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AJNR Am J Neuroradiol 1989, 10 (2) 345-350
<http://www.ajnr.org/content/10/2/345>

This information is current as
of November 22, 2024.

MR Imaging of Intracranial Carotid Occlusion

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The MR scans of seven patients with intracranial carotid occlusion (five proved, two presumed) were reviewed to evaluate the MR signal characteristics seen in this disorder. Five patients had clinical signs of cerebral infarction. Of the remaining two patients, one was asymptomatic and the other had a long-standing occlusion and headaches. We correlated the MR findings with cerebral angiography in five patients and with CT scans in six patients. All occluded vessels demonstrated MR signal predominantly isointense to brain on proton-density- T1- and T2-weighted images. Since there is an absence of flow, the MR signal is based on the intrinsic properties of the arterial thrombus and possibly on the chronicity of the occlusion. The pathogenesis and histopathology of intravascular thrombus differ significantly from extravascular hematoma, which accounts for the differences in their MR signal characteristics.

The demonstration of occluded intracranial vessels may solidify the diagnosis of stroke in cases in which clinical and/or CT findings are equivocal. In patients presenting with infarction, an occluded carotid artery by MR may obviate the need for angiography; however, the demonstration of a patent carotid in conjunction with infarction suggests the possibility of an embolus, which may require angiography. We believe that MR is a valuable adjunct to CT in evaluating patients with cerebrovascular infarction.

For the past decade, CT has been the radiologic mainstay in the evaluation of cerebral infarction. In most instances, it can reliably define the location, extent, type (bland vs hemorrhagic), and chronicity of the infarction. However, MR imaging has been shown by several authors to be more sensitive in the detection of cerebrovascular accidents (CVAs), especially early in the course of the infarction [1-8]. This sensitivity is the result of the superior ability of MR imaging to detect an increase in water content within ischemic tissue, as manifested by an increase in the tissue T1 and T2 relaxation times. MR can also assess the presence or absence of intravascular flow in a noninvasive manner [9-12]. The characteristic flow void, seen as absence of signal on T1- and T2-weighted images, indicates vascular patency and rapid flow [9-12]. An absence of this finding on routine MR imaging is strongly suggestive of slow flow or thrombosis. The ability to study intracranial structures in multiple planes with MR improves the ability to diagnose vascular occlusion. This can be an important corroborative finding in the evaluation of cerebral infarction, especially when the clinical or radiologic diagnosis is unclear. In this study, intracranial vascular occlusion in seven patients was diagnosed by MR and correlated with the CT findings in six patients and with angiography in five patients. It was our objective to characterize and explain the MR signal changes resulting from carotid occlusion.

Subjects and Methods

Seven patients with cerebrovascular disease were studied with MR. The group included four women and three men 33 to 65 years old (mean, 52.6 years). The two institutions involved in this study used the 1.5-T Signa* (four cases) and the Vista 1.5-T and 0.5-T† (three

This article appears in the March/April 1989 issue of *AJNR* and the June 1989 issue of *AJR*.

Received February 17, 1988; accepted after revision July 6, 1988.

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AJNR 10:345-350, March/April 1989
 0195-6108/89/1002-0345

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cases) superconducting magnets. Multisection, spin-echo (SE) pulse sequences were obtained in all patients. Proton-density- T1- and T2-weighted images (using dual echoes) were obtained in all patients scanned on the 1.5-T Signa unit; T1- and single-echo T2-weighted images were obtained in patients scanned on the Picker units. The imaging parameters were 500–1000/25–30/2 (TR range/TE range/excitations) for T1-weighted images and 2000/20–35, 60–120/2 for the proton-density- and T2-weighted images, respectively. The section thickness was 5 mm with an interslice gap varying from 0 to 2 mm. The acquisition matrix varied from 128 × 256 to 256 × 256. Axial, coronal, and/or sagittal images were obtained in most patients. The MR scans were done 4 to 60 days after clinical presentation of stroke (mean, 21.4 days). Of the five patients presenting with stroke, three had cerebral angiography. All three of these arteriograms were performed within 1½ days of the MR scans. Another patient with chronic occlusion had angiography several years before his MR scan. The asymptomatic patient had angiography for the purpose of evaluating significant Doppler abnormalities. Six patients had axial CT scans (on either a GE 9800 or 8800 model) prior to their MR scans (range, 2–6 days). Five of the six CT scans were done only without IV contrast administration; the other scan was performed both pre- and postcontrast. All CT scans were obtained with 10-mm contiguous slices.

