AJNR

Assessment of carotid artery patency on routine spin-echo MR imaging of the brain.

J I Lane, A E Flanders, H T Doan and R D Bell

AJNR Am J Neuroradiol 1991, 12 (5) 819-826
http://www.ajnr.org/content/12/5/819

Assessment of Carotid Artery Patency on Routine Spin-Echo MR Imaging of the Brain

John I. Lane[1]
Adam E. Flanders[1]
Huynh T. Doan[1]
Rodney D. Bell[2]

We retrospectively reviewed the routine spin-echo MR studies of the brain in 12 patients with 13 angiographically demonstrated occlusions and in 14 patients with 16 high-grade stenoses of the carotid arteries. Intraluminal signal that was isointense with adjacent brain on long TR/short TE and long TR/long TE images was 100% specific for atherosclerotic occlusion. Of the 13 proved occlusions, six (46%) had significant degrees of hyperintense intraluminal signal indistinguishable from that observed consequent to slow flow distal to high-grade stenoses. MR detected only five (31%) of the 16 proved high-grade stenoses.

Normal flow void does not exclude significant extracranial carotid stenosis. Occlusion cannot always be distinguished from high-grade stenosis when hyperintense intraluminal signal is encountered. However, a reliable diagnosis of atherosclerotic occlusion can be made when isointense intraluminal signal is observed.

***AJNR* 12:819–826, September/October 1991; *AJR* 157: December 1991**

Several recent reports have described the loss of normal flow void within the proximal intracranial arteries distal to extracranial carotid or vertebral lesions on spin-echo (SE) MR imaging of the brain [1–6]. Hyperintense intraluminal signal has been interpreted as thrombus in an occluded vessel or, alternatively, slow flow distal to a high-grade stenosis. Katz et al. [2] recently reported seven cases of atherosclerotic occlusion that had an intraluminal signal that was predominantly isointense with adjacent brain, implying that this appearance is specific for occlusion. The rationale behind their statement was based on the pathologic nature of atherosclerotic occlusion, which is composed primarily of atherosclerotic plaque and fibrinous clot, containing a paucity of RBC thrombus. Disputing the claim that bright intraluminal signal represents thrombus, these authors suggested that markedly hyperintense signal should be interpreted as slow flow.

Heinz et al. [3] coined the term *partial flow void* to describe the intraluminal signal changes distal to extracranial high-grade carotid stenoses that they observed on routine SE sequences of the brain. Their report raised the hope that extracranial high-grade stenoses could be detected reliably on MR studies of the brain [7]. Brant-Zawadzki [4] subsequently reported a series that included six cases of internal carotid artery (ICA) stenoses with normal flow void and two cases of isointense intraluminal signal in the carotid siphon in the absence of complete occlusion.

It is difficult to ascertain the diagnostic value of these intraluminal signal changes without a critical assessment of their specificity and sensitivity in the detection of occlusive and preocclusive disease. Thus, the purpose of our study was to determine the utility of these signal changes in the diagnosis of compromised arterial flow. Our objectives were to determine whether angiography can be obviated in cases of suspected occlusion and to establish whether the presence of normal flow void can be used to exclude significant extracranial stenoses.

Received October 18, 1990; returned for revision December 17, 1990; revision received February 19, 1991; accepted February 27, 1991.

[1] Department of Radiology, Thomas Jefferson University Hospital, 10th and Sansom Sts., Room 1009 Main, Philadelphia, PA 19107. Address reprint requests to J. I. Lane.

[2] Department of Neurology, Thomas Jefferson University Hospital, Philadelphia, PA 19107.

0195–6108/91/1205–0819

Materials and Methods

Two hundred thirty-six consecutive patients who had both carotid angiography and SE MR imaging of the brain between January 1987 and January 1990 were considered as candidates for this study. Patients were excluded if there was any significant change in their neurologic state between examinations or if either examination was technically unsatisfactory. Inclusion in this retrospective study required angiographically demonstrated total occlusion of the common carotid artery (CCA) or ICA or stenosis of either artery of greater than 90%. The 90% criterion was based on the degree of stenosis required to produce signal changes in an animal model developed by Heinz et al. [3]. A 90%-diameter stenosis was determined by comparing the narrowest diameter of the artery with the normal caliber of the vessel just distal to the lesion. Following the application of these inclusion criteria, 12 patients (13 vessels) with arteriographic occlusion and 14 patients (16 vessels) with high-grade stenosis were identified. Twenty age-matched control subjects with no hemodynamically significant disease were selected from the study population.

Angiograms were obtained by using intraarterial digital subtraction or conventional film-screen and subtraction techniques. For selective digital subtraction studies, the CCA was injected with 6 ml of 30% sodium diatrizoate at a rate of 4 ml/sec. For conventional studies, 12 ml of 60% sodium diatrizoate was injected at 10 ml/sec for CCA injections.

The MR studies were performed on a 1.5-T Signa scanner (General Electric, Milwaukee, WI). Pulse sequences consisted of short TR/short TE sagittal images, 600/20/1 (TR/TE/excitations), with a 256 × 192 matrix and dual-echo long TR axial and coronal images, 2000/20–80/1, with a 256 × 192 matrix. The slice thickness was 5 mm with a 2.5-mm interslice gap. No gradient-moment nulling or presaturation pulses were used in the studies performed at our institution. Gradient-moment nulling was used in two of three MR studies that had been submitted for consultation from outside institutions and included in our retrospective review. All studies were performed with cardiac gating.

