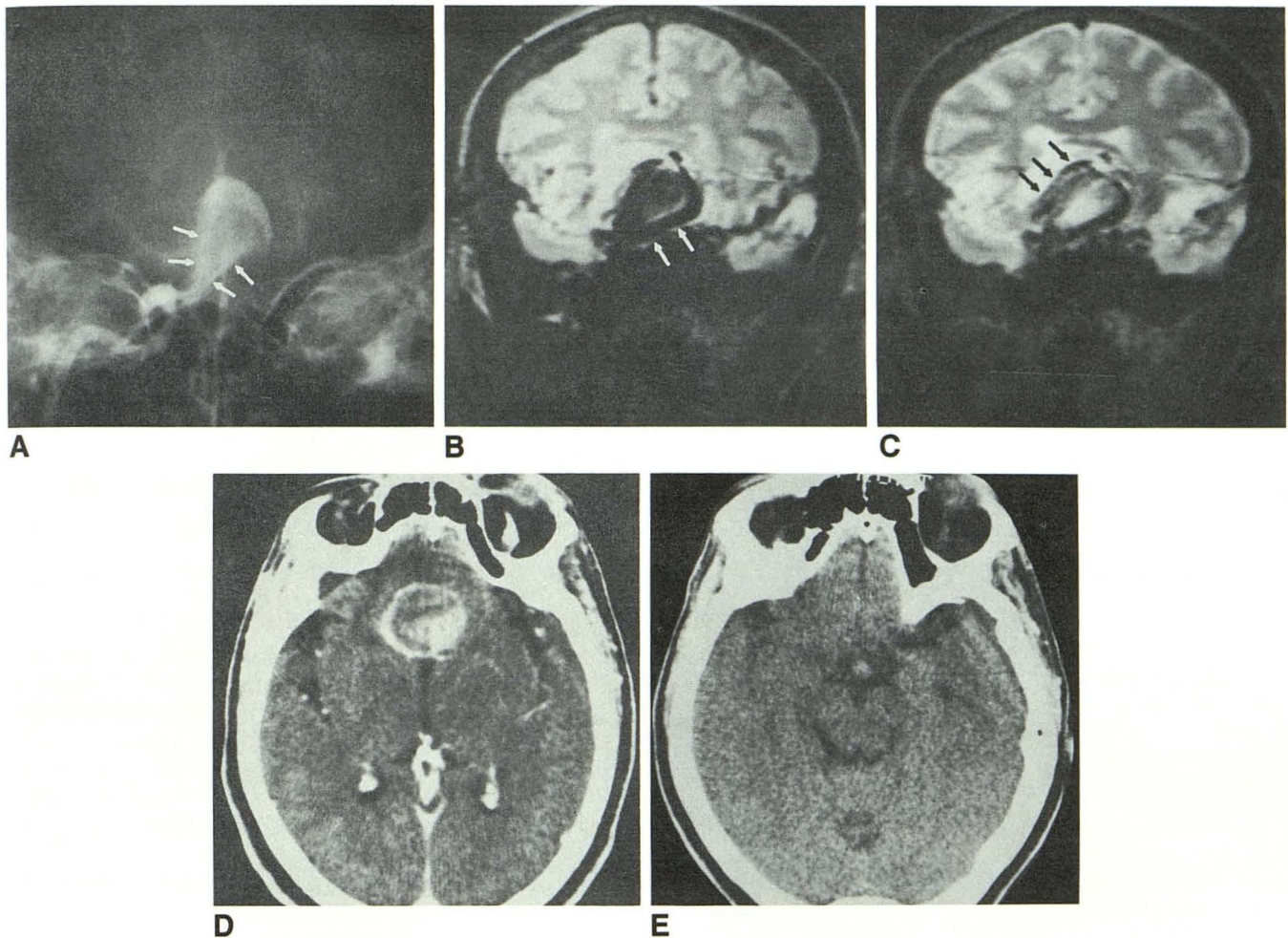


Fig. 1.—A, Right carotid arteriogram shows giant supraclinoid carotid aneurysm. V-shaped jet of rapid flow extends diagonally from neck to dome of aneurysm (arrows). Later films showed stasis of contrast material on both sides of this area and within region it delineates. Thrombus is present along right superior and lateral margin of aneurysm.

B and C, Coronal odd (B) and even (C) spin-echo images passing through aneurysm along plane that contains both thrombus and lumen. Diagonally directed area of signal void corresponds to area of rapid flow seen in A (white arrows). Region of slow turbulent flow inside perimeter of rapid flow has signal changes characteristic of even-echo refocusing. Lateral to this zone of abnormal flow is spontaneous thrombus (black arrows). Motion artifact obscures boundary of aneurysm and laminations typical of spontaneous thrombus.

D, Contrast-enhanced CT scan 19 days after treatment. Aneurysm wall enhances and appears thicker than on previous studies. Spontaneous thrombus remains isodense. After treatment, thrombus is hyperdense. There was no enhancement of either induced or spontaneous thrombus.

E, Nonenhanced scan 14 months after treatment. No abnormality is present. Because of previous adverse reaction to IV contrast medium, contrast-enhanced scan was not obtained.

of an aneurysm. This exception, a supraclinoid carotid aneurysm, was faintly opacified by collaterals.

The findings on posttreatment CT scans were similar in each instance: (1) an increase in attenuation values within the aneurysm on scans obtained at least 24 hr after treatment; (2) development of temporary low-attenuation areas (edema) in the brain adjacent to the aneurysm; (3) enhancement, thickening, and then loss of enhancement of the aneurysm walls; and (4) a significant decrease in the amount of mass effect (Fig. 1) on scans of completely thrombosed aneurysms obtained at a posttreatment interval of 11 months or greater ($n = 3$).

MR observations.—Six of the nine aneurysms were examined within 48 hr of treatment. In all of these there were

marked changes in the signal characteristics of the aneurysm lumens from those previously demonstrated (Table 1). On T1-weighted images the signal intensities from the newly formed thrombi varied slightly from slightly hypointense to slightly hyperintense. All of the induced thrombi were marked hyperintense on proton-density- and T2-weighted images when examined at this follow-up interval (Figs. 3 and 4).