The MR and CT examinations were assessed with respect to intraparenchymal abnormalities and the evaluation of intracranial vas-

cular patency. Angiography was performed in order to prove or disprove the MR findings and to determine if the vessel was occluded or highly stenosed.

Results

The findings are presented in Table 1. All seven patients in the study had occluded intracranial carotids demonstrated by MR. In all cases, CT failed to demonstrate that abnormality.

Of the five patients (cases 1–4 and 6) presenting with cerebral infarction, the MR showed the characteristic findings of CVA as well as the occluded blood vessels. Three of these patients underwent cerebral angiography, and the occlusions were corroborated in each case. In each of these patients, CT demonstrated findings of cerebral infarction, but no carotid arterial abnormality was evident. We assume that the occlusions took place in association with the patient's symptoms. Cases 1 and 2 clearly occluded at the time of their symptomatology, as these patients each had several MR scans proving this. Patients 3, 4, and 6 most likely occluded at the time of their symptoms, although one could argue that the occlusions

TABLE 1: Summary of Patients with Intracranial Carotid Occlusion

Case No.	Age	Gender	Clinical Information	CT Findings	Angiographic Findings	MR Findings*
1	52	F	Diabetic with mucormycosis, superior orbital fissure syndrome, and left hemiparesis	Infarct in right frontoparietal area in MCA distribution	Not done	Right MCA infarct with occluded right intracavernous ICA (7 days)
2	33	F	Osteosarcoma of ethmoid sinus; history of radiation therapy; presented with left hemispheric CVA syndrome and meningitis	Gyral and cisternal enhancement	Not done	Left basal ganglia hemorrhage and occluded left MCA (4 days)
3	51	M	Hypertensive with right hemispheric infarct	Right basal ganglionic hemorrhagic infarct	Occluded right common carotid artery	Occluded right intracavernous ICA with subacute hemorrhagic basal ganglionic infarct (30 days); adequate collateral circulation to ipsilateral MCA
4	52	F	2-month history of ataxia, dizziness, and left-sided numbness	Right basal ganglionic infarct	Occluded right intracavernous ICA	Right basal ganglionic infarct with occluded right intracavernous ICA (60 days)
5	61	M	Chronic headaches, with history of "reported" left cerebral infarct 16 years ago; Doppler: complete left ICA occlusion	Normal	Complete left ICA 16 years earlier	Occluded left ICA
6	54	F	Right hemispheric infarct	Acute right MCA in basal ganglia, internal capsule, and temporal lobe	Occluded right MCA	Occluded right intracavernous ICA with infarct in basal ganglia, internal capsule, and temporal lobe (16 days)
7	65	M	Asymptomatic with carotid bruit; Doppler: occluded left ICA and 60–80% stenosis of right ICA	Not done	Occluded left ICA	Occluded left ICA; adequate collaterals to ipsilateral MCA

* Information in parentheses = number of days between onset of clinical signs and symptoms and performance of MR imaging.

Note.—MCA = middle cerebral artery; ICA = internal carotid artery; CVA = cerebrovascular accident.

occurred at an earlier time and that the symptoms appeared in relation to problems with the collateral circulation. We believe that this is unlikely in these cases in that all three had angiography and none of them showed any evidence of collateral vessels. The asymptomatic patient (case 7) and the patient who presented with chronic headaches but without stroke (case 5) had MR and angiographically proved vascular occlusion. Patient 5 had a normal CT scan with no evidence of parenchymal or vascular abnormalities. Both of the patients (cases 1 and 2) who did not have angiographic confirmation of carotid occlusion had serial MR scans. The initial scans demonstrated flow voids in the carotid arteries and no parenchymal abnormality, while the subsequent MR examinations clearly showed parenchymal infarctions in conjunction with an occluded ipsilateral carotid.