The SE MR studies were reviewed independently of the angiographic studies by three neuroradiologists. Evaluation of the ICAs included assessment of the presence of normal flow void, the configuration of intraluminal signal when normal flow void was absent, and symmetry in the size of the ICAs. The ICAs were scrutinized from the skull base to the subclinoid segments. Any signal changes that were limited to entry or exit slices were excluded. All signal abnormalities had to be seen consistently from the skull base to the subclinoid segments in two orthogonal planes. By virtue of the sequences used, these observations were primarily made on dual-echo long TR images.

Each case was then classified as occlusion, high-grade stenosis, or normal on the basis of the observed intraluminal signal characteristics. The two cases of carotid dissection were excluded from this portion of the study in an effort to avoid potential confusion between intraluminal methemoglobin (consequent to dissection) and hyperintense intraluminal signal (slow flow) distal to a high-grade stenosis. Operating on the premise that atherosclerotic occlusion should appear as isointense intraluminal signal [2], the sole criterion for total occlusion was replacement of normal flow void with intraluminal signal that was predominantly isointense with adjacent brain. The MR criteria for high-grade stenosis included at least one of the following: (1) a reduction in vessel diameter with preservation of flow void; (2) a partial flow-void phenomenon, as described by Heinz et al. [3]; or (3) replacement of flow void with hyperintense intraluminal signal. If no intraluminal signal changes were noted, the study was considered normal.

To determine the reliability of these criteria, truth tables were constructed for both carotid occlusions and carotid stenoses as diagnosed by SE MR imaging. Evaluating the criteria for carotid occlusion required that high-grade stenosis and normals be considered together in the nonoccluded category. Likewise, evaluating the criteria for high-grade stenosis required that occlusions and normals be considered together in the nonstenotic category.

Results

The occlusion group comprised seven men and five women 41–73 years old (median age, 62), the high-grade stenosis group comprised seven men and seven women 47–80 years old (median age, 60), and the control group comprised 12 men and eight women 33–80 years old (median age, 65). The mean interval between MR and angiography was 3 days. All occlusive and stenotic lesions had an atherosclerotic origin except for two that resulted from spontaneous carotid dissection. One lesion resulted in complete occlusion and the other in marked narrowing of the internal carotid lumen.

Nine of 13 occlusions and 15 of 16 high-grade stenoses were located in the proximal ICA. The distal CCA was the site of disease in four occlusions and one high-grade stenosis.

Intraluminal Signal Changes

The MR signal changes that we observed in both angiographically occluded and stenotic groups are listed in Table 1. A schematic display of the patterns of intraluminal signal observed is presented in Figure 1.

Normal intraluminal flow void was demonstrated in the ICAs of all 20 patients in the control group. The partial-flow-void phenomenon was identified in only three of 16 high-grade stenoses (Fig. 2). One case of an occluded left CCA also demonstrated a partial flow void in the left ICA (Fig. 3). Further review of the arteriogram revealed that the ICA reconstituted from retrograde flow through the left external carotid artery via left vertebral collaterals.

Intraluminal signal was predominantly isointense with brain in six (46%) of 13 occlusions (Figs. 4 and 5). This pattern was not identified in any of the cases of high-grade stenosis.

TABLE 1: Intraluminal Signal Changes on Spin-Echo MR Imaging of Angiographically Demonstrated Occlusion or Stenosis of the Carotid Artery

MR Signal	Angiographic Finding		
	Occlusion	High-Grade Stenosis	Normal
Partial flow void	1 (8)	3 (19)	0
Heterogeneous hyperintensity			
Hyperintense peripherally with central isointense focus	3 (23)	0	0
Central hyperintense focus with peripheral isointensity	3 (23)	1 (6)	0
Eccentric hyperintensity	0	1 (6)	0
Homogeneous hyperintensity	0	1 (6)	0
Isointensity	6 (46)	0	0
Normal	0	10 (63)	20 (100)
Total	13 (100)	16 (100)	20 (100)

Note.—Values represent no. (%) of vessels.

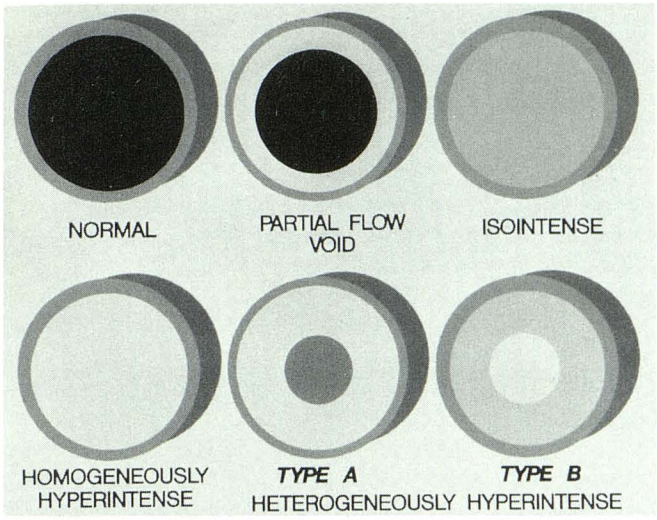

Fig. 1.—Schematic representation of observed intraluminal signal changes on long TR/short TE and long TR/long TE sequences (2000/20–80/1). *Black* represents flow void, *white* represents signal hyperintense relative to brain, and *gray* represents signal isointense with brain.

Hyperintense intraluminal signal had variable appearances on SE MR images; homogeneous and heterogeneous patterns of hyperintensity were observed (Figs. 6–8). These patterns of hyperintense signal were not pathognomonic for occlusion or for high-grade stenosis. One case of hyperintense intraluminal signal was eccentrically located within the vessel lumen.