Four of the nine aneurysms were examined within 5–10 days of treatment (see Table 1). In each instance on T1-weighted images, there were now, as compared with adjacent normal brain, large areas of distinct hyperintense signal present in the induced thrombus. In three it was peripheral; in the other it was central. This remained hyperintense and homogeneous on proton-density-weighted images. In each

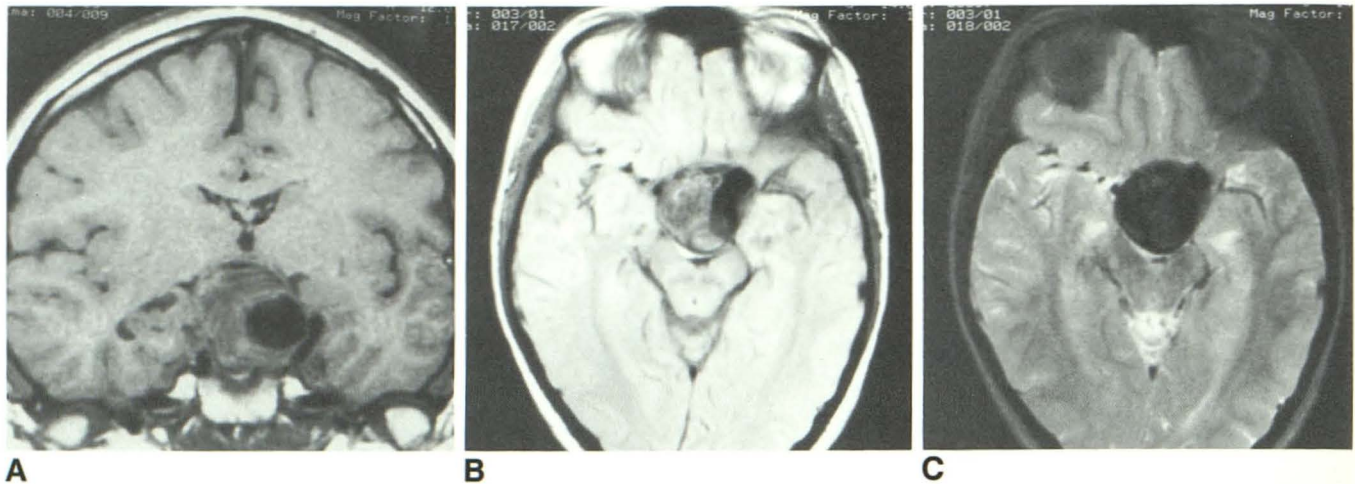


Fig. 2.—A, Pretreatment coronal T1-weighted image shows patient lumen and thrombus within aneurysm. Laminations seen in this thrombus are typical of those seen in other aneurysms.

B, Axial proton-density-weighted image of same aneurysm. Because of flow-related artifact, laminations are less apparent in this plane of section. Both within and around much of thrombus margin are layers of tissues with slightly hyperintense signal.

C, Axial T2-weighted image from same study. The majority of thrombus becomes hypointense; however, a few areas with slightly hyperintense signal remain evident. These are best seen about periphery of thrombus.

TABLE 1: MR Signal Characteristics After Balloon Occlusion of Giant Intracranial Aneurysms

Follow-up Interval/MR Sequence	MR Appearance (No.)	
	Induced Thrombus	Spontaneous Thrombus
24–48 hr (n = 6)		
T1-weighted	Minimally hypo- to hyperintense	Mostly hypointense; peripherally hyperintense
Proton-density-weighted	Hyperintense	Mostly hypointense; peripherally hyperintense
T2-weighted	Hyperintense	Hypointense
5–10 days (n = 4)		
T1-weighted	Hyperintense center (1) and periphery (3)	Area of increased hyperintensity, but mostly hypointense
Proton-density-weighted	Hyperintense center (1) or periphery (3)	Area of increased hyperintensity, but mostly hypointense
T2-weighted	Hypointense	Area of increased hyperintensity, but mostly hypointense
4–6 weeks (n = 4)		
T1-weighted	Hyperintense	Area of hyperintensity increased further; still some hypointensity
Proton-density-weighted	Hyperintense	Area of hyperintensity increased further; still some hypointensity
T2-weighted	Hyperintense	Hypointense (3); hyperintense (1)

instance, on T2-weighted images, the induced thrombus was hypointense when compared with adjacent normal brain (Figs. 3 and 4).

Four of the nine aneurysms were also examined at follow-up intervals of 4–6 weeks after treatment (see Table 1). In each instance, the induced thrombus was uniform and hyperintense on all pulse sequences.

In all of the six cases studied within 48 hr of treatment, the spontaneous thrombus showed some differences from its appearance on the pretreatment studies. To a variable but consistent degree, at this follow-up interval there were thin, irregular streaks within these thrombi that were hyperintense on T1- and proton-density-weighted images but hypointense

on T2-weighted images (Table 1). These were most prominent around the thrombus periphery (Figs. 3 and 4).

Spontaneous thrombi in the four aneurysms studied at a posttreatment interval of 5–10 days showed an increase in the extent of these signal characteristics (see Table 1). Tissue with hyperintense signal was still most prominent around the periphery of the thrombus. In one instance there was a distinct difference in the appearance of that portion of the spontaneous thrombus bordering the induced thrombus as compared with its more peripheral portion (Fig. 3). Similar but less striking changes along this boundary zone were present in two of the other cases as well. Overall, the spontaneous thrombus remained hypointense on the T2-weighted image;