Three of the five patients presenting with stroke had occlusion of the intracavernous internal carotid artery (ICA) shown by MR. All three had infarction in a middle cerebral artery (MCA) distribution, yet the ipsilateral MCA was patent by MR and/or angiography. Examples of this are illustrated in Figures 1 (case 3) and 2 (case 4). The former was a hemorrhagic infarct in a hypertensive patient; the latter a bland one. The other two patients presenting with stroke had MR manifestations of MCA infarction with occlusion of the ipsilateral MCA. One of these was a bland infarct, while the other was hemorrhagic. Our asymptomatic patient had complete occlusion at the origin of the ICA (Fig. 3, case 7). The patient with chronic headaches also had an occlusion of the left intracavernous ICA branch.

Although small foci of increased or decreased signal were visualized within some of the occluded vessels by MR, the overall signal intensity was predominantly isointense to brain parenchyma. This was consistently found on all T1-, proton-density, and T2-weighted images.

Discussion

In the evaluation of patients with cerebrovascular disease, an understanding of parenchymal abnormalities and of the patency of the major intracranial vessels is important. To our knowledge, a series of intracranial carotid occlusions documented by MR has not been previously reported. MR can evaluate vascular patency noninvasively, whereas CT evaluation of intracranial vascular patency generally requires the administration of IV contrast material, and even when performed in that manner vascular cutoff may not be visualized. A recent article [13] reported that CT of acute CVAs may show increased attenuation in the occluded vessel (secondary to acute thrombosis) on noncontrast images, especially if 5-mm cuts are obtained. The sensitivity of this finding in the acute setting is yet to be determined prospectively. It is important to note that acute stroke patients are usually imaged with 10-mm-thick cuts, so this potential finding may often go undetected. None of our cases demonstrated any definite vascular occlusion by CT. It is fair to point out, however, that most patients imaged with CT in the acute setting of a stroke are done only without IV contrast administration. Typically, the clinicians want to ascertain primarily whether a bleed has occurred. In our series, only one of six patients who underwent CT scanning were given IV contrast. The ability of MR to image intracranial vessels by showing the presence or absence of a "flow void" is a tremendous advantage in evaluating their patency.

The principles of evaluating blood flow by MR have been well described [9-14]. The signal intensity from blood within the vessel lumen is a function of numerous parameters. These include patient-dependent factors, such as the direction of flow, the spatial distribution of velocities across the lumen, and the changes in velocity due to the flow-dependence on