The results of our effort to predict stenosis or occlusion on the basis of the MR appearance in those patients with atherosclerotic disease are listed in Table 2. The 20 normal control subjects are also included. Statistical analysis of these results is compiled in Table 3. Of particular note is the poor sensitivity of MR in the detection of proximal high-grade stenoses (33%). When isointense intraluminal signal was used as the sole criterion for atherosclerotic occlusion, a specificity of 100% was obtained. Applying this criterion, however, resulted in a poor sensitivity (50%), since five atherosclerotic occlusions also demonstrated some degree of hyperintense intraluminal signal. There were no false-positive MR diagnoses of occlusion or stenosis in the control group.

Two cases of spontaneous ICA dissection were also included in this study. In one case, which resulted in total occlusion of the proximal ICA, heterogeneously hyperintense signal was seen that was indistinguishable from that produced by atherosclerotic disease on dual-echo long TR images. The second case of dissection, which resulted in marked stenosis of the proximal ICA, produced a partial flow void that also was indistinguishable from that produced by atherosclerotic stenosis.

Discussion

High-velocity signal loss (flow void) within the vessel lumen on SE sequences is a reliable indicator of vessel patency. When this signal loss is replaced by varying degrees of increased intraluminal signal, determination of vessel patency becomes more difficult. Several technical causes of intraluminal signal must be excluded before luminal compromise or occlusion should be considered; among these are flow-related enhancement, diastolic pseudogating, even-echo rephasing, and the use of flow-compensation techniques (gradient-moment nulling). Flow-related enhancement typically is encountered in the first few entry or exit slices of the imaged volume. Familiarity with the location and appearance of flow-related enhancement allows differentiation from pathologic signal changes [8]. Diastolic pseudogating can be eliminated with the use of cardiac gating. Even-echo rephasing can be eliminated by using odd rather than even TEs on long TR se-

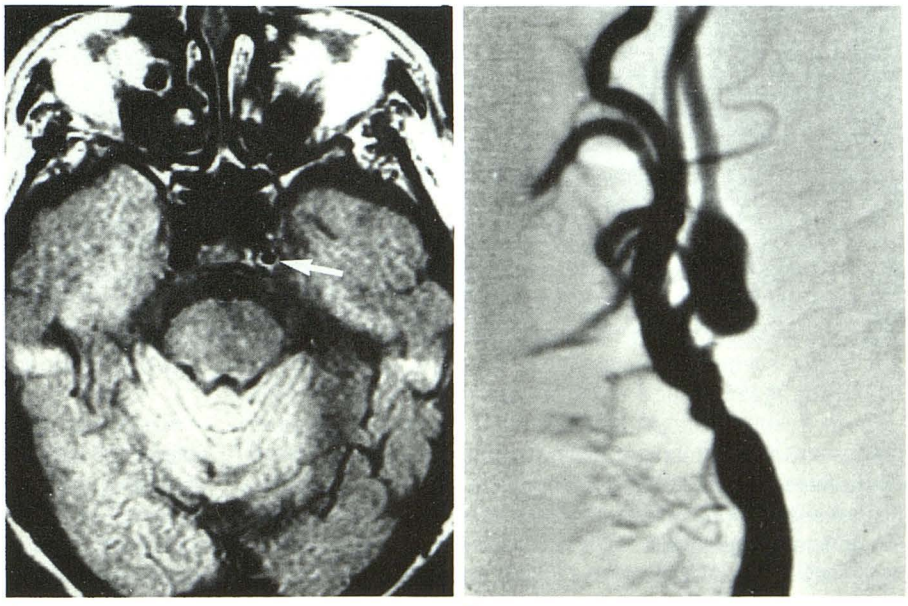

Fig. 2.—Partial flow void.

***A*, Long TR/short TE image (2000/20/1) shows decreased luminal diameter with peripheral rim of increased signal (partial flow void) in siphon (*arrow*) of proximal left internal carotid artery (ICA). Note normal flow void in contralateral ICA.**