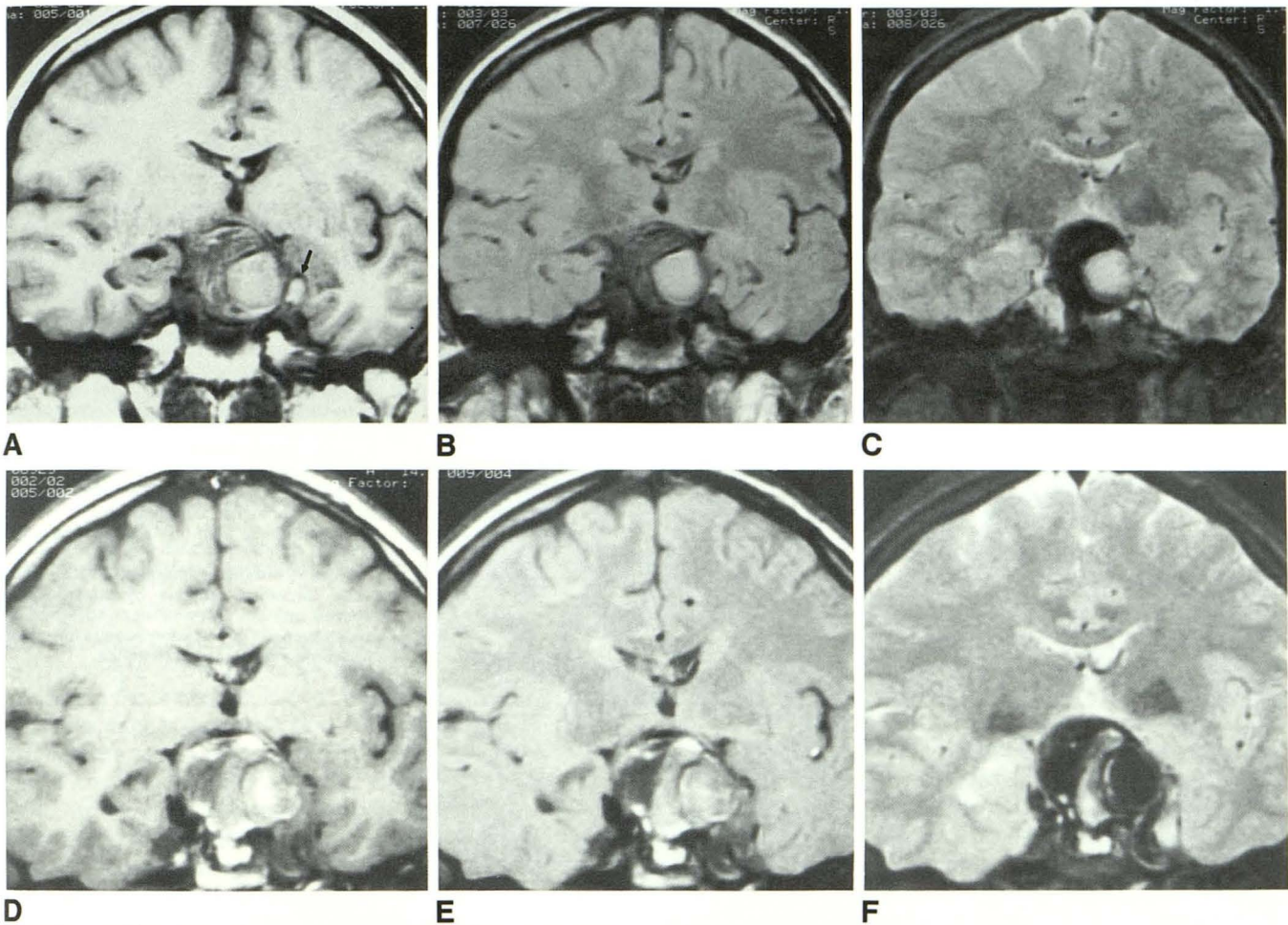


Fig. 3.—A, Coronal T1-weighted image 24 hr after treatment. Induced thrombus within aneurysm and adjacent internal carotid artery (arrow) is slightly hyperintense. Streaklike areas with more hyperintense signal are present in and around adjacent pretreatment thrombus. These have increased since pretreatment scan. Line of dark signal separates induced from spontaneous thrombus.
 B, Coronal proton-density-weighted image from same study. Induced thrombus is homogeneous and hyperintense as compared with gray matter. Well-defined margin persists between spontaneous and induced thrombus.
 C, Coronal T2-weighted image from same study. Induced thrombus remains hyperintense while pretreatment thrombus becomes quite hypointense. Some edema is evident in brain adjacent to aneurysm dome.
 D, Coronal T1-weighted image 5 days after treatment. Central portion of induced thrombus has become more hyperintense. Increasing waferlike zones of hyperintense signal have developed in spontaneous thrombus. Portion of spontaneous thrombus bordering induced thrombus has lost its laminations and has signal characteristics different from more peripheral portion.
 E, Coronal proton-density-weighted image from same study. Areas of hyperintensity are present in both spontaneous and induced thrombus. Portion of spontaneous thrombus abutting posttreatment thrombus is almost all hyperintense, while peripheral portion is largely hypointense.
 F, Coronal T2-weighted image from same study. Almost all of the posttreatment thrombus and most of the pretreatment thrombus is hypointense. High-intensity tissue persists, however, in zone of pretreatment thrombus abutting induced thrombus.

however, the degree of hypointensity was less than that observed on earlier follow-up studies. The signal intensities from these spontaneous thrombi also were somewhat less uniform than they had been on previous studies (Fig. 4). Similar changes were present in the four patients examined at posttreatment intervals of 4–6 weeks.

All six aneurysms examined within 48 hr of treatment showed a slight increase in mass effect. In two of these there was also evidence of development of edema in the brain adjacent to the aneurysm (Fig. 3). All of these patients had some headache at this follow-up interval; in the two with evidence of edema, the headache was significant in severity.

Two of the four aneurysms examined at a posttreatment follow-up interval of 4–6 weeks showed some decrease in

mass effect from the aneurysm, and when observed at a follow-up interval of greater than 11 months, three of four aneurysms showed a dramatic reduction in size. In two instances, examinations at follow-up intervals of 26 and 27 months after treatment showed no evidence of any residual mass (Figs. 5 and 6).