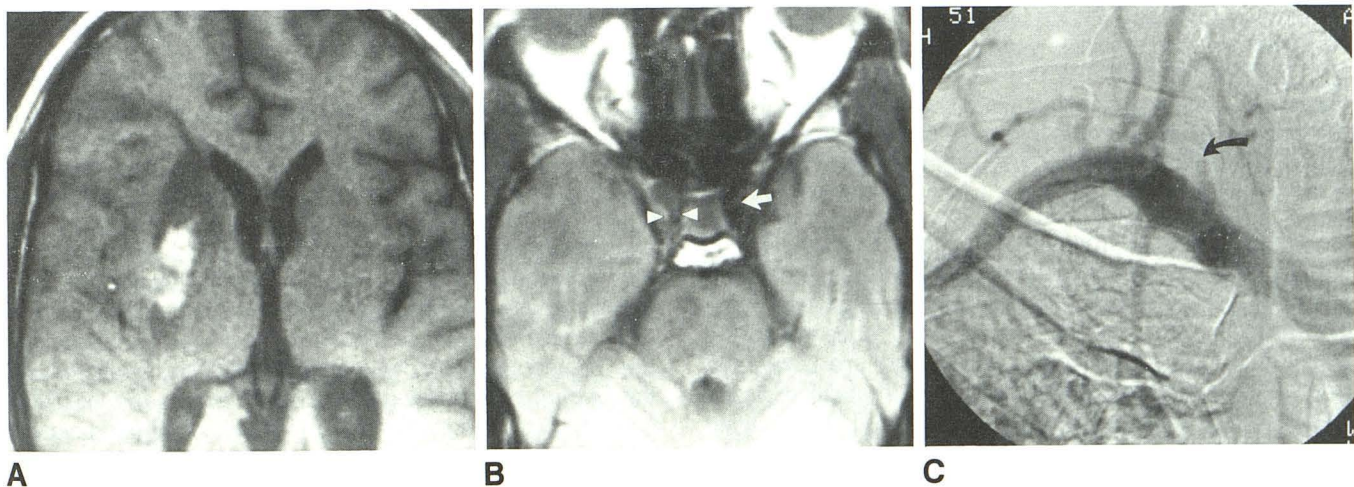


Fig. 1.—Case 3: Hemorrhagic infarct in a right MCA distribution in a patient with occluded common carotid artery.
 A, T1-weighted axial image (600/25) shows subacute hemorrhage in basal ganglia.
 B, Proton-density-weighted axial image (2000/20) shows absence of flow void within isointense intravascular signal in right intracavernous ICA (arrowheads). Compare with patent left intracavernous carotid (arrow).
 C, Digital subtraction angiogram shows stump of occluded right common carotid artery (arrow). Other images (not shown) demonstrated complete occlusion of remainder of right common carotid artery as well as entire internal and external carotids.