***B*, Selective injection of left common carotid artery reveals severe stenosis at origin of left ICA.**

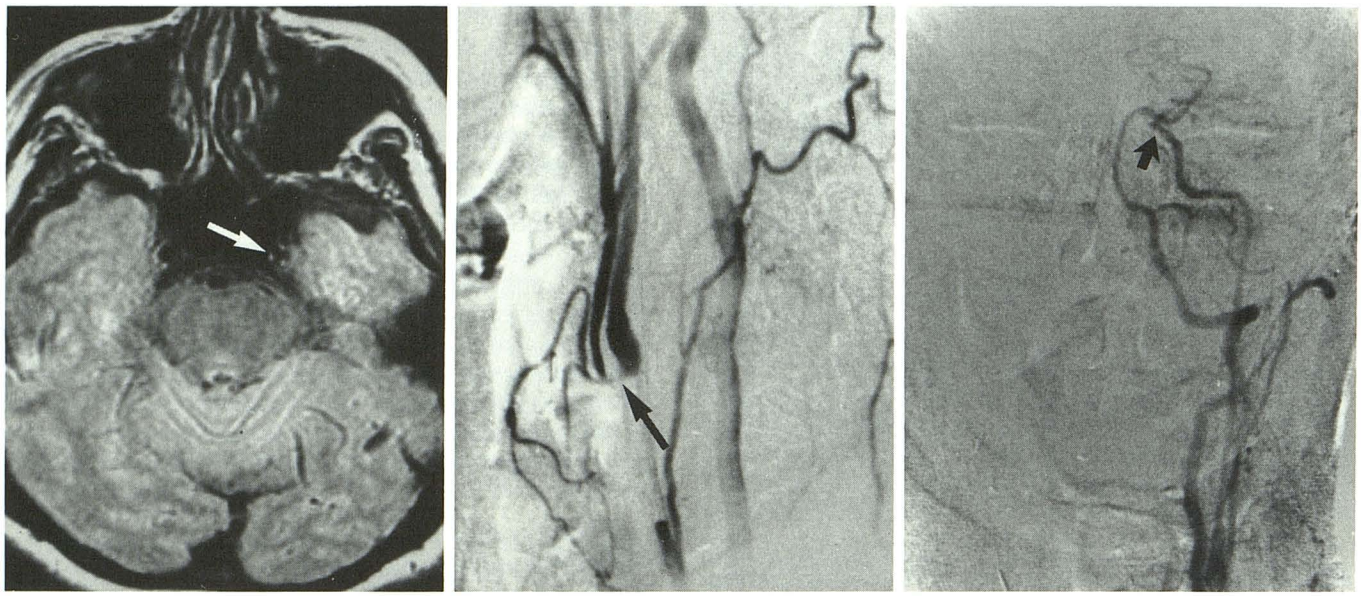

Fig. 3.—Partial flow void.

***A*, Long TR/short TE image (2000/20/1) shows partial flow void in siphon of proximal left internal carotid artery (ICA) despite angiographically demonstrated complete occlusion of left common carotid artery (CCA). Note similar but less dramatic changes in right ICA of patient studied 3 days after right carotid endarterectomy.**

***B*, Lateral view of selective left vertebral injection shows collateralization to distal left CCA bifurcation and antegrade flow in left ICA (*arrow*).**

***C*, Frontal view after selective injection of left vertebral artery shows opacification of left internal carotid siphon (*arrow*).**

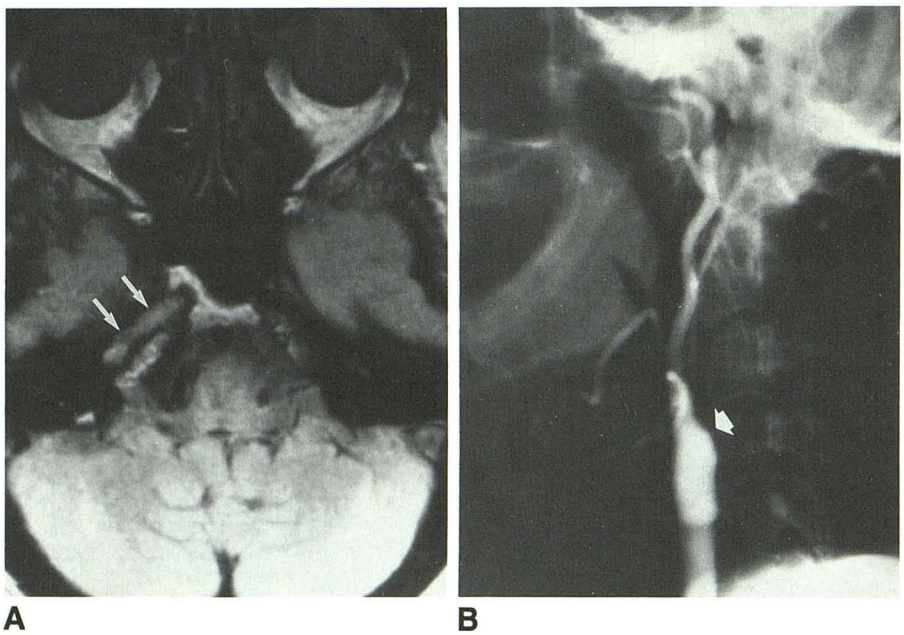

Fig. 4.—Isointense intraluminal signal.

***A*, Long TR/short TE image (2000/20/1) shows complete absence of flow void in horizontal portion of carotid canal (*arrows*) with intraluminal signal isointense with adjacent brain.**

***B*, Selective injection of right common carotid artery shows complete occlusion at origin of right internal carotid artery (*arrow*).**

quences. Gradient-moment nulling was used in only two of 46 studies reviewed in this investigation.

Intraluminal signal changes associated with extracranial ICA and vertebral stenosis and occlusion have been reported previously [1–6]. Alvarez et al. [1] first described the SE MR appearance of an ICA occlusion. They described absence of flow void and very intense intraluminal signal on all pulse sequences in the cavernous portion of the ICA. Angiography demonstrated total occlusion just distal to the origin of the ICA. Katz et al. [2] reported a series of seven atherosclerotic occlusions, all of which contained intraluminal signal that was predominantly isointense with brain on both long TR and short TR sequences. They suggested that the isointense quality of the intraluminal signal was consequent to a paucity