The one aneurysm that showed no decrease in size was the only one in the series that underwent incomplete thrombosis. Follow-up angiography, CT, and MR examinations at intervals of 18 and 24 months after treatment demonstrated first a decreasing and then an increasing lumen size with no change in the mass effect. The signal characteristics of the large spontaneous thrombus were complex and not typical of those seen in the other aneurysms in our series (Fig. 7). They

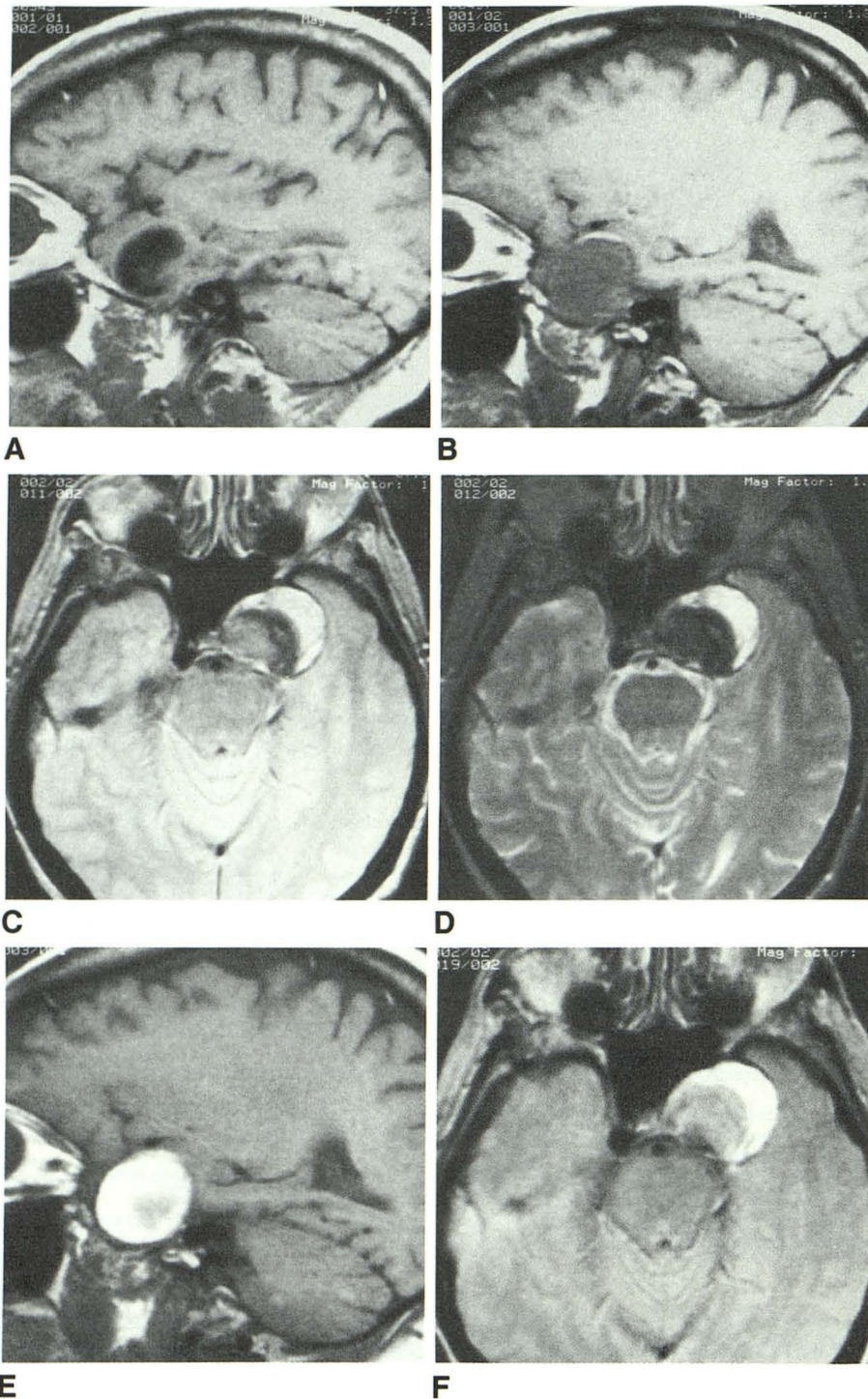


Fig. 4.—A, Parasagittal T1-weighted image through lumen of aneurysm before treatment. Small amount of spontaneous thrombus projects into posterior portion of lumen.

B, Corresponding T1-weighted image 48 hr after treatment. Induced thrombus is homogeneous and isointense as compared with adjacent normal gray matter. Small rim of hyperintense tissue is present along upper portion of aneurysm.

C, Axial proton-density-weighted image from same study. Induced thrombus lies anteriorly and laterally and is hyperintense. Spontaneous thrombus lying along back and medial side of aneurysm is of somewhat variable intensity but overall is hypointense.

D, Axial T2-weighted image from same study. Induced thrombus remains hyperintense. Spontaneous thrombus becomes more hypointense.

E, Parasagittal T1-weighted image 7 days after treatment. Almost all of induced thrombus is now hyperintense.

F, Axial proton-density-weighted image from same study. Induced thrombus is more hyperintense than on scan 48 hr after treatment. Spontaneous thrombus has changed also and now is more hyperintense than on 48-hr posttreatment scan.

G, Axial T2-weighted image from same study. Induced thrombus remains hyperintense. As compared with both pretreatment scan and 48-hr posttreatment scan, spontaneous thrombus has become much less hypointense.

did not change during the follow-up interval. Sedimentation levels were not seen in any aneurysm at any follow-up interval.