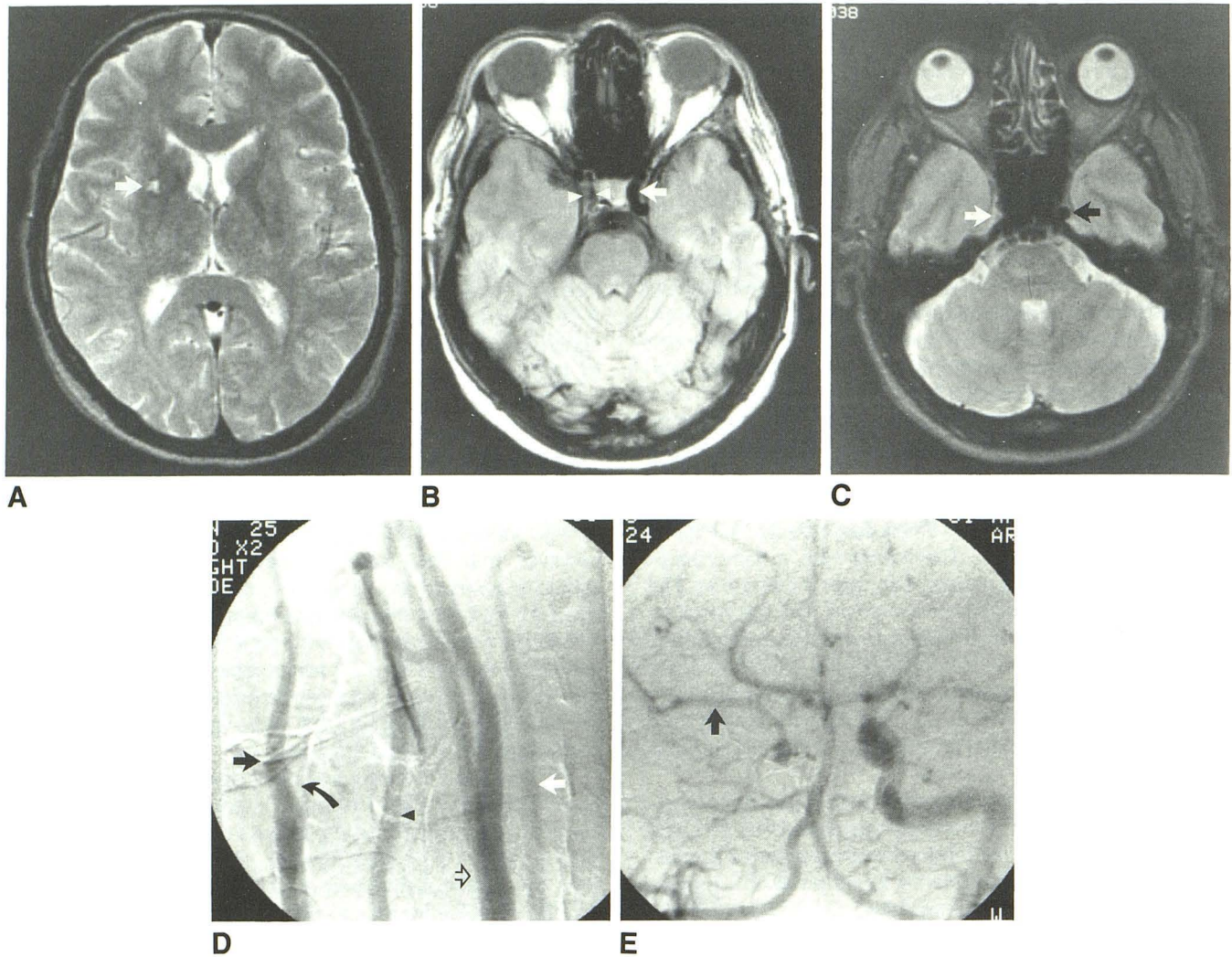


Fig. 2.—Case 4: Basal ganglia infarct in patient with occluded right ICA (60 days after onset of symptoms).

- A**, Axial T2-weighted image (2000/80) shows basal ganglic infarct (arrow).
B, Proton-density-weighted axial image (2000/20) shows isointense right intracavernous ICA (arrowheads) compared with flow void seen in normal left ICA (arrow).
C, T2-weighted axial image (2000/80) through precavernous portion of ICA shows absence of right ICA (white arrow) compared with normal left side (black arrow).
D, Digital arch arteriogram shows occluded right ICA (curved black arrow), normal right external carotid artery (straight black arrow), right vertebral artery (arrowhead), left common carotid artery (open arrow), and left vertebral artery (white arrow).
E, Note occluded right ICA but with collateral circulation to fill right MCA (arrow).

the cardiac cycle. MR sequence-dependent factors include the type of pulse sequence, whether the section is an entry or exit slice (flow-related enhancement), the spin-echo type (odd or even), the orientation of the imaging plane and the phase- and frequency-encoding axes within this plane, and whether cardiac gating is employed [11]. In addition, tissue characteristics such as proton density, T1, and T2 relaxation times also play a role in the contrast observed between the vessel lumen and surrounding tissues.

Rapidly flowing blood normally gives a characteristic signal void as a result of a combination of "time-of-flight" effects and turbulence. The "time-of-flight" effect results from the motion of blood through a slice during the time between the 90° and 180° pulses in the sequence. Material that originally receives the 90° excitation pulse moves out of the slice, does not

experience the 180° refocusing pulse, and hence does not form an echo. In addition, material that moves into the slice between the two pulses only experiences the refocusing pulse and likewise does not contribute to the signal. Turbulent flow also contributes to the loss of signal intensity by increasing the amount of dephasing of the signal across the individual pixel. As dephasing increases, the small contributing signals add together less coherently and the received signal decreases.

In all our cases, there was absence of the "flow void" that one usually sees in the lumen of normally flowing arteries. This is secondary to the lack of "time-of-flight" effects and turbulence in the occluded vessels. The intravascular signal seen in all the occluded vessels (in multiple planes) was basically isointense to normal brain on T1-, proton-density,