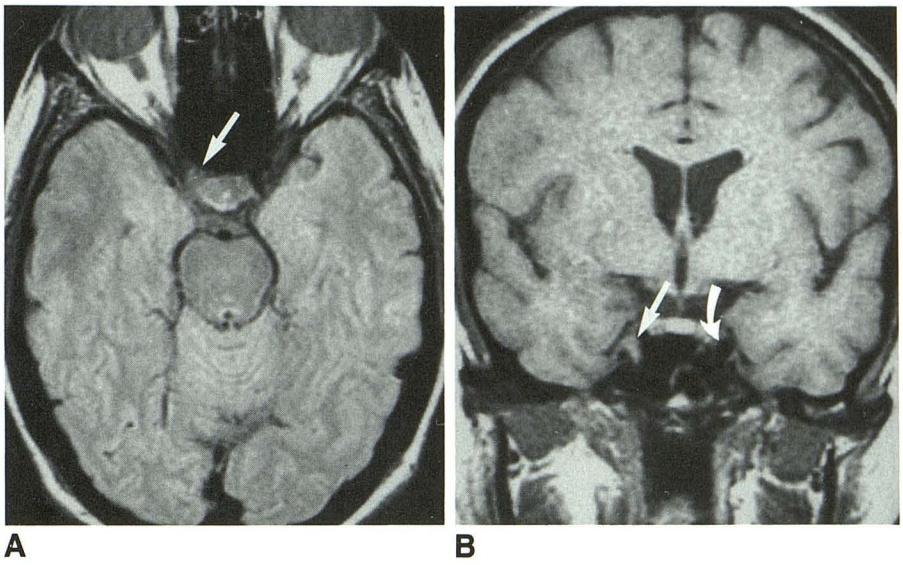

Fig. 5.—Isointense intraluminal signal in totally occluded right internal carotid artery.

***A*, Long TR/short TE axial image (2000/20/1) shows intraluminal signal isointense with adjacent brain (*arrow*).**

***B*, Short TR/short TE coronal image (600/20/2) shows isointense intraluminal signal in proximal cavernous portion of right internal carotid artery (*straight arrow*). This appearance suggests a lack of methemoglobin within occluding thrombus; compare with normal flow void in left internal carotid artery (*curved arrow*).**

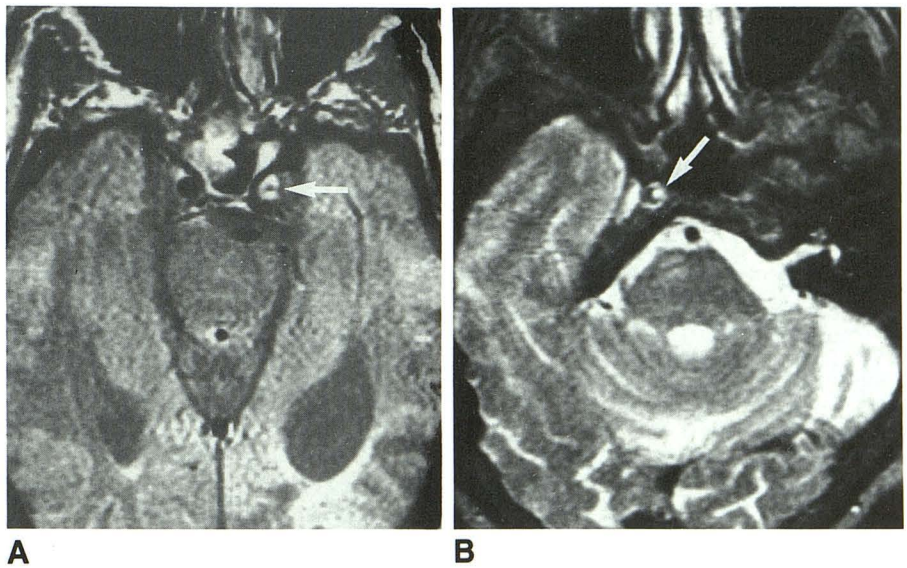

Fig. 6.—Patterns of heterogeneous hyperintensity in presence of total occlusion.

***A*, Long TR/short TE image (2000/20/1) shows hyperintense intraluminal signal peripherally with central focus of lower-intensity signal (*arrow*) in a patient with complete occlusion of left internal carotid artery.**

***B*, Long TR/long TE image (2000/80/1) in a patient with a totally occluded right common carotid artery. Central focus of hyperintensity is surrounded by lower signal intensity (*arrow*).**

of RBCs within the thrombus, which is composed predominantly of fibrin and platelets. They predicted that if markedly hyperintense intraluminal signal was encountered (as in the case reported by Alvarez et al.), this would be most consistent with slow flow (antegrade or retrograde) rather than atherosclerotic occlusion. Brant-Zawadzki [4] has disputed the specificity of isointense intraluminal signal. His series included two cases of isointense signal in the carotid siphon, reportedly produced by slow flow.

Several important differences in technique should be considered before comparing the results of our study with those of Brant-Zawadzki [4]. Of the 100 cases reviewed by Brant-Zawadzki, 22 studies had been performed on a 0.5-T magnet with a 10-mm slice thickness. The author did not specify which of the 14 studies in which carotid arteriograms were abnormal were obtained on the low-field unit. In our estimation, large voxel size could become a significant problem when using a 10-mm slice thickness to evaluate the carotid canal and siphon. If this vessel were marginally patent, volume averaging could cause a small flow void to appear as isointense signal. A potentially more important difference in technique involves the use of motion-compensation gradients in studies performed on high-field units. A well-known disadvantage to the use of gradient-moment nulling is the replacement of normal flow void with hyperintense intraluminal signal in a slow-flow state. Although the use of spatial presaturation can reduce the amount of intraluminal signal produced by gradient-moment nulling, the signal is not always eliminated entirely by using this additional technique [9]. It is conceivable, therefore, that the combination of gradient-moment nulling and spatial presaturation could produce gray (isointense) intraluminal signal in a patent vessel [9]. Although this is not a problem with normal high-velocity flow in the carotid arteries, this technique may cause a marginally patent vessel to appear