Discussion

Pretreatment Assessment

When angiography, CT, and MR were compared for their abilities to provide information that allowed distinctions between those aneurysms suitable for surgical therapy and

those not amenable to such treatment, they were seen to be complementary. MR provided the clearest indication of an aneurysm's relationships to adjacent neural and cisternal structures. This results from a combination of the benefits derived from superior contrast resolution and multiplanar capabilities. MR was not useful, however, in defining the relationships between an aneurysm and cortical bone or a pneumatized sinus. Whereas CT was of little value in determining precise relationships of an aneurysm to neural, cister-

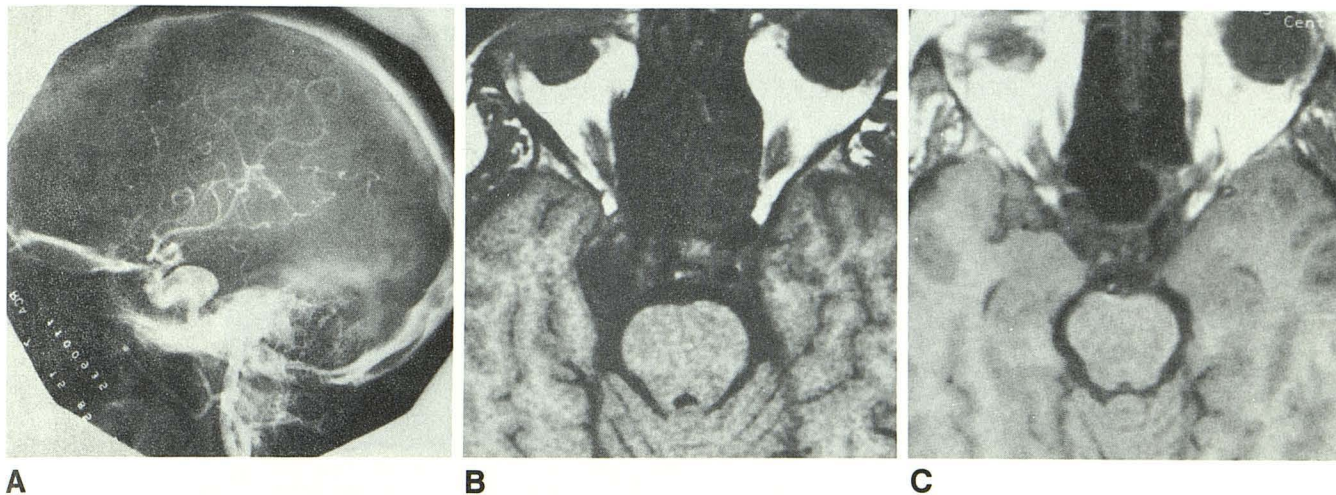


Fig. 5.—**A**, Lateral projection of right common carotid angiogram shows giant intracavernous aneurysm.
B, T1-weighted image before treatment shows marked enlargement of right cavernous sinus. Some mass effect is evident on medial surface of adjacent temporal lobe.
C, T1-weighted image 26 months after treatment. Except for absence of flow void in internal carotid artery, no abnormality is present. Proton-density- and T2-weighted images showed same findings.

nal, and vascular structures, it was superior to both MR and angiography in allowing recognition of mural calcification and in defining the relationships of an aneurysm to the skull base; this advantage is important since wall calcification and proximity to the skull base are two important factors that may significantly hinder successful clip application. MR was superior to the other techniques for both recognition and characterization of pretreatment aneurysm thrombus.

In our study, the precise relationships of an aneurysm to its parent vessel were seen adequately only with angiography. Although MR also provided information of this nature, in all instances it was inadequate for deciding whether an aneurysm was suitable for surgical clipping. The use of cine MR with gradient-refocused imaging as described by Tsuruda et al. [10] further enhances the capabilities of MR in this regard. We believe the potential for error caused by partial-volume artifacts, limited spatial resolution, and suboptimal flow-related enhancement still makes assessment with angiography necessary.

Assessing the Effectiveness of Treatment

Partial thrombosis of a giant aneurysm, even if massive, does not eliminate the risk of subsequent rupture [1, 11]. Enlargement of partially thrombosed giant aneurysms has also been documented [11, 12]. Although the signs and symptoms caused by the mass of a giant aneurysm often respond dramatically to induction of thrombosis without removal of the aneurysm itself, this benefit has usually been attributed to a reduction of arterial pulsations on adjacent neural structures; there are only a few well-described examples in which the mass has diminished after aneurysm thrombosis [1]. To our knowledge, there has been no previous discussion of the effect of almost complete as compared with total aneurysm thrombosis on the evolution of the mass associated with intracranial aneurysms; in fact, distinction usually is not made between those aneurysms that are almost

completely thrombosed and those that are entirely thrombosed [13].

Our experience indicates that when total thrombosis is achieved there often will be a decrease in the mass of the aneurysm over a period of time. Persistence of even a small lumen seems to make evolution less likely. This is compatible with the observation in pathologic material that often in aneurysms that contain extensive thrombus, a small lumen remains in contact with a largely thrombosed sac. The periphery of the thrombus is composed of fresh thrombus and RBCs, while its center is made up principally of dense fibrous material; that is, a central organized thrombus with a marginal, fresh thrombus and flowing blood [12].

Six of nine aneurysms in our series had MR follow-ups adequate to assess the change in mass effect occurring after treatment. Five of these had total thrombosis, and at intervals ranging between 6 weeks and 27 months, all showed evidence of decreased mass effect. One aneurysm with at least 90% obliteration of its lumen showed no change in mass effect over a follow-up interval of 2 years.

In our series, unequivocal evidence of total aneurysm thrombosis was provided only by angiography. Although the use of cine gradient-refocused imaging techniques augments both the anatomic and physiologic information available from MR evaluation of vascular lesions, the limitations of these techniques remain incompletely defined [10]. The frequent presence of aneurysmal wall calcification, flow-related phenomena, and the signal characteristics of evolving thrombus, in our opinion, make it unlikely that even these methods will allow one to be certain that tiny but potentially lethal residual aneurysm lumens do not persist.