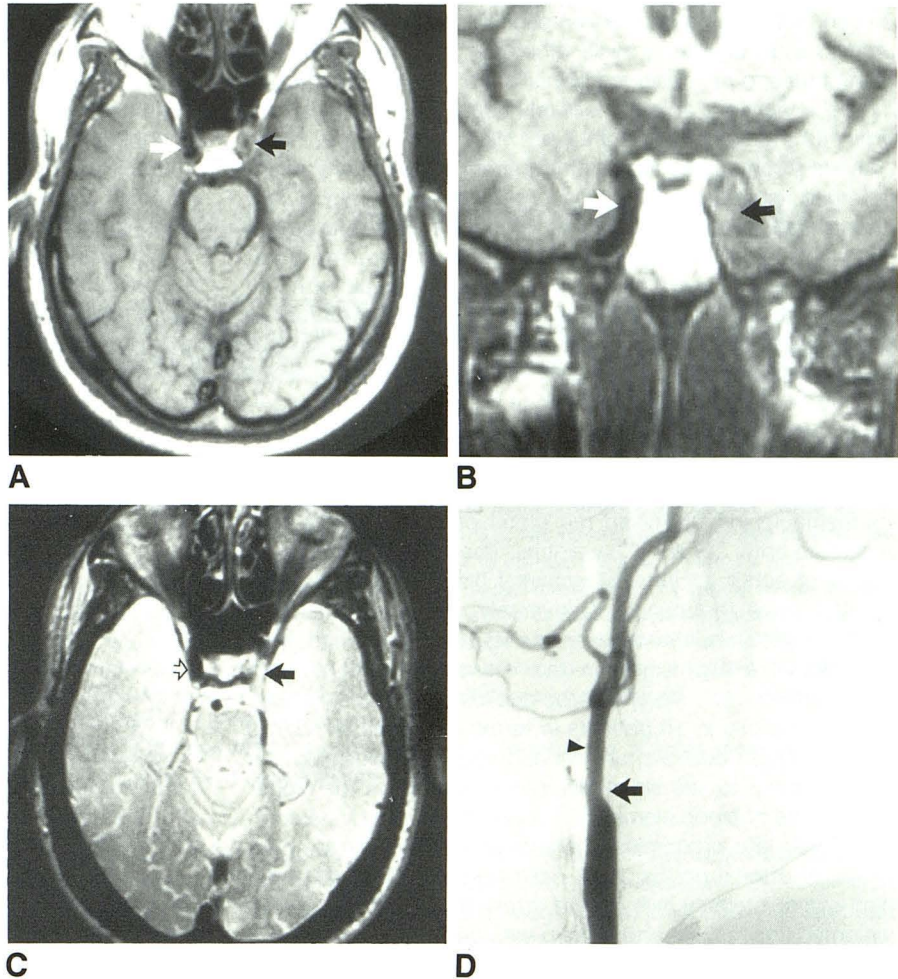
Fig. 3.—Case 7: Occlusion of left ICA in asymptomatic patient.

A, T1-weighted axial image (691/26) shows lack of flow void in left ICA with signal intensity equal to surrounding brain parenchyma (*black arrow*). Normal flow void in right carotids indicated by *white arrows* in A and B.

B, Coronal T1-weighted image (691/26) shows findings similar to those in A (*arrows* are also similar).

C, Axial T2-weighted image (1991/60) again shows predominantly isointense signal in occluded vessel (*closed arrow*) compared with normal right side (*open arrow*).

D, Digital subtraction angiogram of the left common carotid artery injection shows total occlusion at origin of left ICA (*arrow*). Note normal external carotid artery (*arrowhead*).



and T2-weighted images. Likewise, none of the occluded carotids showed hyperintense intraluminal signal. Intravascular hyperintensity can be seen secondary to flow-related enhancement (particularly for entry slices seen with a short TR), diastolic pseudogating, the use of motion-compensating gradients, cardiac gating, and symmetric even-echo rephasing [12]. All these mechanisms may produce increased intraluminal signal in normal veins, where flow is relatively slow compared with arterial flow. In our experience, none of these will produce increased signal in a normally flowing carotid artery. In fact, flow-related enhancement, diastolic pseudogating, and even-echo rephasing require some flow in order to produce a bright signal [9]. No second-echo rephasing was seen in our cases.

In cases 3–6, in which a second echo was utilized, the signal intensity in the occluded carotids was identical (isointense) on both the first and second echoes. We can presume, therefore, that if one sees a high signal in a carotid artery, diminished flow (secondary to a high-grade stenosis or to a low cardiac output state) is present. Since all our MR scans were done at least 4 days after the clinical event, it is presumed that slow flow progressed to complete occlusion in most, if not all, these cases. It is postulated that MR performed within the first 24 hr after a stroke may demonstrate increased signal intensity intravascularly as long as

flow, though diminished, is maintained. Certainly, a gradual thrombotic occlusion would be more likely to produce these findings than would an acute embolic occlusion. Further investigation of these issues may elucidate the flow dynamics of stroke early in its evolution. We therefore contend that when carotid arteries contain neither a flow void nor a high signal, that flow is neither normal nor sluggish. The isointense signal represents stationary protons in an occluded vessel. This is substantiated in our angiographically proved cases in which all demonstrated occlusion rather than stenosis. On the basis of the progression of both intraluminal and parenchymal abnormalities, we do believe that our two patients who had serial MR scans (cases 1 and 2) would have shown a similar angiographic picture. These factors support the conclusion that the observed signal intensities within these occluded vessels is a function of the proton-density and relaxation characteristics of the intravascular thrombus rather than flow, which would dominate the observed signal intensity if these vessels were patent. The proton-density and relaxation characteristics of arterial thrombus are dependent on its histopathologic composition.