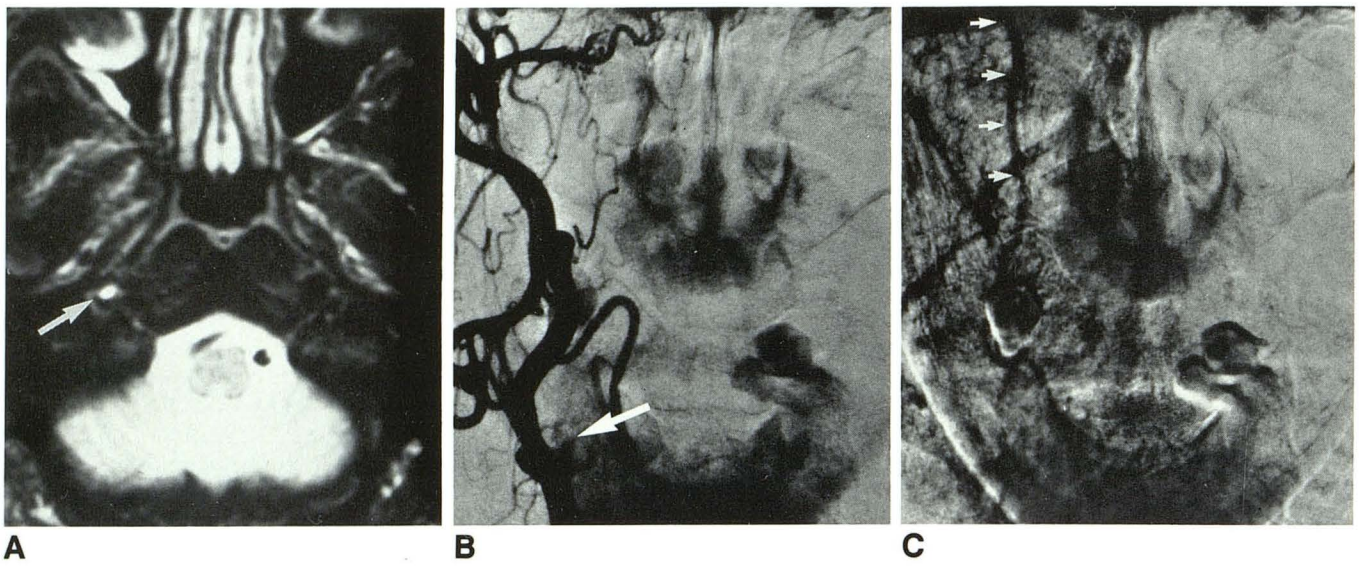

Fig. 7.—Homogeneous hyperintensity distal to severe stenosis.
A, Long TR/long TE image (2000/80/1) with gradient-moment nulling without presaturation shows markedly hyperintense intraluminal signal in distal cervical portion of right internal carotid artery (ICA) (*arrow*).
B, Early arterial phase of right common carotid injection suggests total occlusion of right ICA (*arrow*).
C, Late arterial phase shows slow antegrade flow of contrast material into right ICA (*arrows*).

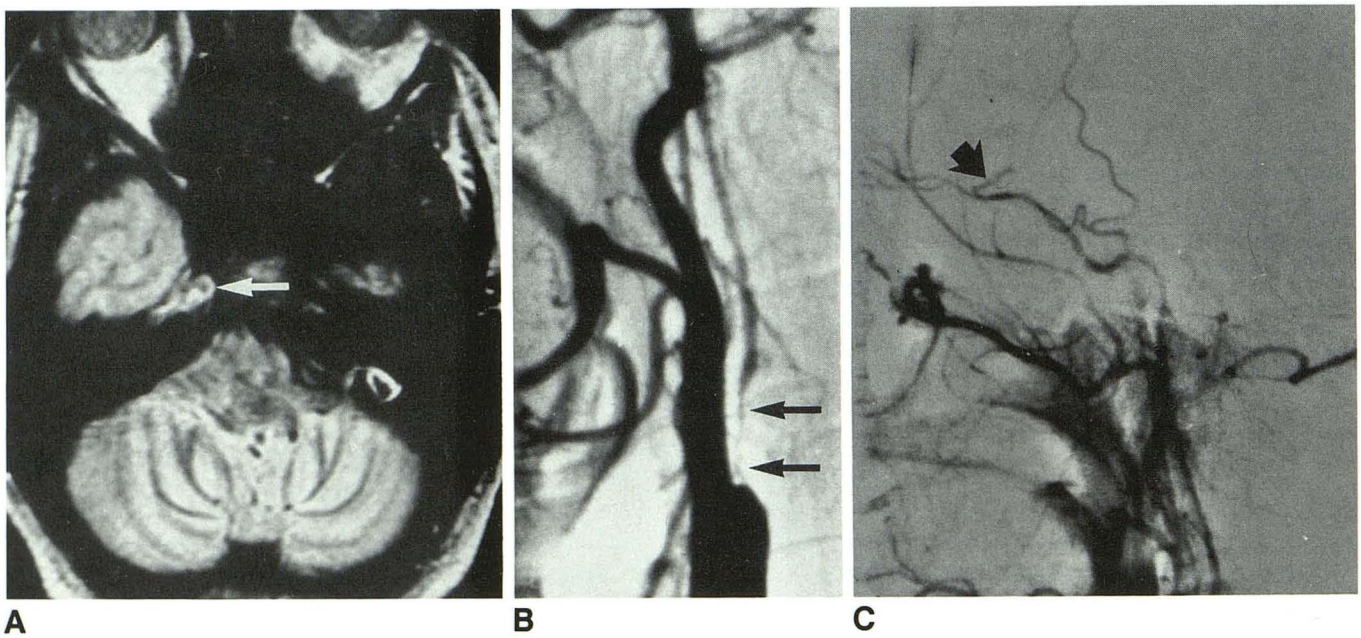

Fig. 8.—Heterogeneous hyperintensity in presence of high-grade stenosis.
A, Long TR/short TE presaturation image (2000/20/1) with gradient-moment nulling shows central focus of isointensity surrounded by hyperintense signal in distal petrous segment of right internal carotid artery (ICA) (*arrow*).
B, Selective injection of right common carotid artery shows angiographic "string sign" involving right ICA (*arrows*).
C, Lateral projection of late arterial phase shows opacification of right carotid siphon (*arrow*).