MR Characteristics of Arterial Thrombi

A thrombus is a "solid mass or plug formed in the living heart or vessels from constituents of the blood" [14]. Depending on its etiology, its site of formation, and the charac-

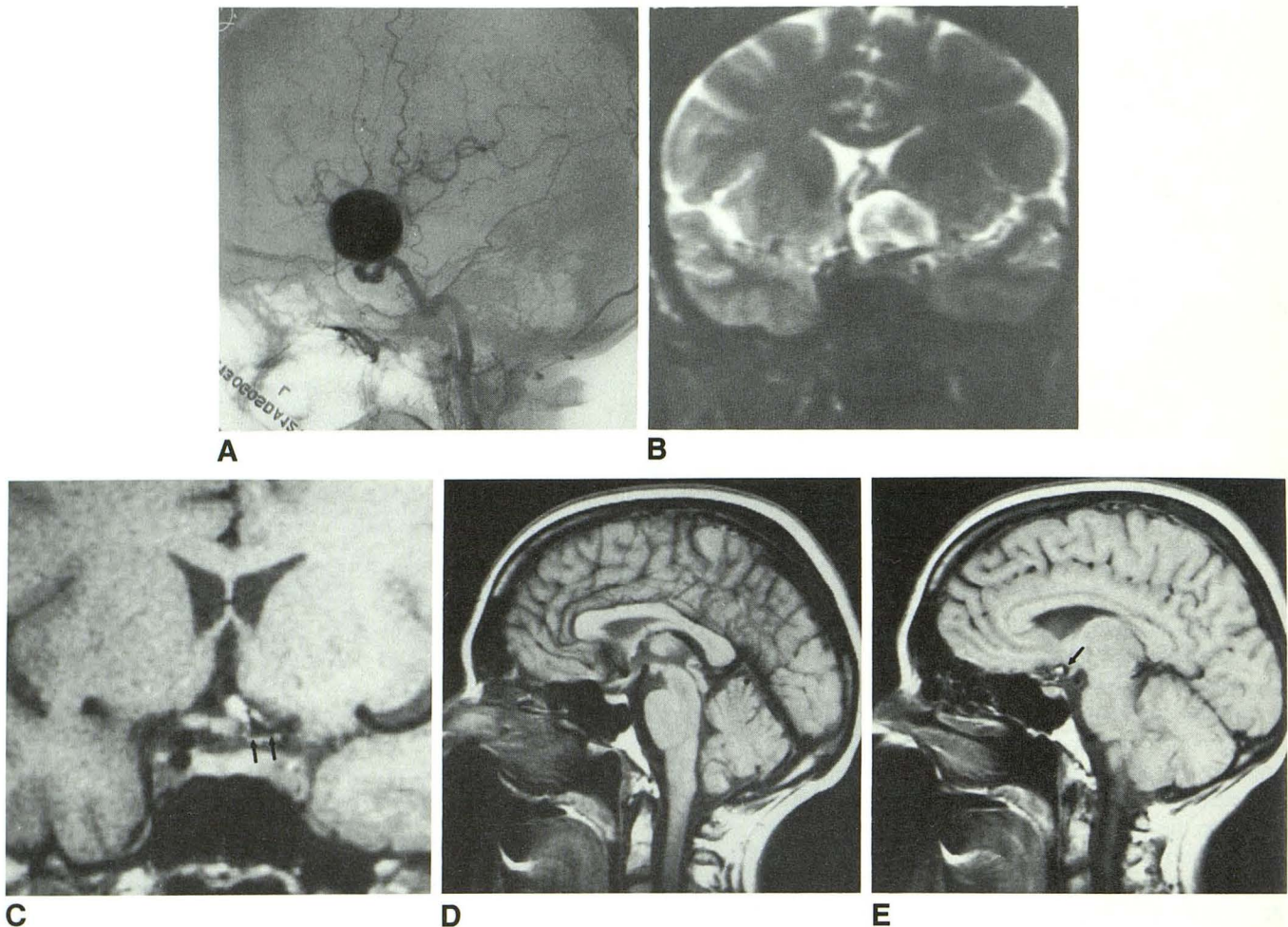


Fig. 6.—**A**, Lateral projection of left carotid angiogram shows giant parophthalmic aneurysm. Before treatment no thrombus was present in this aneurysm.
B, Coronal T2-weighted image 10 days after treatment shows mass effect from aneurysm.
C, Coronal T1-weighted image 11 months after treatment. Reduction in mass effect is striking. Small area of hyperintense signal persists just above left side of optic chiasm. At its base is triangular area of homogeneous signal void (arrows).
D, Sagittal T1-weighted image from same study. No abnormality is present.
E, Parasagittal T1-weighted image just to the left side of **D**. Only a small area of hyperintense signal persists at origin of aneurysm. U-shaped area of signal void is present just behind this (arrow).

teristics of blood flow where it originates, the composition of a thrombus is a spectrum ranging between thrombi composed predominantly of platelets and fibrin (i.e., arterial or white thrombi) and those in which a much larger component of their bulk comprises RBCs (i.e., venous or red thrombi) [15]. Both by definition and composition, a thrombus is clearly distinguished from a hematoma.

Thrombus formation and dissolution is dynamic, and once formed, a thrombus may evolve through lysis, embolization, or organization, none of which are mutually exclusive. Completely organized thrombi are converted entirely into smooth muscle and collagen. During organization, as the result of thrombus lysis, contraction, and the formation of new capillaries derived from the wall of the vessel containing the thrombus, new endothelialized channels form in and around an organizing thrombus. Thrombus organization is impaired when there is injury to the vessel wall containing a thrombus,

and when this is severe, organization may either not occur or be incomplete [15–17].

It is impossible to predict how a particular thrombus will evolve, and to our knowledge, there are no pathologic studies of the evolution of thrombi within aneurysms of the intracranial circulation. It seems probable, however, that a thrombus that originates in a vessel, the walls of which are not severely injured and can be isolated completely from the circulation, is more likely to organize completely and consequently decrease in size than is one that occurs under other circumstances. None of the aneurysms in our series that decreased in size had evidence of severe arterial wall injury, that is, calcification. Persistence of any residual aneurysm lumen inhibits complete thrombus organization.