Arterial thrombi are composed of alternating layers of fibrin and platelets irregularly united with very small quantities of red blood cells and coagulated blood. These laminated layers are known as the lines of Zahn. Arterial thrombi are known

as white thrombi because of their lack of red cells within the precipitated fibrin network. In the typical thrombotic arterial occlusion, atherosclerotic changes fill the majority of the lumen, with the remainder representing thrombus. It is this combination of arterial wall, atheroma, and thrombus that contributes to the observed signal in the occluded arteries. In no case were we able to definitely separate the arterial wall from its intraluminal contents on MR. On the other hand, venous thrombus as well as intraparenchymal or any extravascular hematoma is composed primarily of red blood cells that simply coagulate, much as blood would clot in a test tube [14]. The MR signal characteristics of intracranial hematomas have been well described on high-field-strength systems [15]. Since intraparenchymal hematomas and venous thrombus are composed mostly of red cells, the MR signal characteristics over time depend on the relaxation times of oxidation breakdown products of hemoglobin (i.e., deoxy-hemoglobin, methemoglobin, and hemosiderin). The occluded carotid vessels, which contain few red cells, do not have any significant contribution from these substances. For example, methemoglobin from lysed red blood cells would give high signal on both T1- and T2-weighted images in the subacute phase of either an intraparenchymal bleed or in a superior sagittal sinus thrombosis. Conversely, the relative lack of methemoglobin in intravascular arterial thrombus will not tend to produce a high signal in the vessel. Heier et al. [16] described the MR signal characteristics of intracranial vascular occlusions in 16 patients in terms of chronicity but did not distinguish between arterial and venous occlusions. We would expect to see signal differences based on the higher percentage of blood elements in a venous thrombus as compared with the predominant intimal atheroma seen in most cases of arterial occlusion. A case report [17] described a high signal intensity in a carotid artery illustrated on the T2-weighted images. An arteriogram was performed, and it was thought from this that thrombus was present in the left internal carotid artery. In addition, there was cross-filling of the left A1 and M1 segments via the anterior communicating artery from the right common carotid artery injection. We believe that the high signal most likely was secondary to retrograde filling of the supraclinoid and cavernous segments of the internal carotid artery with slow flow. Another possibility would be an unusually high content of methemoglobin in an arterial occlusion in relation to the amount of atheroma narrowing the lumen.

We have indicated that arterial occlusion gives, for the most part, an isointense MR signal on all spin-echo sequences. There are, however, small focal areas of either increased or decreased signal intensity scattered within several of the occluded vessels. We believe that focal punctate areas of hyperintensity may represent small amounts of methemoglobin in a subacute thrombus. Conversely, we postulate that small areas of hypointensity may be the result of fibrin and hemosiderin deposits in a chronic, organized thrombus. Both these substances are seen in abundance in chronic thrombi [14] and both may cause diminished MR signal. We do not believe that the small foci of intraluminal hypointensity are secondary to small collateral vessels, since the shape of the low signals are not serpiginous and the angiographic cases did not indicate the presence of any collaterals within or adjacent to the cavernous sinuses.

Several important clinical inferences can be made on the basis of these MR findings. First, in cases in which the clinical signs and/or CT are equivocal, the presence of an occluded carotid artery by MR may serve as an important adjunct in making the diagnosis of cerebral infarction. Second, in patients presenting with CVAs, the presence of an occluded ipsilateral carotid by MR may obviate the need for carotid angiography. Conversely, in patients demonstrating a patent internal carotid artery by MR in conjunction with a cerebral infarction, angiography may be required to look for a possible embolic source.

Until such time as specialized "MR angiography" [18, 19] sequences become available for widespread clinical use, we believe that spin-echo MR can be used to diagnose carotid occlusion. By MR, intravascular signal isointense to brain on proton-density-, T1-, and T2-weighted images strongly suggests this diagnosis. As a result of the superior ability of MR to evaluate both intraparenchymal and intravascular abnormalities, we believe that this imaging technique should serve as an important adjunct to CT in the evaluation of patients with cerebrovascular disease.

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