isointense. Our study included only two studies with gradient-moment nulling. We observed markedly hyperintense intraluminal signal in one case of angiographically demonstrated high-grade stenosis when MR was performed with gradient-moment nulling (without spatial presaturation) (Fig. 7). In a similar case of angiographic high-grade stenosis, MR performed with gradient-moment nulling and spatial presaturation demonstrated heterogeneously hyperintense signal with a small central focus of isointensity (Fig. 8). The other three cases with a similar appearance on MR performed with cardiac gating appeared occluded only on angiography. Admittedly, the advantages of using gradient-moment nulling to

TABLE 2: Angiographic and MR Assessment of Patency of the Carotid Arteries in Occlusion and Stenosis

MR Interpretation	Angiographic Finding		
	Occlusion (n = 12)	Stenosis (n = 15)	"Normal" (n = 20)
Occlusion (n = 6)	6	0	0
Stenosis (n = 11)	6	5	0
Normal (n = 30)[a]	0	10	20

Note.—The two cases of spontaneous carotid dissection are not included in this table to avoid potential confusion between intraluminal methemoglobin consequent to dissection and hyperintense intraluminal signal (slow flow) distal to a high-grade stenosis.

[a] Comprises 10 patients from the study group and 20 normal control subjects.

reduce or eliminate artifacts created by pulsatile flow far outweigh the disadvantages of artifactual intraluminal signal. If carotid occlusion is suspected on the basis of scans obtained with gradient-moment nulling and presaturation, additional MR angiographic sequences may be useful for confirmation.

In our experience, the partial-flow-void phenomenon was a reliable indicator of decreased flow within a partially collapsed lumen. Although Brant-Zawadzki [4] concluded that this appearance in the normal patient too often was simulated by volume averaging in the horizontal portion of the petrous segment, we did not find this to be an inconsistency, as we required the presence of the same signal abnormalities in the proximal intrapetrous and subclinoid segments. The use of a partial flow void as a criterion for diagnosing extracranial high-grade stenosis was quite insensitive, being present in only three of 16 cases. It should be remembered that partial flow void is not specific for high-grade stenosis, since it can also be seen within the reconstituted vessel distal to a total occlusion. Our results clearly indicate that the appearance of normal flow void on SE MR images of the brain by no means excludes significant (even preocclusive) atherosclerotic stenosis of the extracranial vessels. Brant-Zawadzki had reached a similar conclusion. No correlation was established between the quality of intraluminal signal changes and the degree of stenosis. This is not surprising, since the dynamics of blood flow are determined by more than the cross-sectional area of a stenotic lumen. Further in vivo studies correlating Doppler parameters, such as velocity and turbulence, with signal changes on SE MR are needed to clarify this relationship.

In our experience, the finding of isointense intraluminal signal from the skull base to the supraclinoid bifurcation on all pulse sequences is specific for complete occlusion. We concur with Katz et al. [2] that such an appearance can obviate invasive angiographic confirmation in the appropriate clinical setting. T1-weighted sequences in other than the sagittal plane were obtained in only one case of carotid occlusion in our series (Fig. 5). No evidence of intraluminal methemoglobin was evident in this single case. This would appear to support the contention expressed by Katz et al. that thrombus distal to an atherosclerotic occlusion is not composed of significant amounts of methemoglobin.

Six of the 12 atherosclerotic occlusive lesions had significant degrees of hyperintense intraluminal signal that prevented us from making the diagnosis of complete occlusion. It should be noted, however, that six of eight cases of heterogeneously hyperintense intraluminal signal proved to be atherosclerotic occlusions on angiography. Since the onset of atherosclerotic occlusion was not clinically apparent in any of the cases reviewed, no correlation was possible between the age of occlusion and the intraluminal signal pattern. This hyperintense intraluminal signal, which was identified in half of the atherosclerotic occlusions, may represent acute or subacute thrombus with a greater RBC component. Alternatively, small, angiographically occult channels of recanalization could produce areas of high signal consequent to slow flow.

Angiography remains the gold standard for the diagnosis of atherosclerotic stenosis and occlusion. However, this diagnostic test is not without its limitations. Several authors have reported their experiences with angiographically occluded ICAs that, on surgical exploration or repeat angiograms with large volumes of contrast material and prolonged injection times, have been noted to be marginally patent [10–13]. Retrograde flow via the ophthalmic artery into the carotid siphon and petrous and even cervical segments of the ICA has been documented with these modified techniques [11]. Only routine volumes and injection rates were used during the angiographic procedures reviewed in this study. Given these technical considerations, we cannot exclude the possibility that we failed to opacify the lumina distal to severely stenotic lesions that were included in the occlusion group, nor can we be sure that some totally occluded vessels did not reconstitute via small collaterals from the external carotid artery or contralateral ICA. Obviously, this amount of flow would be hemodynamically insignificant but might have important implications regarding intraluminal signal changes on