The MR appearance of thrombi, like intraparenchymal hematomas, is diverse and is influenced by a multiplicity of factors that may in turn influence one another in ways not yet

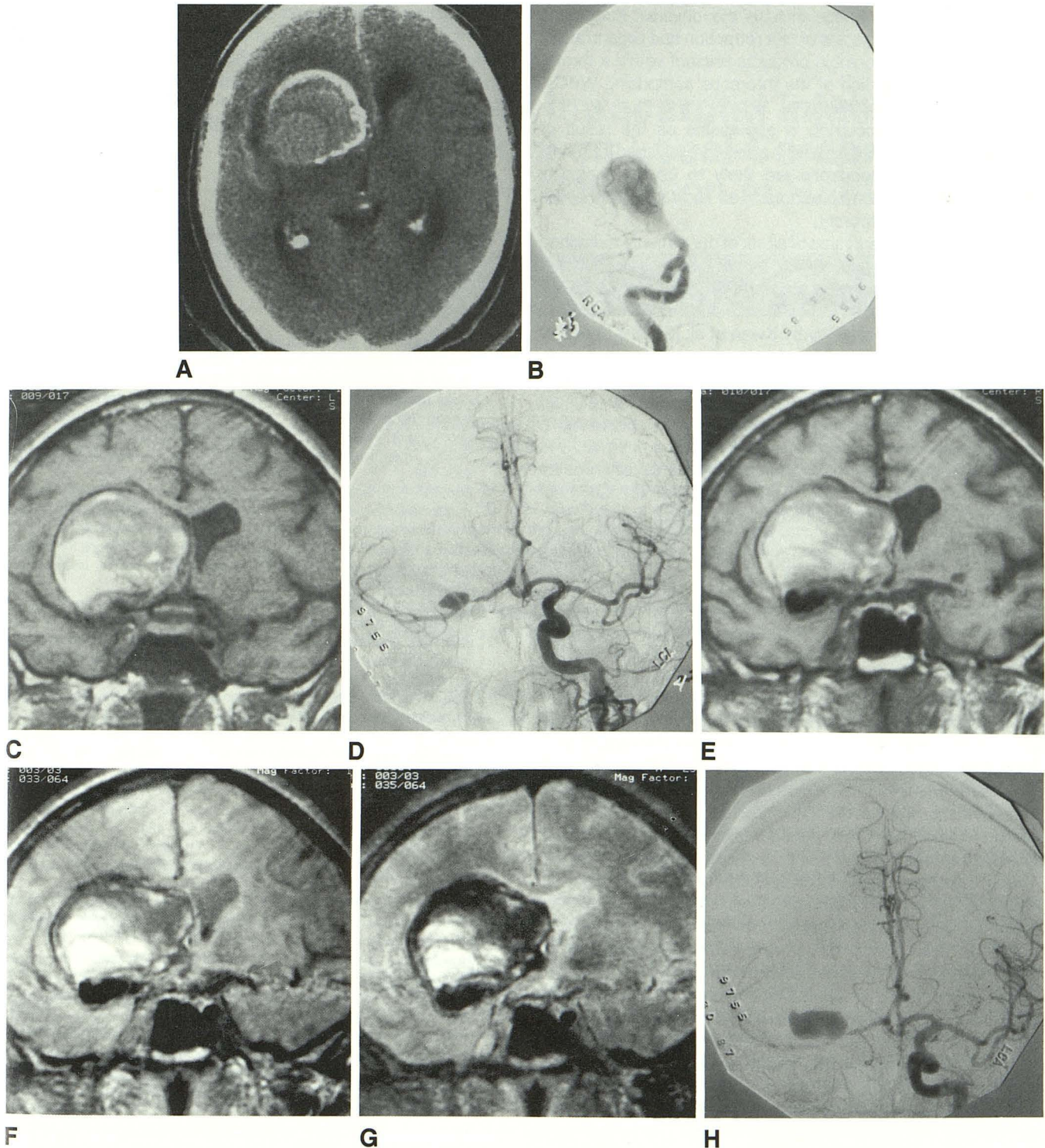


Fig. 7.—A, Nonenhanced CT scan before treatment. Blood is present in interhemispheric and right sylvian fissures. Aneurysm lumen is of higher density than is adjacent thrombus. There is thick calcification in much of aneurysm wall.

B, Frontal projection from right carotid angiogram the next day.

C, Coronal T1-weighted image 17 months after treatment. Small, indistinct area of flow void persists at aneurysm base. Thrombus with varying signal intensity fills the rest of aneurysm.

D, Frontal projection from right carotid angiogram the day after scan shown in C. Lumen of aneurysm has greatly decreased since treatment; it corresponds to area of flow void seen on MR scan.

E, Coronal T1-weighted image 6 months later. Except for increase in amount of flow void at aneurysm base there is no change in size or configuration of aneurysm. There is some variation in signal intensities from those observed in C.

F, Coronal proton-density-weighted image from same study. Thrombus comprises tissue of both hyperintense and slightly hypointense signal intensity. Note thin, peripheral rim of hyperintense signal. This was also present on images obtained 17 months after treatment.

G, Coronal T2-weighted image from same study shows both hyperintense and hypointense signal in thrombus. These characteristics were also present on study 17 months after treatment.

H, Frontal projection of right carotid angiogram confirms increase in lumen size.

thoroughly defined. These include the physical state of a thrombus, that is, the state of clot retraction and organization; the varieties of hemoglobin products present within a thrombus; and the proportion of the thrombus comprising WBCs, platelet products, and fibrin [18, 19].

Those thrombi occurring in aneurysms as the result of balloon occlusion of the parent artery are initiated primarily by stasis and consequently are likely to be more closely related in composition to venous (i.e., red) thrombi than to arterial (i.e., white) thrombi.

Thrombi in the aneurysms of all six of our patients examined within 48 hr after treatment had signal intensities consistent with those of nonflowing blood, the erythrocytes of which still contain oxyhemoglobin [20]. These observations coincide with those reported recently by Kwan et al. [9]. The speed at which thrombosis occurred in these aneurysms as well as its extent at this follow-up interval are unknown. The absence of sedimentation in any of these aneurysms implies that, at least within 48 hr of being isolated from the circulation, blood within an aneurysm is thrombosed.