TABLE 3: Effectiveness of MR Imaging in the Diagnosis of Carotid Occlusion and High-Grade Stenosis

Disorder	No. of Studies				No. (%)				
	True Positive	False Positive	False Negative	True Negative	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value	Accuracy
Carotid occlusion	6	0	6	35	50 ± 32[a]	100 ± <1	100 ± <1	85 ± 11	87 ± 10
Stenosis	5	6	10	26	33 ± 27[a]	81 ± 14	45 ± 33	72 ± 16	66 ± 14

Note.—Criteria for occlusion were complete absence of flow void and intraluminal signal isointense with brain on all pulse sequences. Other patterns of intraluminal signal were interpreted as not occluded. Criteria for high-grade carotid stenosis were demonstration of partial flow void or any of the patterns of hyperintense intraluminal signal interpreted as slow flow. In this table, all cases of isointense intraluminal signal and cases of normal flow void were considered nonstenotic.

[a] Represents 95% confidence limits.

SE MR images of the brain. These possibilities may explain the inhomogeneously hyperintense signal that was encountered in approximately 50% of our angiographic occlusions.

Our own experience reflected the difficulties associated with the angiographic diagnosis of occlusion. Two cases in our review, one involving a critical ICA stenosis (Fig. 7) and the other a reconstituted CCA (Fig. 3), initially were misdiagnosed as total occlusions on angiography. Signal changes inconsistent with the angiographic diagnosis were observed on SE MR, prompting reevaluation of the angiographic studies, which subsequently demonstrated marginally patent vessels in both cases.

The MR appearance of carotid dissection was first described by Goldberg et al. [14]. The intraluminal signal changes observed in our two cases correlated well with the angiographic appearance. However, the intraluminal signal on axial and coronal long TR images was not specific, being indistinguishable from changes produced by atherosclerotic occlusion or stenosis. Axial short TR images through the area of dissection would allow a more specific diagnosis by demonstrating subintimal clot [15].

In summary, in our experience isointense intraluminal signal is specific for atherosclerotic occlusion. If this pattern is demonstrated consistently in two orthogonal planes on dual-echo sequences, we do not believe conventional angiography is necessary to confirm the diagnosis. However, atherosclerotic occlusion often contains varying degrees of hyperintense intraluminal signal that may prevent differentiation between occlusion and high signal produced by slow flow. Although a loss of flow void with variable hyperintense intraluminal signal is seen more commonly with occlusion than with high-grade stenosis, invasive angiography or MR angiography in combination with Doppler sonography will be needed to exclude marginal patency in these cases. The majority of high-grade stenoses produce no detectable signal changes on SE MR images of the brain. Partial flow void, although not a common finding, is a reliable indicator of proximal luminal compromise.

ACKNOWLEDGMENTS

We thank Saundra Ehrlich for assistance with statistics and Scott Faro for assistance in compiling the cases.

REFERENCES

1. Alvarez O, Edwards JH, Hyman RA. MR recognition of ICA occlusion. *AJNR* **1986**;7:359–360
2. Katz B, Quencer R, Kaplan J, Hinks RS, Post JD. MR imaging of intracranial carotid occlusion. *AJNR* **1989**;10:345–350
3. Heinz E, Yeates A, Djang W. Significant extracranial carotid stenosis: detection on routine cerebral MR images. *Radiology* **1989**;170:843–848
4. Brant-Zawadzki M. Routine MR imaging of the ICA siphon: angiographic correlation with cervical carotid lesions. *AJNR* **1990**;11:467–471
5. Uchino A, Ohnari N, Ohno M. MR imaging of intracranial vertebral artery occlusion. *Neuroradiology* **1989**;31:403–407
6. Knepper L, Biller J, Adams HP, Yuh W, Ryals T, Godersky J. MR imaging of basilar artery occlusion. *J Comput Assist Tomogr* **1990**;14:32–35
7. Russell EJ. Detection of significant extracranial carotid stenosis with routine cerebral MR imaging. *Radiology* **1989**;170:623–624
8. Bradley WG, Waluch V. Blood flow: magnetic resonance imaging. *Radiology* **1985**;154:443–450
9. Felmlee JP, Ehman RL. Spatial presaturation: a method for suppressing flow artifacts and improving depictions of vascular anatomy in MR imaging. *Radiology* **1987**;164:559–564
10. Clark OH, Moore WS, Hall AD. Radiographically occluded, anatomically patent carotid arteries. *Arch Surg* **1971**;102:604–606
11. Countee RW, Vijayanathan T. Reconstitution of "totally" occluded internal carotid arteries: angiographic and technical considerations. *Neurosurgery* **1979**;50:747–757
12. Gabrielsen TO, Seeger JF, Knake JE, Burke DP, Stilwill EW. The nearly occluded ICA: a diagnostic trap. *Radiology* **1981**;138:611–618
13. O'Leary DH, Mattle H, Potter JE. Atheromatous pseudo-occlusion of the ICA. *Stroke* **1989**;20:1168–1173
14. Goldberg HI, Grossman RI, Gomori JM, Asbury AK, Bilaniuk IT, Zimmerman RA. Cervical ICA dissecting hemorrhage: diagnosis using MRI. *Radiology* **1986**;158:157–161
15. Brugieres P, Castrec-Carpo A, Heran F, Goujon C, Gaston A, Marsault C. Magnetic resonance imaging in the exploration of dissection of the ICA. *J Neuroradiol* **1989**;16:1–10

The reader's attention is directed to the commentary on this article, which appears on the following pages.