In the period 5–10 days after treatment, as erythrocyte lysis, hemoglobin breakdown, and thrombus organization occurred, these stasis thrombi assumed some of the MR characteristics of parenchymal hematomas. By 4–6 weeks after treatment, the predominant signal intensity in these thrombi corresponded to that expected for extracellular methemoglobin. They differed from the typical appearance of an evolving hematoma, however, in that at this, as well as at longer follow-up intervals, tissue with a signal intensity corresponding to that of hemosiderin was less prominent in these thrombi than in parenchymal hematomas of corresponding volume.

The spontaneous or pretreatment thrombi in these aneurysms were very different from the induced thrombi, both in their initial appearance and in their evolution after treatment. On pretreatment MR, thrombus did not have the appearance characteristic of an evolving hematoma. After isolation from the circulation, they underwent complex changes that overall acted to shorten their T1 and to greatly lengthen their T2. These alterations began to be apparent on scans obtained as early as 24 hr after treatment. Similar but more extensive changes were seen on scans obtained at a 5- to 10-day posttreatment interval; these were even more apparent on images obtained 4–6 weeks after treatment.

Because detailed histopathologic information about the evolution and organization of intraaneurysmal thrombi is lacking, explanation of the reason for these changes is at best speculative. The influence, if any, that these two types of thrombi exert on each other is unknown.

The dynamic nature of intraarterial thrombus formation, dissolution, and reformation is established and is well illustrated by the serial imaging studies in our patients. A review of the literature combined with our experiences with these nine cases, as well as others not having adequate sequential MR studies to be included in this report, indicates that reduction in the mass effect caused by giant aneurysms can be expected only when complete thrombosis occurs. Reduction in mass effect occurs frequently after complete thrombosis of giant intracranial aneurysms.

Thrombus formation and dissolution is a complex, dynamic process. Active thrombus in incompletely thrombosed aneurysms differs from isolated organizing thrombus in completely thrombosed aneurysms. Spontaneous thrombus and thrombus induced in aneurysm by parent artery occlusion differ in mechanisms of formation and composition. Their MR characteristics are also different.

ACKNOWLEDGMENTS

We thank our neurosurgical colleagues, T. Duff, L. Ramirez, and S. Toutant, and M. Boyer for help and cooperation.

REFERENCES

1. Drake CG. Giant intracranial aneurysms: experience with surgical treatment in 174 patients. *Clin Neurosurg* 1979;26:12–95
2. Giannotta SL, McGillicuddy JE, Kindt GW. Gradual carotid artery occlusion in the treatment of inaccessible internal carotid artery aneurysms. *Neurosurgery* 1979;5:417–421
3. Sundt TM Jr, Piepgras DG. Surgical approach to giant intracranial aneurysms. Operative experience with 80 cases. *J Neurosurg* 1979;51:731–742
4. Berenstein A, Ransohoff J, Kupersmith M, et al. Transvascular treatment of giant aneurysms of the cavernous carotid and vertebral arteries. Functional investigation and embolization. *Surg Neurol* 1984;21:3–12
5. Fox AJ, Vinuela F, Pelz DM, et al. Use of detachable balloons for proximal artery occlusion in the treatment of unclippable cerebral aneurysms. *J Neurosurg* 1987;66:40–46
6. Worthington BS, Kean DM, Hawkes RC. NMR imaging in the recognition of giant intracranial aneurysms. *AJNR* 1983;4:835–836
7. Atlas SW, Grossman RI, Goldberg HI, et al. Partially thrombosed giant intracranial aneurysms: correlation of MR and pathologic findings. *Radiology* 1987;162:111–114
8. Olsen WL, Brant-Zawadzki M, Hodes J, Norman D, Newton TH. Giant intracranial aneurysms: MR imaging. *Radiology* 1987;163:431–435
9. Kwan ESK, Wolpert SM, Scott RM, Runge V. MR evaluation of neurovascular lesions after endovascular occlusion with detachable balloons. *AJNR* 1988;9:523–531
10. Tsuruda JS, Halbach VV, Higashida RT, Mark AS, Hieshima GB, Norman D. MR evaluation of large intracranial aneurysms using cine low flip angle gradient-refocused imaging. *AJNR* 1988;9:415–424
11. Whittle IR, Dorsch NW, Besser M. Spontaneous thrombosis in giant intracranial aneurysms. *J Neurol Neurosurg Psychiatry* 1982;45:1040–1047
12. Artmann H, Vonofakos D, Muller H, Grau H. Neuroradiologic and neuropathologic findings with growing giant intracranial aneurysm. Review of the literature. *Surg Neurol* 1984;21:391–401
13. Hosobuchi Y. Direct surgical treatment of giant intracranial aneurysms. *J Neurosurg* 1979;51:743–756
14. Welch WH. Thrombosis. In: Albutt TC, ed. *A system of medicine*, vol. 6. New York: Macmillan, 1899:155–228
15. Freiman DG. The structure of thrombi. In: Colman RW, Hirsh J, Marder VJ, Salzman EW, eds. *Hemostasis and thrombosis. Basic principles and clinical practice*. Philadelphia: Lippincott, 1982:716–717
16. Caro CG, Pedley TJ, Schroter RC, Seed WA. *Mechanics of the circulation*. New York: Oxford University Press, 1978
17. Carlos T. *Sandritter's color atlas and textbook of histopathology*. Chicago: Year Book Medical, 1984
18. Gomori JM, Grossman RI, Hackney DB, et al. Variable appearances of subacute intracranial hematomas on high-field spin-echo MR. *AJNR* 1987;8:1019–1026
19. Sostman D, Pope CF, Smith GJW, Carbo P, Gore JC. Proton relaxation in experimental clots varies with method of preparation. *Invest Radiol* 1987;22:509–512
20. Olsen KL, Kucharczyk W, Keyes WD, Norman D, Newton TH. Magnetic resonance characterization of non-flowing intravascular blood. *Acta Radiol [Diagn]* (Stockh) 1986;369:63–66